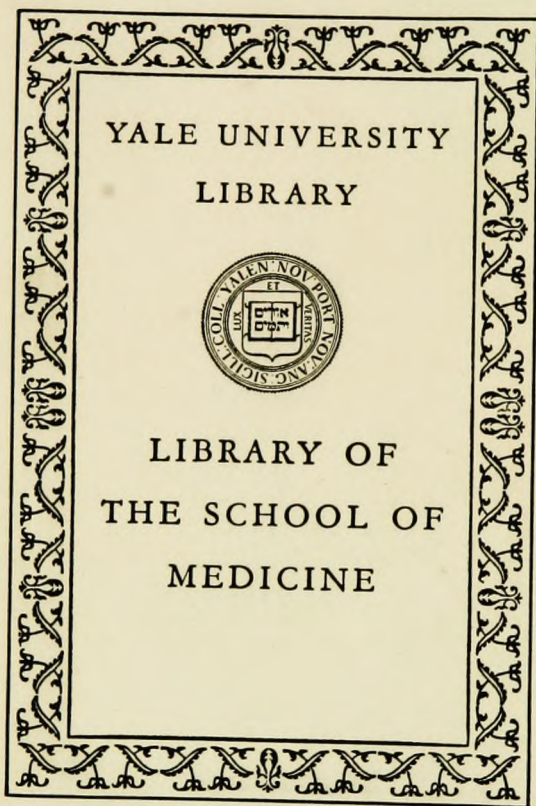


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DISEASES OF CHILDREN

DISEASES OF CHILDREN

BY VARIOUS AUTHORS

EDITED BY

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ILLUSTRATED

LONDON

EDWARD ARNOLD

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PREFACE

As the boundaries of knowledge are extended, it is inevitable that the limitations of individual experience should become more defined. In all departments of science during the last quarter of a century the advanced lines have been so rapidly pushed forward that it has become increasingly impossible for any single mind to compass the whole field. As in other branches of medicine, so in the study of disease in childhood, there has been a constant progress in the accuracy and extent of our knowledge. Recognizing and rejoicing in this fact, the editors were anxious to enlist in their enterprise the services of those whose knowledge is the result of peculiar attention to special portions of the field to be surveyed, and they desire to express their indebtedness to the readiness and energy of the allies whom they were so fortunate as to secure. With such assistance they have been encouraged to aim at a comprehensive and authoritative account of the present state of our knowledge of the Diseases of Children, set forth with a completeness which they believe has not hitherto been attempted in this country. To all their coadjutors, and to other colleagues and institutions, whose treasures of specimens and illustrations have been freely open to them, they owe and offer their warmest thanks.

A. E. G.

F. E. B.

H. T.

March, 1913.

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Facing page 309

PROLEGOMENA

I

FOREWORD

A. E. GARROD

FROM the child rather than from the malady the study of diseases of children receives its special impress. A line of cleavage, different in direction from those which split off other special branches of medical study, divides the ill of childhood from those of adult life. He who studies sick children has the whole of medicine and surgery for his field, and must take account of every organ; his only limitation is that imposed by the age of his patients.

He is concerned with the period of infancy, in which the patient is wholly dependent upon the ministrations of others for food, warmth, cleanliness, and all necessities of life, and with the period of active growth, in which intake must exceed output. In these periods of life errors of management, and disease even though transient, may leave indelible traces upon bodily or mental development; the intake is apt to be regulated by taste rather than by appetite, and the judgment needed for the avoidance of what is injurious, and the choice of what is best, is as yet in abeyance.

The youth of the patient calls for the exercise of special qualities in the medical attendant, and no man can be a good children's doctor who has not a real love of children. Examination of a sick child is in many respects easier than that of an adult. The organs are nearer to the hand and ear, are less often obscured by superfluous fat, and are enclosed in less rigid armour; but against these advantages there are not a few drawbacks to be set. The young child cannot tell us what he feels, and the seat of pain in an infant has often to be inferred from its actions. Examination is often hampered by noisy protest, if not by actual struggles, and the simplest instrument of diagnosis inspires terror in some children. Even when patient under examination, the young child seldom co-operates in the effort to reach a diagnosis.

There is no period of life in which careful dieting is more essential than in infancy, and this fact is generally recognized. Nature also recognizes it, and provides for the infant an optimum diet—maternal milk. She regulates the supply, and provides the several constituents needed—proteins, carbohydrates, and fats—in suitable proportions. As regards the human infant, the advance of civilization has to a large extent thwarted the beneficent provisions of Nature, and a large proportion of our children are brought up on milk indeed, but upon milk of animals of other species. The differences in the proportions of the several constituents can be corrected, to a greater or less extent, by dilution or humanization; but however closely cow's milk is brought to resemble human milk, it cannot be rendered

human. What degree of disadvantage this entails to the bottle-fed infant we are not yet in a position to judge.

In dieting older children we have to take into account the fact that an excess of intake is needed, since not only the due nutrition of the existing, but also the building up of new tissues has to be provided for. The growing organism needs to be continuously overfed, as measured by the adult standard of feeding.

There is evidence to show that any treatment to which milk be subjected tends to impair its value as a food, and nowadays infantile scurvy is more often due to excess of care than to gross errors in diet. Doubtless, in our ignorance, we often eliminate or destroy those mysterious constituents of the diet which contribute, in ways of which we know little as yet, to the welfare of the organism. Although not themselves foods, they are essential to the due utilization of the food taken.

Much of the high mortality amongst infants has its origin in dietetic causes. The food is liable to be infected by organisms which give rise to gastro-enteritis and absorption of toxic substances which readily overcome the feeble resisting power of the infant.

The diseases which are peculiar to children are comparatively few in number, but there are not many maladies which fail to acquire a peculiar stamp when they attack the growing organism, and the same symptom may suggest a different set of causes, according as the patient who exhibits them is a child or an adult. Growth and development may be retarded or arrested by diseases affecting various individual organs, and lesions of certain glands which largely control development produce in children effects from which adults are immune. The differences between the cretin and the patient who has acquired myxœdema in later life, between the pituitary giant and the adult sufferer from acromegaly, sufficiently illustrate this point. Again, the immature bones of the child become the seats of special morbid processes, such as rickets and the various kinds of epiphyseitis.

The immaturity of the nervous system also exerts a profound influence upon the form which disease assumes in children. In them the nerve centres are much more liable to explosive discharges than in adult patients, as witness the convulsions which result from comparatively trivial causes. The presence of round-worms in, or the absorption of toxic substances from the intestine, may suffice to induce convulsions in a child; and it is a well-known fact that a convulsion may replace a rigor at the onset of an infective fever. In early life the brain, spinal cord, and their membranes are very liable to bacterial invasions, and the various forms of meningitis, poliomyelitis, encephalitis, and tuberculous tumour formation, hold prominent places among the morbid accidents of childhood. Nor must the influence of lesions of nerve structures upon growth and development escape mention, an influence to which the wasted limbs and arrested bones of many victims of infantile paralysis bear eloquent testimony.

It is only natural that those infectious diseases which confer immunity from future attacks should be commoner in earlier than in later life, but apart from such immunity it cannot be doubted that the tissues of the child offer a more acceptable culture medium to bacteria generally than do those of the more mature individual.

A survey of the wards of any children's hospital in this country serves to bring out the fact that, leaving the infectious fevers aside, three pathogenic organisms are chiefly concerned in the causation of disease in early life. These are the pneumococcus, the tubercle bacillus, and the organism of rheumatic fever. There is reason to believe that on the Continent rheumatism is less prevalent than it is

amongst English children, and there can be no doubt that both geographical and racial factors play important parts in connection with the distribution of diseases; nor is the climatic factor open to dispute. Even in the British Isles, with their population derived from the fusion of several distinct races—Saxon, Scandinavian, and Celtic, with an admixture of French, Flemish, and Jewish blood—we can trace the influences of stock and parentage.

Each of the micro-organisms referred to produces different clinical pictures in children and in adult patients respectively. The comparative rarity of pulmonary phthisis in children, and their far greater liability to glandular infection, to meningitis, to tuberculous joint lesions, and peritonitis, bear witness to this fact as regards the tubercle bacillus. The prevalence among children of broncho-pneumonia, of pneumococcal empyema, and pericarditis, as contrasted with the predominant place held by lobar pneumonia amongst pneumococcal lesions in adults, is an equally striking phenomenon; but the differences of type of rheumatic attacks in childhood and later life afford the most conspicuous example of all.

The remarkable liability of the cardiac structures—pericardium, myocardium, and endocardium alike—to the attacks of the rheumatic organism, the comparative immunity of the joints, the frequency of chorea and of subcutaneous nodules, which are seldom met with even in early adult life, give to the rheumatism of childhood so distinctive a character that it is hardly to be recognised as the same disease as the rheumatic fever of older patients, in which articular pains and swellings play so conspicuous a part.

A like special stamp is impressed upon many other diseases when the sufferer is a child, as witness the lesions of congenital syphilis. Infantile scurvy, too, has its peculiar features, of which the most conspicuous is the frequency of subperiosteal hæmorrhages—lesions practically unknown in the scurvy of adults, and doubtless determined by the immaturity of the osseous system.

In its medical aspects, even the period of childhood needs to be further subdivided. The new-born infant is liable to special pathological accidents, some of which are due to infections of the umbilical area, whilst others, such as the skin affections of the sclerema group, admit of no such ready explanation. The later period of infancy, again, has its special dangers, and in later childhood certain hereditary maladies, of which pseudo-hypertrophic and other muscular dystrophies, and Friedreich's ataxia, may be referred to, tend to manifest themselves.

Even the age incidence of infective maladies differs within the period of childhood, and the sporadic form of meningococcal meningitis is usually met with in infants who have not yet reached the age at which tuberculous meningitis becomes common.

Congenital defects and malformations are more commonly seen in children than in adults, for not a few of the more serious of these tend to shorten materially the term of life. Thus, atresia of the bile-ducts proves fatal in infancy, and few sufferers from the graver varieties of congenital heart disease reach maturity.

As a compensation, there are not a few diseases which are rare, and some which are unknown, in childhood. Children rarely suffer from diabetes or exophthalmic goitre, and their relative immunity naturally extends to that group of maladies which may be described as the diseases of later life. In this group are included malignant growths of the carcinoma class—granular kidney and arterio-sclerosis, atheroma, gout, and osteo-arthritis. They escape, too, the grosser results of erroneous living, and the maladies which result from injurious occupations.

It is evident that the diseases of children offer a wide field of study. The fact that the patient is a child calls for a knowledge of special details in every branch of our work; details of symptomatology which enable us to recognize a disease under the unfamiliar aspects which the youth of the patients is apt to impart to it, and to appreciate the significance of physical signs; dietetic details to guide us in the administration of food suitable to the immature condition of the digestive functions of our patients; and details of therapeutics, to enable us to give with caution drugs which are not well borne by children.

Within the walls of a children's hospital, as of any hospital devoted to a particular purpose, there is encountered a special atmosphere, made up of special lines of thought and special lines of procedure, based upon the cumulative experience of generations of workers, and the interchange of ideas amongst men of like interests.

The more completely a work which deals with the diseases of children succeeds in formulating, and putting upon permanent record, this mass of floating knowledge, the more nearly will the object with which it is written be attained.

II

HEREDITY

A. M. GOSSAGE

THE characteristic qualities of a particular human being may be regarded as derived from two sources—those with which the child starts life endowed and those that he acquires during the course of his existence. Obviously, the latter are to a large extent dependent on the former. For instance, the mental acquirements are mainly the result of education, but are also dependent on the capacity of the brain to profit by experience, or, in other words, on the nature of the individual's original brain. Similarly, the diseases that anyone suffers from are mainly due to his exposure to the causes of those diseases, but also depend on his inherent power to resist the operation of those causes. Our equipment at the start of life depends on who our parents were, and thus the study of heredity is as important for the medical practitioner as the study of the patient's mode of life and environment. Unfortunately, heredity is a subject involved in the clouds of controversy, in which the ascertained facts are few and the hypotheses many.

It is possible for parents directly to pass on a disease to their offspring. For instance, a child may be directly infected with syphilis by either parent at the time of conception. Again, infection may pass from mother to foetus through the placenta: the offspring of a woman suffering from smallpox may be born covered with a varicellous rash, and there is some evidence that tubercle may in rare instances be conveyed in the same way. Pregnant animals artificially infected with tubercle have sometimes passed the infection on to their progeny, and it is difficult to explain some very early cases of tuberculosis in infants on any other hypothesis. On the other hand, the condition of the blood which renders the mother immune to certain infections may be conveyed to the foetus through the placental circulation. For example, a mother infected with syphilis during pregnancy may have an apparently healthy child, but is said never to infect that child after its birth, although her own syphilis may be florid (Profeta's law). A better example is afforded by vaccination: the infant of a woman successfully vaccinated during gestation is usually immune to vaccination for many months after birth. The immunity after an attack of measles lasts a very long time, and a mother may present to her unborn child some of the immunity she acquired from an attack in childhood. This is a possible explanation of the comparative mildness of measles in European countries, where nearly everyone has the disease in childhood, compared to the severity of an outbreak amongst a community where the complaint has been previously unknown. None of these are, however, properly speaking, examples of heredity; they are due to the conveyance by physical means of something definite—e.g., an infecting bacillus or the chemical substance which causes immunity—from parent to child.

It is a natural supposition that the health of the mother during pregnancy would affect that of the unborn child quite apart from any question of infection. There is a certain amount of evidence that a weakly child tends to be produced when the mother's condition of health is poor; but, on the other side, there are frequent examples of a woman who is suffering from a severe disease, such as pulmonary tuberculosis, bringing forth a perfectly healthy child, and even when the mother is in a condition of chronic starvation the *fetus* often seems to get sufficient nourishment. The most marked example of the influence of maternal physical condition on the child is afforded by the Mongolian imbeciles who are usually born at the end of a long series of pregnancies, or where the mother is over forty, and thus near the end of her reproductive period. When born of younger women, health during pregnancy has been usually very poor; but, still, Mongols occasionally appear when the mothers are young and in good health, and subsequent children may be normal. It has been supposed that parental alcoholism leads to the production of children who are feeble both mentally and physically. There is, however, no convincing proof of this supposition, apart from the fact that feeble-minded parents tend to be alcoholic and to have feeble-minded offspring. Naturally, the presence of poisons in the maternal circulation has a prejudicial effect on the *fetus*, and frequently in the earlier stages of pregnancy leads to abortion. Thus, abortion occurs commonly in many acute specific disorders, such as pneumonia and enteric fever, and also is not infrequent in chronic lead-poisoning.

Coming to the consideration of heredity, properly so called, it is important to remember that Weissmann has shown that the child is derived from the "germ-plasm," which has nothing to do with the parental organism except that the parents were themselves derived from the same germ-plasm, and that the germ-plasm is imbued and nourished inside the body of the parent. After the union of the two portions of germ-plasm supplied by the separate parents, the fertilized ovum divides into two parts very early, one of which alters and develops to a great extent to form the diverse somatic cells, whilst the other remains in its primitive state as the future germ-plasm of the new individual. Thus, anything acquired by the parents after their birth will not influence the future child. Because a father has studied and acquired a knowledge of mathematics, this will not make it easier for his son to learn mathematics, though both may derive a taste and capacity for that particular branch of learning from the family germ-plasm.

The new member of the human community arises from the union of two cells—one part of the germ-plasm of the father, and the other of that of the mother. After the union of the nuclei of these two cells, cell division takes place, and two daughter cells are cast out (the polar bodies); and then further division occurs, which leads to the development of a new individual. It is remarkable that the stainable portion of the nuclei forms before division in all cells a definite number of rods—the chromosomes—and that there are only half the number of chromosomes in the generative cells, ovum or spermatozoon, to what there are in all the other cells of the body. Thus, in the somatic cells of man there are thirty-two chromosomes, whereas there are only sixteen in the generative cells. On the union of two germ cells the chromosomes do not unite, but remain separate, so that the fertilized ovum contains as many chromosomes as the somatic cells of the adult organism, and double the number present in each of its constituent germ cells, half in every cell being derived from the mother, and half from the father.

Several interesting books have been written by Archdall Reid on the appli-

cation of Darwin's theory of evolution by natural selection to man. Offspring resemble their parents, and yet always differ from them in certain minor points, some of which should give some of them a better chance of adaptation to their surroundings than their parents or brothers and sisters. These small differences are known as continuous variations, to distinguish them from more marked discontinuous variations, or mutations. Where the struggle for existence is severe, those a little better adapted to the environment than their fellows should have a better chance of surviving and leaving descendants. It is assumed that those with favourable variations are more likely to have children of their own advantageous constitution, and also to have some children who show a further advance in favourable variation. In this way, with lapse of time and a sufficiently severe struggle for existence to eliminate the unfit, the species should gradually progress in the direction of becoming more and more perfectly adapted to its conditions of life. The variations are not, of course, all favourable, but the individuals with the unfavourable modifications would tend to die out and leave few or no descendants. If it be accepted that evolution takes place by the selection of slight favourable modifications in this way, it is a logical deduction that human races should be immune to infectious diseases, according to their past experience of them. It is an observed fact that individuals differ in their power of resisting infection by the various pathogenic micro-organisms; and provided the sources of infection be sufficiently widespread, the less resistant should die, the more resistant recover or not be attacked. The more resistant would then have the better chance to breed and to hand on their comparative immunity to their children, who should have a greater chance to show a still further advantageous variation, and the immunity of the race to the particular infection should slowly improve. Apparent illustrations may be found in measles and malaria. Measles is almost universal amongst Europeans, whereas it is rare in South American Indians; but when it occurs, is very severe and fatal. Malaria is notoriously fatal to adult Europeans, and is certainly much less so to West African negroes, who nevertheless are nearly all attacked in childhood.

This particular theory of evolution by the action of natural selection on continuous variations has of late years been vigorously attacked by many biologists, who point out that artificial selection by breeding from the most fit has only been able to advance a species to a very small extent, and that then further advance is impossible, and it is difficult to maintain the advance already made—e.g., in speed in racehorses. They also urge that large variations, where a marked advance is made at a bound, do occur, and that by breeding these large variations can be chosen to persist. If the premises be thus doubtful, the deduction requires other independent evidence before it can be accepted. It has already been shown that it is possible to account for the comparative immunity of Europeans to measles by quite a different hypothesis, and the apparent greater resistance of the negro to malaria may also be otherwise explained. In malarious districts nearly everyone is at some time attacked, and the less resistant would die off as children, leaving only the more resistant to reach adult life, "salted," or rendered still more immune, by their previous attacks. In every generation about the same proportion of susceptible and resistant would be born, but only the latter would reach adult life. If this is the correct explanation of the greater resistance of the black adult, then white children brought up in a malarious district should suffer no more than the natives, and where the two races have lived together for several

generations white and black should be equally affected. There is not yet much reliable evidence on which to found a conclusion, but Leonard Rogers reports that in India white children are found to suffer less than the native children in the same place; and Deaderick records that the white population of the Southern States of America are less affected than the black. The difference is favour of the whites would be due to the fact that they are less exposed to infection. On the facts as at present ascertained, Reid's law, "that nations are immune to infectious diseases according to their past experience of them," must be taken as not proven.

It is fairly generally accepted by medical practitioners that persons differ in their liability to different infections, and that a parent is prone to hand down his increased liability to his child. The infection in which this is regarded as most marked is tuberculosis, but it is that in which it is most difficult to get convincing proof. The fact that many members of a family suffer from tuberculosis may be due rather to their greater exposure to infection and to bad sanitary surroundings—e.g., small, ill-ventilated houses in a damp situation—than to any physical peculiarity which renders them more liable to infection. Records, however, from families in good circumstances, where the sick have been separated from the healthy, renders it almost certain that such an increased liability is derived by a child from its parents, and this doctrine is accepted at the present day by insurance companies. This view receives much support from the fact that it is possible to differentiate two breeds of wheat, one susceptible to "rust," and one resistant, and that, on crossing these and further breeding from the cross, the individual plants in the successive generations are attacked or free from the disease in the proportions expected by Mendel's laws. An attempt to add definiteness to the general belief has been made by Karl Pearson, who by statistical methods claims to have proved that the child of a phthisical father or mother has a definite mathematical greater liability to be attacked by tuberculosis than one whose parents are healthy.

The statistical method has been applied by the school known as the Biometricians to the solution of a number of biological problems. By it it can be shown that the children of tall parents tend to be taller than the average, but less removed from the normal than their parents. Statistics do not, however, demonstrate whether this is due to all the children being above the average, or to some of them being very tall and the rest ordinary. In other words, it is impossible to tell what will occur in individual instances from the general conclusions arrived at by the study of statistics. Further, in applying this method to research, it is essential that the conditions grouped together for statistical comparison shall be clearly defined and really comparable. For instance, one can lay no weight on conclusions drawn from the investigation of the hereditary transmission of such ill-defined mental characteristics as vivacity or conscientiousness. From statistical study Galton evolved the Law of Ancestral Inheritance, which says that every ancestor contributes his quota to the constitution of an individual, and that the resemblance of any one to each of his parents may be represented by the decimal figure 0.5, to his grandparents by 0.25, and to each great-grandparent by 0.125, and so on. While this law is undoubtedly true on the average if sufficient numbers are taken, it is quite untrue of the individuals composing those numbers. A further conclusion drawn from statistics was that the qualities of the separate parents tend to blend in the offspring. This may be true of those slighter differences which have been called "continuous variations" but is cer-

tainly not true of the more marked differences which are called "discontinuous variations," or "mutations." There is, however, one striking example in human beings where there seems to be a blending in the child of marked differences in the two parents. This is afforded by the cross between a European and a Negro, where the child is apparently a blend of the colour and other characteristics of the two races. On further crossing between two such cross-bred individuals, all the children are again blends of the two races, and crossing with either a black or white is said to lead in the offspring to an approach towards this particular parent. Nevertheless, on the whole the statistical method of inquiry has not led to any great light being thrown on the problem of human heredity.

Another school of biological thought arrives at its conclusions by the study of numerous individual examples of breeding. It owes its origin to the Abbé Mendel, who about the year 1865 studied the effect of crossing different varieties of pea. He found that, where one variety which was characterized by either tall stems or green pods or smooth seeds was crossed with another which had either dwarf stems or yellow pods or wrinkled seeds, the resulting plants had all tall stems or green pods or smooth seeds. If these cross-bred plants were then allowed to self-fertilize, they produced in the next generation, the tall plants a mixture of plants in the proportion of three tall and one dwarf; those with green pods a mixture of three green pods and one yellow; and those with smooth seeds a mixture of three smooth seeds and one wrinkled. Of the tall, green-podded or smooth-seeded plants of this second generation, two out of the three, on self-fertilizing, again produced plants in the proportion of three tall, green-podded or smooth-seeded peas and one dwarf, yellow-podded, or wrinkled-seeded; whereas the other of the three produced only tall, green-podded or smooth-seeded plants, whose offspring remained true to type, however often they were self-fertilized and fresh plants raised. On the other hand, the dwarf, yellow-podded or wrinkled-seeded peas produced offspring similar to themselves whenever they were self-fertilized. The conclusion seems inevitable that all the plants resulting from the first cross contained two factors—one derived from each parent—but only one factor showed itself—e.g., tallness, green pods, or smooth seeds—and this factor is said to be "dominant," whilst the other concealed factor—dwarfness, yellow pods, or wrinkled seeds—was said to be "recessive." On self-fertilization it seemed evident that one pure dominant was produced, two impure plants which showed the dominant factor—e.g., tallness—but contained also the recessive factor (dwarfness) and one pure recessive (dwarf). To explain these facts and inferences, Mendel suggested the hypothesis that all the cells of the body contain two factors, one derived from the male parent's germ-plasma, and one from the female parent's; but that in the germ cells, or gametes, these factors separated from one another, or segregated, so that each gamete only contained one; and of the total number of gametes produced by an individual, half would contain the factor derived from the father, and half that derived from the mother. Obviously, the factors supplied by the different parents might be the same (both that for tallness) or different (one tall and one dwarf). Thus in the cross between the tall and dwarf peas all the cells of the progeny would contain the factor for tallness and dwarfness except the germ cells, half of which would contain only that for tallness, and half only that for dwarfness. On self-fertilization it would be an equal chance whether an individual gamete would meet one of its own kind or one of the opposite kind, so that, where a number of plants result, they should be in the proportion of one formed

by the union of a tall gamete with another tall gamete, two by the unions of tall gametes with dwarf gametes, and one by the union of a dwarf gamete with another dwarf gamete. If tallness be represented by a , and dwarfness by b , then each cell in the offspring of a cross between a tall and a dwarf pea would have the composition ab ; but a , being dominant, would be the only thing shown. Self-fertilization may be regarded as the conjunction of two exactly similar parents, and the gametes concerned would with such cross-bred plants be equally a and b , and the union of these should result as follows:

Cross-bred plants	$2ab$	\times	$2ab$	
Gametes	$2a + 2b$	\times	$2a + 2b$	
Breeding plants	aa	$+$	$2ab$	$+ bb$

Pure dominant. Impure dominant. Pure recessive.

It would, naturally, be possible to cross the impure dominants ab with either pure dominants aa or pure recessives bb . In the first case the gametes would be—

$$2a + 2a \times 2a + 2b,$$

and the resulting plants—

$$2aa + 2ab,$$

that is to say, they would be all tall, but half would be pure with regard to tallness, whilst half would contain the recessive dwarfness concealed. In the second case the gametes would be—

$$2a + 2b \times 2b + 2b;$$

and the resulting plants—

$$2ab + 2bb;$$

that is to say, half would be tall impure dominants, and half dwarf pure recessives. Actual breeding experiments confirmed these theoretical predictions.

Mendel's researches have been greatly extended during the last few years, and similar phenomena have been found to exist for many factors both in plants and animals. Naturally, all examples of heredity are not as simple as that first worked out by Mendel, and it has been necessary to supplement his hypothesis by others. For instance, colours in flowers are supposed to be determined by the meeting together of more than one factor; but the probability of the truth of this further hypothesis is rendered extremely by the close approximation of the numbers of each particular colour obtained in various crosses with what is expected by theory. In some cases dominance is not exhibited, but in a cross there is a blend of the two opposing factors. For example, the blue Andalusian fowl results from the union of a speckled white with a black fowl; but blue Andalusians when bred together produce progeny in the proportion of one speckled white, two blue Andalusians, and one black, as would be expected if they are to be regarded as always a hybrid, or heterozygote, between the black and white. There are, naturally, some cases where the results do not quite agree with the Mendelian hypothesis, but these may yet be found to be due to a somewhat complex interaction of different factors. It is, of course, possible that this particular type of inheritance is only exhibited with regard to a certain number of opposing qualities; whereas in all other instances the offspring may be a blend of the germ-plasma of its parents. On the other hand, further research may show that this mode of heredity is universal, and that there is always segregation in the germ cells. It seems probable that the dominant quality is always due to the presence of some additional factor, in the absence of which the recessive quality shows itself.

When one comes to consider human beings, one finds that there are a number of instances of heredity apparently of this same type. Human families are small, and it is very often difficult to obtain accurate details as to all the children, including miscarriages. It is usually impossible to interview personally individuals from more than three generations of a family, so that an investigation has to depend on the information derived from the older members as to the condition of their ancestors, and such information is often inaccurate. Exact correspondence with the numbers demanded by theory must not be expected, and data derived from human families cannot afford very strong arguments for or against Mendelism. One is justified in saying, however, that if Mendelian heredity and segregation prevail in plants and animals, as shown by experiments, then they are also found in human beings. On the other side, human heredity supplies one of the strongest arguments against the universality of segregation. The crossing of two such dissimilar races as the negro and the European results in a blend, and the offspring of two such crossed individuals are said to be also always a blend, and it is asserted that segregation never occurs—i.e., that a pure black or a pure white never results from such a marriage, as would be expected according to the Mendelian hypothesis. Yet the interbreeding of Jew and Gentile seems to show definite segregation, according to Salzman, the characteristic facial features of the Jew being recessive to those of the Gentile.

Most of the examples of human heredity are drawn from abnormalities and the results accruing from the union of abnormal with normal individuals. There are, however, certain common characteristics which clearly obey the Mendelian laws. For instance, red hair is a recessive to other types of hair, so that red-haired children often are born to parents who themselves show no sign of red, but each of whom probably carries the factor for it concealed; children whose parents both have red hair are all red-headed. Eye colour affords another example. The colour of the eye depends on pigment in the iris, which may be situated in front of or behind the muscle, and its presence or absence in front of the muscle may be readily ascertained by an observer with a good light. Hurst has divided human eyes into three classes; those with no pigment in front of the iris, those with a ring of brown pigment round the pupil, and those with a large quantity of pigment all over the front of the iris, causing it to be all brown. The pigment is brown pigment visible to the naked eye, and has nothing to do with pigment visible only by the microscope; this latter may be found in all eyes, but is probably of different composition to the brown pigment mentioned above. All-brown eyes are dominant to ringed eyes, and both are dominant to blue unpigmented eyes; hence it follows that the children of blue-eyed parents have always blue eyes; that the children of parents both with ringed eyes may have blue or ringed eyes, but never completely brown; and that the children of brown-eyed parents may have brown, ringed, or blue eyes. These facts may, of course, be readily confirmed by anyone for himself.

Abnormalities are naturally rare, and hence an abnormal person probably inherits his abnormality, if it has been obtained by inheritance, from only one parent, provided that the abnormality is dominant to the normal condition. Recessive abnormalities must be derived from both parents, and hence tend chiefly to occur where the parents are consins; and since as a rule the parents do not exhibit the abnormality—i.e., are impure normal individuals—the abnormality only occurs in about one out of three of the offspring. Examples of such recessive abnormalities have been recorded by Garrod in alkaptonuria and steatorrhea. Of

apparently dominant abnormalities there are a large number—e.g., tylosis plantaris and palmaris, night-blindness, congenital cataract, claw hands and feet, etc. Since these conditions are rare, each abnormal individual almost certainly carries as a recessive in addition the normal factor. On marriage with a normal mate, half the children should be abnormal and half normal; and in recorded family histories this comes out with a fair approximation to accuracy. For instance, in a collection of twenty-nine families with tylosis plantaris et palmaris made by the writer, of the children of abnormal persons with normal spouses, 238 were abnormal, and 188 normal, which is fairly close to the expected equality, when allowance is made for the fact that abnormal individuals are more likely to be remembered in past generations than normal ones. The marriage of two abnormal persons is naturally of extreme rarity, but one instance is reported by Nezelof. A man and a woman, both with webbed toes, had seven web-toed children and one normal boy. The normal child had six normal children; whereas all the offspring of the

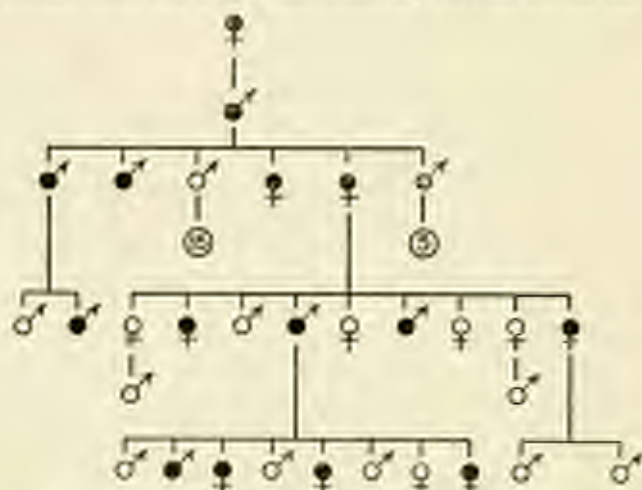


FIG. 1.—FAMILY AFFECTED WITH MONODACTYLIA.

Black indicates an affected individual, white a normal one. Figures in a circle represent numbers of normal children. (After Kohnsiegel.)

abnormal members of the family with normal mates were affected. The actual numbers in this family do not correspond with Mendelian expectation, probably because it is too small; but it is suggestive that some of the children of the first pair were pre-dominant (all of whose children should be abnormal), as would be expected in the proportion of one pure dominant, two impure dominants, and one pure recessive.

In these families the children of the normal members are all normal, but in some other families the abnormal condition is sometimes handed down through an unaffected individual. Examples of this are afforded by diabetes insipidus, epidermolysis bullosa, multiple cartilaginous exostoses (where the unaffected person who passes the condition down to her descendants seems to be nearly always a female), etc. No adequate explanation of this escape of certain carriers of the abnormality can be offered in human beings, but in animals or plants a similar skipping of a generation may be due to the need of the presence of more than one factor, so that the particular quality may show itself, or, again, to the presence of an inhibiting

factor in certain individuals. In some cases dominance may be incomplete, as seems to occur in the extra toes of fowls. In spite of this complexity, the evidence of the occurrence of segregation in the germ cells is as complete as in the case of simpler dominance, and the normal and abnormal progeny of a person capable of transmitting the abnormality are about equal in number. Since, however, a certain number of the apparently normal contain the abnormal factor, there is in this class no excess of the abnormal. Epidermolysis bullosa may be taken as an example. In 28 families there were 185 affected and 213 unaffected.

An abnormality may show itself at birth, or its appearance may be delayed until the child is a few years old, or even until puberty. In the latter cases there would naturally be a great excess of the apparently normal in the family, owing to many members dying before reaching the age at which the abnormality should show itself. Multiple telangiectases generally first becomes apparent after puberty, and in three families in which this complaint occurred there were twenty-seven normal persons to only fourteen affected.

Most of the conditions so far considered are not specially injurious to health, though epidermolysis bullosa and claw-hand must hamper some persons in earning their livelihood; and multiple telangiectases sometimes leads to early death. It

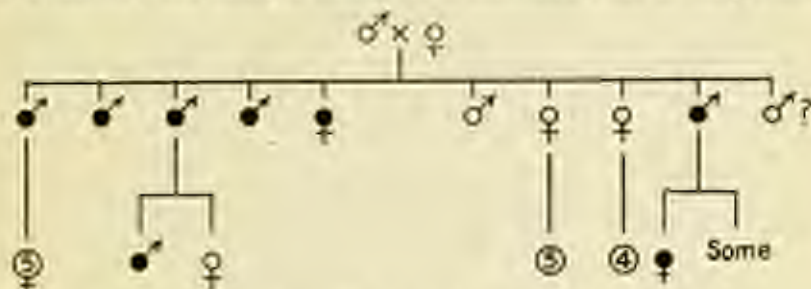


FIG. 2.—FAMILY WITH MULTIPLE CARTILAGINOUS EXOSTOSES.

is noticeable that in no case does the abnormality tend to be bred out by intermarriage with normal persons, but persists in a definite proportion of the offspring, though it cannot be regarded as ever an advantage or an adaptation to the surroundings. There is here, therefore, a contradiction to the prevailing belief that only those variations from the normal persist which help the race to be better adapted to its environment.

Certain attributes in animals are almost limited to one sex—for example, the presence of horns in a certain breed of sheep. The horns generally only occur in the male, and the condition would be explainable on the Mendelian hypothesis by supposing that it is dominant in the male and recessive in the female. Thus, a hornless male would be pure with regard to the absence of horns, whereas a hornless female might be impure in this respect, and a horned male might be born from the conjunction of two such hornless parents. On the other hand, a horned female could only result from the union of a horned male and a female who carried hornedness as a recessive, and would necessarily be pure. Therefore all the male progeny of a horned female should have horns, whatever the condition of the father. The results of breeding experiments are in agreement with this theoretical expectancy. Similar sex-limited conditions are found in human beings, striking examples being afforded by hæmophilia, colour-blindness, and hypertrophic muscular paralysis.

As would be expected, all the male children of a colour-blind female are colour-blind. In hypertrophic muscular paralysis, as a rule, the disease renders the patient incapable of procreation; so that there are no details as to the progeny of female sufferers. According to Balloch, hemophilia, properly so called, does not occur in females, and the hereditary tendency to bleed which is found in some families, where females also suffer, belongs to a different category. In true hemophilia, unaffected males marrying with normal women not belonging to bleeder families never pass on the condition to their children; but it is obvious that the hemorrhagic tendency may be transmitted through several generations of the apparently normal females of affected families, as well as being handed down by a bleeder male to his grandson through his unaffected daughter. It is remarkable that a man who is a bleeder seldom, if ever, transmits the condition to his son; in fact, there seems in the children of a bleeder to be a repulsion between maleness and the factor for bleeding. Hemophilia frequently has an apparently spontaneous origin, no similar case being known in the relatives of the patient. It is probable, however, that in these instances it has really been transmitted from a bleeder family through a number of unaffected females (cf. also Chapter IX, p. 543).

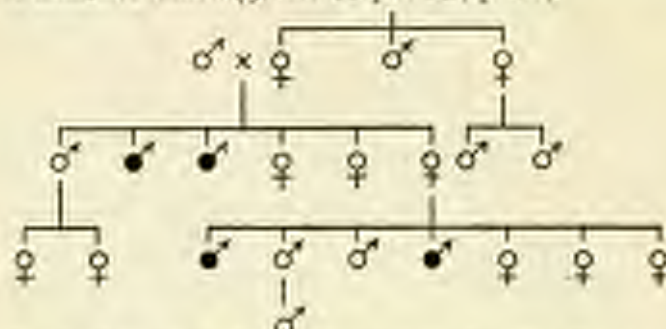


FIG. 2.—FAMILY WITH PSEUDO-HYPERTROPHIC MUSCULAR PARALYSIS.

Hemophilia causes the death of the majority of the male sufferers in early life, and pseudo-hypertrophic muscular paralysis incapacitates most of its victims from procreation; it thus almost appears as if this peculiar method of hereditary transmission were specially designed to preserve these noxious conditions. The preservation of hemophilia is further assisted by the enormous families which the women of bleeder race commonly have. The notion, however, of a special design for the preservation of a condition which would otherwise quickly die out is negatived by the fact that the same type of inheritance is found to obtain in colour-blindness, which, as it does not injure either health or capacity, requires no such method of preservation.

Sufficient has been said to emphasize the importance of the study of heredity, and also its difficulties and the need of further research. Of the various schools of biological thought, at present that known as the Mendelian has shed the most light on the subject of human heredity, and to define its scope and possible limitations the collection of accurate family histories is urgently required. Already the medical practitioner pays regard to his patient's relatives when considering the lines of treatment, and recognizes the value of taking special precautions where tubercle or rheumatism have attacked several members of the family, or where

several have shown signs of nervous instability. More drastic measures have been advocated by some under the name of "eugenics," measures which aim at the prevention of the future occurrence of abnormalities by forcibly preventing the present abnormal from breeding. It is probable that in this way such conditions as tylosis plantaris et palmaris, claw-hand, etc., could be stamped out; but it is a doubtful question whether they can be regarded as sufficiently noxious to warrant such an interference with the liberty of the individual. A stronger claim may be urged with regard to feeble-mindedness, which seems to be inherited on Mendelian lines, and which is responsible for much vice and crime, and so expense to the community; but even here more exact definition of what is meant by "feeble-mindedness" and more accurate family records are required before a convincing case could be presented for legislation. The elimination of those tragic conditions, hæmophilia and pseudo-hypertrophic muscular paralysis, would justify strong measures; but it must be recognized how severe they would have to be. All the females of affected families, although in themselves healthy and apparently normal, would have to be prevented from having children, and that, too, in spite of the fact that only some of them are capable of transmitting the abnormality, for the only proof of a woman of these families being perfectly normal is the bearing of several normal male children. It would be impossible, also, to trace out all the possible mothers of abnormal males, since the condition may pass through many generations of apparently normal females—generations in which males may be absent or scarce.

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III

IMMUNITY, SERUM-THERAPY, AND VACCINES

H. THURSFIELD

THE laboratory study of the problems of immunity has made great progress in the years that have elapsed since the discovery of bacteria as the cause of disease. That discovery at once opened the door to experimental research into the modes by which the body resists the attack of the infecting organism; and though the problem of the acquisition of immunity is far from solution, the researches into this subject have led to the introduction of new methods of treatment, some of which are already firmly established, others as yet in the stage of experiment. This article is an attempt to review shortly the present state of our knowledge with regard to the problem of immunity, and of the various therapeutic methods which have been evolved from the study of the problem.

An early result of this study was the discovery by Metchnikoff that various kinds of cells, especially the leucocytes of the blood, were possessed of actively destructive powers towards the invading organisms. This power he named "phagocytosis," and to the activity of the phagocytosis he attributed immunity. Further research showed that, though this was indeed a part of the truth, the leucocytes were not the only factor in the resistance offered to infection. It was found that the plasma of the blood, when all corpuscles had been removed, possessed substances which destroyed the micro-organisms. This bactericidal power of the blood-serum was attributed to hypothetical bodies, to which the name of "alexines" was given. Further researches revealed the presence in the blood-plasma of other substances which possess the power of neutralizing the poisonous products of bacteria, and to these the name of "antitoxins" has been given. Other groups of substances have been found in the blood-plasma, which possess special properties—haemolysins, agglutinins, precipitins, and opsonins—the study of which has thrown much light upon the problem of immunity; but for the present purpose it will be sufficient to confine our attention to phagocytosis, bacteriolysis, and antitoxins.

Phagocytosis.—When an organism invades the tissues of the body, the leucocytes gather together at the point of invasion to repel the attack; the nature of the attraction is unknown, but is briefly spoken of as "chemiotaxis"; and there can be no doubt that the activity of the phagocytic process plays a large part in the protection of the body tissues. Since Metchnikoff's work, however, Wright and his followers have shown that substances present in the serum of the blood, to which the name "opsonins" has been given, are necessary for active phagocytosis, these substances acting, probably, by diminishing the resistance of the bacteria to the attacks of the phagocytes. Their precise nature, and their relation to other

substances found in the blood-serum, are matters of much dispute; but the important point for the therapist is that it is possible to increase the quantity of these substances contained in the blood-serum, and so to render the invading bacteria less likely to overcome the resistance of the leucocytes. It is, in fact, chiefly from this consideration that the whole practice of injecting patients with bacterial vaccines has grown up, though such vaccination certainly has other effects besides the production of opsonins.

Bacteriolysis.—If an animal is injected with a minute non-lethal dose of certain pathogenic organisms, and some time allowed to elapse, it is found that the animal can tolerate a larger dose. Increasing doses at measured intervals increase its power of resistance, till it can in the end tolerate a dose many times larger than would be required to kill a normal animal. This immunity is found to be due to the production of bodies—bacteriolysins—in the blood-serum, which possess the power of destroying the bacteria. The serum of such an animal injected into a human being does not protect him from the bacterial poisons which have been already manufactured in his body, but does to a certain extent protect him from invasion by the bacteria. Such serums are known as "anti-microbial" or "anti-bacterial" serums, and have been used with success in some diseases of human beings. The most notable success of such a serum has probably been obtained with Selava's anti-anthrax serum. Anti-bacterial serums do not neutralize bacterial poisons; they are quite different from the antitoxic serums.

Antitoxins.—In the year 1890 it was shown by Behring and Kitasato that it was possible to render animals immune to the poison of tetanus by injecting them with continually increasing doses of the poison, beginning with minute amounts; further, that the serum of such immune animals, when injected into a non-immune animal, enabled this latter to overcome the effects of the tetanus poison, if given within a short time of the invasion of the bacilli. The same was shown to be true of the toxins of diphtheria. These antitoxins were shown to have no bacteriolytic power; they act by neutralizing the poison, not by destroying the organisms.

The antitoxin of diphtheria has been employed in human beings to an enormous extent, and has met with the greatest success; indeed, diphtheria has, thanks to the beneficent action of antitoxin, to a large extent lost its terrors for the patient and the physician.

Complement and Amboceptor.—Further research into the processes which underlie the production of immunity has resulted in the discovery that in an animal which has been immunized by repeated small doses of bacteria or bacterial toxins the immunity acquired is due to the interaction of three substances. These three substances are: (1) The antigen—i.e., the substance which has been injected (e.g., a bacterial vaccine); (2) the immune body, otherwise called the "amboceptor" or "copula," which is produced by the operation of the antigen on the tissues; (3) the complement, a substance normally present in the blood-serum. The interaction which occurs consists in the amboceptor forming a link (copula) between the complement and the antigen, thus neutralizing the effect of the antigen upon the body tissues. The amboceptor is specific, and can combine only with its related antigen; the complement is non-specific, and can interact with any amboceptor and its related antigen. The amboceptor is thermostable, and is not destroyed by heating the serum to 55° C.; complement is thermolabile, and is destroyed by such heating, and the serum is

then said to be "inactivated." When the interaction between the antigen, the ambceptor, and the complement, has occurred, this last is said to have been "fixed."

These facts have been utilized to devise a test which is of wide application, but has especially come into prominence in the diagnosis of syphilis. The principle involved may be stated as follows: If a rabbit is injected with the blood corpuscles of a sheep (antigen), an immune body (ambceptor) is formed in the rabbit's serum. If then the rabbit's serum is brought into contact in a test-tube with a suspension of sheep's corpuscles in normal saline solution, interaction occurs between the sheep's corpuscles (antigen)—the immune body which has been formed in the rabbit's serum—and the complement normally present, and hæmolysis occurs. If the rabbit's serum has been previously heated to destroy the complement, no interaction can occur, and no hæmolysis. Such a preparation of inactivated rabbit's serum and sheep's corpuscles is an "incomplete hæmolytic system."

If now a second mixture is made of (1) syphilitic antigen—e.g., an emulsion of the liver tissue of a syphilitic foetus—(2) of inactivated serum from a patient suspected of syphilis, and (3) of fresh guinea-pig's serum (complement), supposing that the specific immune body related to the antigen is present in the suspected serum, it will link up the complement and the antigen (fixation of complement); if the specific immune body is not present, no interaction will take place, and the complement will be left free in the mixture. This mixture is then added to the "incomplete hæmolytic system." If the complement is still free, hæmolysis rapidly occurs, the complement being fixed to the sheep's corpuscles by the immune body of the rabbit's serum; if the complement is fixed already, no hæmolysis will occur. Hence the deduction is made that the specific immune body is or is not present in the suspected serum. If there is hæmolysis, the complement was not fixed, and the immune body was therefore not present in the suspected serum; if there is no hæmolysis, the complement was already fixed, and the immune body was present—i.e., the patient has syphilis.

The foregoing is an account of the principle involved in Wassermann's test. In actual practice there are numerous details, to which careful attention must be paid in order to insure accurate results.

Agglutination.—An allied but slightly different process takes place in the phenomenon known as "agglutination" or "clumping" of bacilli. In the presence of an invading microbe agglutinins are produced in the blood-serum, which possess the power, when brought into contact *in vitro* with an emulsion of the specified microbe, of "clumping" the organisms into masses. This phenomenon has been widely employed, especially in the diagnosis of typhoid fever, and is commonly spoken of as "Widal's reaction," from the observer who first employed it. The exact nature of agglutinins is unknown; they are probably not directly concerned with the production of immunity, and yet there is evidence that agglutination does diminish to a marked degree the virulence of the bacteria. They are closely allied to the precipitins of the blood-serum, and it is possible that the actual agglutination is the result of the precipitation of a proteid which serves to bind the bacteria together.

Forms of Immunity.—From a consideration of the above-mentioned facts it is possible to state briefly the different forms which immunity may take. Natural immunity to certain infections, it must be assumed, is due to the fact that the individuals who are immune (1) possess leucocytes with an unusual phagocytic

power, or opens in more than usual quantity; or (2) that they possess the specific immune body already fully developed in their tissues; or (3) that the invading bacteria or toxins find no possibility of uniting with the tissue cells—are, in fact, for such individuals merely inert matter with which the tissue cells have no concern. Acquired immunity, similarly, may be due to (1) an enhancement of the powers of the opsonins and the phagocytes, or (2) to the development of anti-toxins, and (3) bacteriolysis. Such acquired immunity produced in an individual by the activity of his own tissues is called "active immunity." Where, on the contrary, immunity has been conferred by the administration of a dose of anti-toxin or of anti-microbial serum, it is called "passive immunity." Such passive immunity, due, not to the living activity of the tissues, but to a temporary addition of already manufactured protective substances, is of short duration in the majority of instances.

The application of the principles above discussed to the practice of medicine must be considered in two ways: (1) As regards the diagnosis of disease; (2) as regards the prophylaxis and cure of disease.

I. DIAGNOSIS.

A. In order to ascertain the nature of the infecting organisms, the most simple and direct procedure is to investigate the lesions produced by it, and where these lesions are obvious and accessible this is the best and easiest method. The examination of the exudate upon the tonsils, for example, may reveal the presence of the diphtheria bacillus; the examination of the sputum may show the tubercle bacillus. In many instances, however, difficulties confront the inquirer, and these are chiefly of two kinds: (1) the lesion produced by the bacterium may be inaccessible; (2) the infecting organism may be so mingled with other organisms, or so sparsely present, as to render its direct identification a matter of chance or luck. The inaccessibility of the lesions has been to a considerable extent reduced of recent years by the free employment of puncture. Thus, the employment of lumbar puncture has enabled the pathologist to identify in many cases the organisms which give rise to meningitis, and the same is true of many infections of the joints and the pleural cavities. Puncture of solid organs, such as the liver or spleen, is also freely employed, and puncture of the consolidated lung is rightly becoming a more frequent occurrence. In other instances the infecting organisms can be detected by the simple process of demonstrating its presence in the circulating blood, either, as in malaria, by direct examination, or, as in typhoid fever, by culture of portions of the blood withdrawn for the purpose. Whatever the nature of the infection, it cannot be too strongly emphasized that direct film examinations or cultures, or both, should be made of all pathological material withdrawn from the body. No abscess should be opened, pleural or peritoneal cavity explored, without such an examination, which is a necessary preliminary to all attempts at producing immunity.

B. Sero-Diagnosis.—Where the direct identification of the infective agent is impossible for any reason, it may yet be possible to arrive at a very high degree of certainty as to its nature. The methods employed depend upon the demonstration in the serum of the patient of substances which are specific to the suspected disease. It is obvious that such a demonstration is liable to a number of

fallacies, and hence the performance of these tests requires considerable training, the use of "controls," and the observance of various conventions which have been devised to eliminate so far as possible sources of error.

(a) **OPSONIC INDEX.**—It has been claimed that by a carefully-devised method it is possible to compare the amount of opsonin present in a patient's serum with that of normal individuals. This method has been most largely employed in the diagnosis of tuberculous infection, the assumption being that, where the patient's opsonin deviates widely from the normal, he is the subject of tuberculosis. Much controversy has taken place on this subject, but, without entering into the arguments, it may be briefly stated that, in the writer's opinion, the fallacies in connection with this test are so numerous, and so little appreciated, that the test is of slight value to the clinician.

(b) **AGGLUTINATION TESTS.**—The presence of specific agglutinations in the serum of the patient can be demonstrated in typhoid fever, the paratyphoid infections, infections with *Bacillus coli communis*, *B. Gaertneri*, the micrococcus of Malta fever, and, with less certainty, in a few other infections. Practically the chief use made of the method is in the detection of typhoid fever. In this disease the specific agglutinins are present in the serum about the end of the first week of the disease; in a few cases somewhat earlier. It is marked as a rule during the course of the disease, and persists after convalescence for a variable time, sometimes up to ten or fifteen years. The positive reaction—*i.e.*, the clumping of typhoid bacilli by the patient's serum—is strong evidence of the existence of the infection, only exceeded in value by the direct demonstration of the bacilli in the blood or feces; while, on the contrary, a negative reaction—*i.e.*, the absence of clumping—is of comparatively little significance. In the writer's experience, on only two occasions has a positive reaction been obtained in the absence of typhoid infection, and in both instances there were other features which made the diagnosis of typhoid fever highly improbable. On the other hand, negative reactions are often obtained in cases which are afterwards shown to be typhoid infections, these negative reactions being most often the result of the neglect of some obvious precaution in the technique of the experiment, but yet occurring often enough, even with the most careful worker, to render their significance almost valueless. A series, however, of negative reactions in the same case at frequent intervals is of value.

With regard to some of the other infections named, the agglutination reaction is of considerably less importance; it is not so constantly present, and in some instances not so closely specific, as in the case of typhoid. Thus, in certain instances of *B. coli* infection agglutination may occur, not only with this organism, but with some of the other members of the coli-typhoid group. In the case of Malta fever, however, a positive reaction is of the greatest importance.

It must, of course, be remembered, with regard to tests based upon the presence of hypothetical substances in the serum, that, while they may be of extraordinary advantage to the clinician, they can never be of the same value as the actual demonstration of the infecting organism. The demonstration, for example, of the *B. typhosa* in the blood of a patient is of vastly greater importance and value than the agglutination reaction, in the interpretation of which there are possible fallacies.

(c) **THE WASSERMANN REACTION**, which has already been described, is of value for many cases of obscure infection, but has as yet been used chiefly in the diagnosis

of syphilis. A positive reaction is obtained in the great majority of cases which are infected with syphilis, and a negative reaction in the presence of well-marked lesions is, unlike the Widal test, of almost more value than the positive result. Further, the Wassermann test can be used as an indicator of the efficiency and of the duration of treatment.

The Wassermann reaction has also been used in the diagnosis of hydatid disease, and affords great help in the distinction of these parasitic cysts from other similar tumours.

C. Vaccine Diagnosis.—If a patient whose tissues are the seat of infection with a specific micro-organism be injected with a dose of an emulsion of that organism or of its toxins, there may occur a local or general reaction, and this reaction may be purposely sought for diagnosis. The method has been extensively employed in the case of tuberculosis, but has been as yet little used in other infections.

Tuberculin has been used with this object in various ways, the most trustworthy of which is the injection of small doses of Koch's old tuberculin beneath the skin. A positive reaction consists in a rise of temperature of one degree above the normal, often accompanied by a general malaise, headache, and pains in the limbs, and possibly also an inflammatory reaction in the affected region. To carry out this test, the patient should be in bed, and should have a steady normal temperature, carefully charted for several days. The first dose of tuberculin should be 0.0005 c.c. ($\frac{1}{2}$ milligramme), injected beneath the skin. If no reaction occurs, this is followed by 0.001, 0.002, and 0.005 c.c. at intervals of two days. A constant negative result—i.e., the absence of any reaction—is good evidence of the absence of tuberculous infection; and a positive reaction is certain evidence of the presence of tuberculosis.

The doses stated above are suitable for a child aged four years and upwards; for infants half or a quarter of these amounts are sufficient.

The cutaneous reaction of von Pirquet is obtained by rubbing a small drop of undiluted old tuberculin into a scarified surface on the skin, a control puncture being made at the same time with normal saline solution. A positive reaction is indicated by the development within twenty-four to forty-eight hours of an area of oedema and redness, with sometimes a few vesicles around the puncture. The value of this test is dubious. On the one hand, it is claimed that the test is so delicate that a positive reaction is obtained in cases where there are no active, but only obsolete, lesions; on the other, it is agreed that a certain number of undoubtedly tuberculous patients do not give the reaction, these failures being chiefly met with in the more acute forms of generalized tuberculosis and in tuberculous peritonitis and meningitis. In the writer's own experience, a positive reaction is too often obtained to enable him to feel much confidence in its value; but in infants and children under five years of age a negative reaction is to be trusted. By some authorities more trustworthy results are claimed from the use of a series of dilutions of the tuberculin.

A modification of this test has been lately introduced, which consists in the use of an ointment containing the tuberculin rubbed into the unbroken skin. Of this there is as yet no sufficient knowledge.

Calmétte's tuberculin reaction consists in the development of a conjunctivitis following the instillation of old tuberculin, separated from its glycerine and appropriately diluted, into the conjunctival sac. The method has in a few cases led to

serious inflammatory trouble, and as, in addition, the interpretation of the results is often very difficult, it has been generally abandoned.

In the case of bacterial endo-toxins of other organisms, very little use has been made of the method. In the gonorrheal joint disease of adults an injection of a post-coccal vaccine is sometimes followed by a more or less severe reaction in the affected joints, and the same result has occasionally followed the use of vaccines in *E. coli* infections of the urinary tract. In children's diseases the writer knows of no instances where this method has been used.

II. PROPHYLAXIS AND THERAPEUTICS.

For the prophylaxis or cure of disease, the possible modes of procedure, based upon the principles considered above, are three in number: (1) The promotion of active immunity by injection of appropriate doses of an organism or its toxins; (2) the promotion of passive immunity by the injection of an antitoxin to neutralize the poison already circulating in the body; or of an anti-microbic serum to inhibit the growth of the invading organism.

Sensitized Vaccines.—Until lately it was believed that there was no mutual antagonism between active and passive immunity, that the action of a serum did not inhibit that of a vaccine, and that there was no reason why the physician should not attempt to secure a degree of passive immunity by the use of a serum, while at the same time he was aiming at the production of an active immunity by the injection of a vaccine. Recent research has tended to show that the administration of a serum interferes to some extent with the production of active immunity by vaccines. Borelka has accordingly elaborated a method of improving the efficacy of a vaccine by combining with the specific antibody contained in the serum. To this product he has given the name of "sensitized vaccine." A vaccine prepared in the ordinary way is mixed with serum containing the specific antibody, and allowed to stand at the room temperature for twelve hours. The bacteria unite to themselves the antibody present in the serum. The emulsion is then washed clear of any traces of serum, the bacteria killed by heat or by 0.5 per cent. phenol, and the sensitized vaccine is ready for use.

Experimentally, animals immunized by this form of vaccine are less liable to exhibit toxic effects, and become immune more quickly and more certainly than with ordinary bacterial vaccines. Clinically, sensitized vaccines have been as yet but little used; but the results obtained are distinctly encouraging, and, in view of the striking experimental success, they are certain of an extended trial.

Antitoxin-Therapy.—Of the various antitoxic sera which have been prepared, two alone have attained any considerable reputation—those of tetanus and diphtheria.

This last is now firmly established, and is in general use; nor can an impartial observer have any doubt as to the enormous alteration in the prognosis of the disease which has resulted. In the case of the tetanus antitoxins the results are much less striking; and although experimental work with animals has demonstrated its efficacy under certain conditions beyond civil, yet in practice these conditions are seldom possible of attainment, and the use of the remedy often, therefore, disappoints the physician.

The only other sera considered to be definitely antitoxic which have had any marked success are Yersin's plague serum, which appears from numerous observations to be of considerable value, and Dunbar's "pollantin," an antitoxin for the poison of hay fever.

Anti-bacterial Sera.—The great majority of the sera which have been prepared are antimicrobic in their action, although in some cases there is some small evidence that they are slightly antitoxic as well. Generally speaking, the antimicrobic sera have disappointed expectation; and though brilliant results are from time to time recorded, these successes are gained without any certainty, and apparently in obedience to no law.

The list of anti-microbic sera is a long one. Those which should, in the writer's opinion, always be employed at the earliest opportunity are some brand of polyvalent anti-streptococcus serum, Selazo's anti-anthrax serum, and Ruppel's or Flexner's anti-meningococcus serum. With all of these striking results have been obtained, and, though no one can predict that in an individual case the serum will be successful, the percentage of success is high enough to make the failure to employ them a neglect of an obvious duty.

The other anti-microbic sera include anti-coli-bacillus serum, anti-dysentery serum, anti-typhoid serum (Chantemesse), anti-gonococcal serum, anti-pneumococcal serum (Pain), anti-staphylococcal serum, and various anti-tubercle sera. Of these, probably the best are Chantemesse's anti-typhoid serum and Pain's anti-pneumococcal serum; but the evidence as to their efficacy is conflicting.

In addition to these specific sera, claims have been made in favor of the use of various sera (1) to combat secondary infections; (2) to cure or improve conditions in which the infecting organism is either not known or for which no efficacious specific anti-serum has yet been prepared.

Thus, (1) anti-streptococcus serum has been used beneficially in the secondary streptococcal infections of tuberculosis and typhoid, and (2) claims have been made for the great benefits derived from the use of diphtheria antitoxic serum in pneumonia, in septic conditions, in asthma, in tuberculosis, and in many joint diseases. This list is so diversified as to rouse scepticism. But, as a result of the reported benefits in so wide a list, some observers have been led to believe that an effective raising of resistance to microbial invasion may be brought about by the use of normal horse serum; and although convincing proof is in the nature of the case impossible, certain clinical results are remarkable. There is, in the writer's opinion, little doubt of the efficacy of normal serum as a local application to suppurating wounds—e.g., carbuncle—but the evidence of benefit in other conditions and by other methods of administration is conflicting.

METHODS OF ADMINISTRATION OF SERA.—Till a few years ago the universal method of administration of sera was subcutaneous; more recently intravenous injection has been tried, and administration by the mouth and by the rectum have also their advocates. In the writer's judgment, the subcutaneous method still holds the field for the bulk of cases, while in certain circumstances the intravenous path has the advantage of introducing the serum more rapidly into the circulation. Should this method be selected, it must be remembered that it is essential that the serum be considerably diluted, and warmed to the temperature of the body, before introduction. Oral and rectal administration have some theoretical disadvantages, and in the present state of knowledge cannot be said with certainty

to attain the results of the other two methods, which should therefore always be chosen by preference.

The **ill effects** which occasionally follow the injection of sera must be briefly alluded to. The injection of diphtheria antitoxin has in a few cases been followed by sudden death, with symptoms of syncope; while in other cases symptoms of alarming collapse have occurred, with vomiting and erythematous eruptions, followed by recovery. The cause of these symptoms remains obscure, but it is known that the element of danger resides, not in the antitoxin, but in the horse serum in which it is conveyed. The ill effects seem to be associated (1) with large doses; (2) with serum drawn from certain horses; (3) with serum very recently prepared; and to be obviated to a certain extent (i.) by heating the serum to 60° C.; (ii.) by preliminary administration of calcium chloride or lactate.

The cases, however, in which these immediate ill effects are observed are very uncommon, while, on the other hand, the train of symptoms known as the "serum disease" are fairly frequent. These symptoms appear from the eighth to the fifteenth day following the injection, and consist in the appearance of rashes, erythematous, vesicilliform, scarlatiniform, hæmorrhagic, or urticarial; pains in the joints and body; occasionally a moderate degree of fever; and general malaise. They usually pass off in a few days. Rarely some degree of albuminuria may appear with the rash, but it has no serious significance.

Anaphylaxis.—If a foreign protein—and horse serum contains such a substance—be administered to an animal, a period of ten to twelve days allowed to elapse, and then a second dose given, the animal in a few minutes or a few hours develops alarming symptoms, collapse, convulsions, and in many cases death. To this phenomenon the name "anaphylaxis" (hyper-sensibility or super-sensibility) has been given, and it is probably to the development of this condition that many of the alarming symptoms which occasionally follow second doses of therapeutic serums are due. Instances of anaphylactic phenomena in human beings are not at present very numerous, probably because the interval between the doses given is not usually sufficiently long to enable the hyper-susceptibility to be produced; and experimentally it has been shown that the hyper-susceptibility can be obviated by the administration within the ten-day period of a second dose. Further doses within what may be called the "anaphylactic" period are not then accompanied by the characteristic phenomena. The writer has, however, knowledge of at least two cases in which the administration of a second dose of serum within the "anaphylactic" period—i.e., after the ninth or tenth day—has been attended with alarming symptoms of collapse, high fever, restlessness, and nausea. None of the cases so far reported has terminated fatally. It is clearly wise, in view of this knowledge, to administer serum in such a manner as to avoid the complication—that is, either to give a second dose within the week, preferably within forty-eight hours, or to defer the second dose till the expiration of a month from the first.

VACCINE-THERAPY.

Bacterial vaccines are preparations in which the micro-organisms are suspended in normal saline solution in quantities which are approximately known. The injection beneath the skin of such preparations is one of the most certain methods experimentally of producing active immunity, and though, in the case of the human being, the therapeutic results are less constant, and perhaps some-

what disappointing, yet there can be no doubt that, with increasing experience, this therapeutic method will come more and more into favour. At present it is probably employed far more in this country, thanks to the inspiring work of Sir A. E. Wright, than on the Continent or in America, and in certain diseases its therapeutic efficacy has been completely demonstrated. The limits to its usefulness are determined largely by the limits of our knowledge of specific bacteria, and by the fact that in most infections the invading microbe has already made good its lodging in the tissues before the physician can employ the remedy.

It must be noted that Pasteur's vaccination against hydrophobia does not differ in principle from the vaccination by means of known organisms. In this case the virus is unknown, but the infecting agent is certainly contained in the emulsions of spinal cord employed in Pasteur's process. Further, the similarity of the attributes of the virus of epidemic anterior poliomyelitis to those of the virus of rabies gives rise to the hope that the physician will before long be placed in the position of being able to command a method of vaccination which may serve to restrict the spread of an epidemic; although the effects of the virus of poliomyelitis are so much more rapid than those of the rabies virus that it is probably too much to hope for a therapeutic agent.

Bacterial Vaccines are employed (1) as prophylactic and (2) as therapeutic agents.

1. **PROPHYLAXIS** in this country has been practically confined hitherto to the single case of *typhoid fever*, for the other two diseases in which a measure of success has been attained—*plague* and *cholera*—are, fortunately, of rare occurrence. Experimentally, however, it is known that a considerable degree of immunity can be conferred on animals to staphylococcal and streptococcal infections, and it is probable that where there is a decided risk of such infections—e.g., in operations about the mouth and nose—that the practice of the prophylactic use of staphylococcal and streptococcal vaccines will become more general. Again, the prophylactic use of vaccines for recurrent influenza, common colds, and recurrent bronchitis, has been advised; but the difficulty in these cases is that it frequently happens that the affection is caused by different micro-organisms on successive occasions, and while a certain degree of immunity has been procured against invasion—e.g., by the pneumococcus—the next attack is due to the micrococcus catarrhalis, the bacillus of Friedländer, or the bacillus of influenza.

In children, therefore, who are likely to be exposed to the risk of infection with typhoid fever, it is advisable to recommend that they should receive prophylactic doses of typhoid vaccine. The doses usually employed for an adult are a first dose of 500 millions of the dead bacteria, followed ten days later by a second dose of 1,000 millions. The first dose is usually attended by a considerable reaction, swelling and redness at the site of injection, headache, fever, and malaise, lasting twenty-four to forty-eight hours; the second dose usually produces little or no reaction. For children aged six to twelve years the writer has employed doses of half these amounts. It must be noted that the inoculations should be made at such a time as will allow of an interval of a month before arrival in the country where the risk of infection is expected, since it is believed that before immunity is established there is a brief period during which liability to infection is enhanced. For this reason it is inadvisable to use prophylactic vaccination in the actual presence of an epidemic. In children from six to twelve years of age, the doses recommended for prophylactic usage before operation of *V. typhosus*

pyogenes aureus or *Bacillus coli communis* vaccine are from 250 to 500 millions, given ten days before the proposed operation; of *Staphylococcus pyogenes*, 50 to 100 millions. Of this proceeding the writer has no personal experience.

2. AS THERAPEUTIC AGENTS.—In the present wave of enthusiasm for vaccine-therapy there is probably no single disease in which the method has not been tried and in which success has not been claimed. In the writer's judgment, the failures are more numerous than the successes, but the method is still in a very crude stage, and it offers so much promise, and so little attendant disadvantage, that wherever the infecting organism can be isolated a vaccine should be manufactured and administered. Stock vaccines should be used in those cases where for any reason it is found impossible to isolate or cultivate the invading organism; but it is generally agreed that the effect of stock vaccines is less certain.

LOCAL INFECTIONS.—*STAPHYLOCOCCI*.—Undoubtedly the greatest success has been attained in chronic local infections, especially those due to the *Staphylococcus pyogenes aureus*. Boils and chronic staphylococcic skin infections treated by this method are cured with greater ease and rapidly than in any other way, and though there is a certain small percentage of failures, inexplicable so far, the successes outnumber these enormously. The list of these infections includes boils, ciliary Nephritis, postular acne, and sinuses left after operations which have become infected with this organism. The doses given are still largely a matter of individual judgment, but in the writer's opinion moderate doses of 100 to 300 millions are the best. A local infection of the skin of the leg in a child of three years, which had resisted the ordinary methods of treatment for several weeks, cleared up in about a fortnight with two injections of 300 millions. The doses should be repeated at intervals of a week, and it is advisable to give several doses after the healing of the infected area, to endeavour to secure the patient from a relapse. Of course, the ordinary cleanliness and avoidance of irritation must be observed during the inoculations. With regard to sinuses, it is important to remember that not infrequently there is more than one organism present, and that failure to secure dealing with a staphylococcic vaccine may be subsequently explained by the discovery of another organism—e.g., a streptococcus or *B. pyogenans* or *B. coli*—the presence of which in the first cultures was overlooked.

MIDDLE-EAR DISEASE is most often due to a streptococcic infection, but in some instances staphylococci and pneumococci are found, either alone or in conjunction. In such cases vaccines of the mixed organisms should be prepared and injected. The doses of pneumococci and of streptococci should not exceed 50 millions in the first inoculations.

GONOCOCCI.—Gonococcal vulvo-vaginitis is a very troublesome and persistent complaint in infants and young children. The results of vaccine-therapy in this condition are, in the writer's experience, disappointing, though some observers have reported good success. The initial dose should not exceed 20 millions, and in many instances even smaller amounts seem to be preferable.

BACILLUS COLI COMMUNIS.—Infections of the urinary tract with *B. coli* and its congeners are favourable cases, though it is doubtful whether vaccine-therapy ever succeeds in curing the disease. It is, however, certain that symptoms are relieved and the worst effects of the infection are avoided. Therefore, in patients who are not progressing, doses of the micro-organism, isolated from their urine,

should be given at weekly intervals, the doses beginning with 25 to 50 millions, and increased up to 200 to 300 millions.

B. DIPHTHERIA.—Vaccines have been employed with considerable success in those cases where the organism persists in the fauces during convalescence. The vaccine should be prepared from the patient's own organism, and the dose should be from 25 to 50 millions.

B. TYPHOUS.—The same treatment is applicable to the chronic local peritonitis which occasionally follows typhoid fever.

ACTINOMYCOSES.—One case of successful treatment of actinomycosis of the lung has been recorded. The patient was aged fourteen, and the vaccine was prepared from an agar culture of the streptothrix, the dose being approximately measured by suspending in saline solution quantities of the growth taken up by a platinum loop of known dimensions.

B. TUBERCULOSIS.—Koch's new tuberculin consists of dried tubercle bacilli, extracted and triturated with 20 per cent. glycerin, and is thus essentially a bacterial emulsion, freed from soluble toxins. It has been used very extensively, especially in the treatment of tuberculous glands, tuberculous peritonitis, and tuberculous skin affections. Observers are divided in their opinions as to the efficacy of the treatment, and also as to the dose required. The writer's own opinion is that in children there is little evidence of beneficial results, but that occasionally the use of the drug appears to benefit the course of the disease. Where it is thought desirable to employ it, the dose should, in his opinion be $\frac{1}{100000}$ milligramme. The injections should not be repeated oftener than once a week. As mentioned previously, "opsonic control" appears to him to be entirely fallacious; but some authorities of wide experience believe it to be advantageous.

GENERAL INFECTIONS.—CHRONIC STAPHYLOCOCCAL PYÆMIA is probably the most amenable to vaccine therapy of the generalized infections. The writer has treated two such cases with apparent good result, but the course of the disease is notoriously uncertain. The doses should probably be smaller than in the local infections—not more than 20 millions.

RHEUMATIC ENDOCARDITIS.—Vaccination with streptococci isolated from the throat of the patient have been tried, but the writer has not so far seen any evidence that the proceeding has any influence on the infection; though, again, it is impossible to deny that vaccination may have warded off some of the worst effects of the inflammation of the endocardium.

ACUTE INFECTIONS.—The application of vaccine treatment to acute infections is more recent, and it is impossible at present to give any indication as to the results. In a few cases of erysipelas, an acute localized infection, small doses of 10 to 15 millions of streptococci have seemed to be beneficial in some cases; but in two instances within the writer's experience—one of recurrent, one of migrating erysipelas of long duration—the injection of the vaccine was entirely without effect. The evidence in favour of the use of vaccines in pneumonia is fairly strong. Some writers have proclaimed that vaccines shorten the disease in a remarkable way. In children, however, pneumonia is so erratic a disease that any deductions drawn from a small number of cases is apt to be fallacious, and the writer knows of no large series treated by this means. The best results are, in his opinion, likely to

be attained in the prolonged cases of pneumococcic broncho-pneumonia in infants and in those cases in which resolution is long delayed. In such cases a vaccine, prepared if possible from the patient's own organism, should be given in doses of 5 to 25 millions, the smaller doses being employed in the more acute cases. If the organism cannot be isolated, a stock vaccine may be used.

In typhoid fever evidence is accumulating slowly that moderate doses—10 to 15 millions—have a beneficial effect upon the course of the disease; but the writer has no personal experience of this treatment in children.

Lastly, in the acute streptococcic generalized infections, small doses of the appropriate vaccine can do no harm; but it appears probable that in these cases there is more to be hoped for from the use of sera.

IV

FEEDING OF INFANTS AND CHILDREN

EDMUND CAULLEY

1. BREAST FEEDING.

2. ARTIFICIAL FEEDING.

1. BREAST FEEDING.

Until the eighteenth century babies were breast-fed by the mother or a wet-nurse. In this country up to the end of the fifteenth century suckling was continued for two to three years. By the end of the seventeenth century the duration of suckling was reduced to eighteen to twenty-four months, the child being weaned when all the teeth were cut. Shortly afterwards weaning was advised at eighteen to twenty months, and since then the period of nursing has been gradually reduced, until at the present day it is rarely continued, under medical advice, beyond nine months. If a woman is healthy and secretes milk, there can be no doubt that she should nurse her baby, especially during the first three months of life. The number of women supposed to be unable to nurse their children would be greatly reduced if they were prepared during pregnancy for this duty, and encouraged by their doctors and nurses to carry it out. As much consideration should be devoted to the question of weaning as to that of a vital operation. The child's future welfare, and even its life, may depend on the decision. And yet it is ludicrous, were it not so pitiful, to find an inquiry how trivial a cause or reason has influenced the decision in favour of weaning, sometimes even in cases in which the mother would have willingly continued nursing.

During the first few days after birth maternal nursing is particularly important. The milk, known as the *colostrum*, appears to contain antigens—substances which produce antibodies in the blood of the child, and render it much less liable to infection. These antigens disappear almost entirely from the milk in the course of a week or two. During the first few weeks of life the digestive functions are very easily upset, and, as it is always difficult to train a very young infant to digest artificial substitutes for human milk, the longer such feeds are postponed, the less is the risk of setting up gastro-intestinal disturbance. After the third month of life the risk is very much smaller. It is also claimed that the advantages of breast feeding are seen long after infancy in the more rapid growth of the child, earlier puberty, and lesser liability to gastro-intestinal disturbance. Even if the mother cannot suckle the child entirely, or for a prolonged period, it is almost invariably advantageous that she should do so to the best of her ability. From the maternal point of view it is advantageous, in that it conduces to complete involution of the uterus, sometimes lessens the chance of impregnation, and is both cheaper and easier to carry out. It creates in the mother a much greater sense

of responsibility in the management of her child, and is often remarkably beneficial to her general health and nutrition.

Failing a maternal milk-supply, that of a wet-nurse is the best substitute, and may be the only means of saving the child's life. Wet-nurses are expensive luxuries, by no means always reliable or available, and may have to be obtained from Paris. The milk does not necessarily suit the baby for whom it is required. Properly modified cow's milk is often safer, for it is more under control, and can be more easily altered in composition. Nevertheless, wet-nurses, by virtue of the fact that they supply human milk, are as a rule superior to other methods of substitute feeding, and it is unfortunate that in this country they are not more readily available. The fear that immoral or insubordinate propensities can be transmitted by the milk to the child is a mere superstition.

Preparation of the Mother for Nursing.—The more healthy the mother, the sounder and more healthy will be her child, and the more fitted will she be to nurse it. The care of the child should begin before birth, even before conception, and the mode of life of the expectant mother must be supervised. Her diet should be rather more liberal than at other times, and contain a good supply of lime salts, proteins, and foods suitable to maintain the regular action of the bowels. A little more milk, meat, and bread, will supply the nutriment needed by the growing foetus. Alcohol is unnecessary, and in excess may be injurious. Mild purgatives may be required. Regular walking exercise at a moderate pace should be taken morning and afternoon up to the end of pregnancy, unless rendered impossible by swelling of the legs or pain. Violent exercise, lifting heavy weights, jolting conveyances, and sea-sickness, must be avoided. Swimming is permissible.

Simple amusements and entertainments are valuable in distracting the mother's thoughts from her condition. Excessive excitement and emotion are deleterious. Relief from household and other worries is advantageous. All the hygienic factors of ordinary life are more necessary for the expectant mother. She must rest for at least an hour in the middle of the day during the later months of pregnancy, and have a good long rest at night. Her clothing must not interfere with the respiratory movements, nor exert pressure or friction on the breasts or nipples. The lower part of the abdomen can be supported by a broad strong girdle, or flannel bandage.

The breasts and nipples must be kept clean. The folds below the breasts should be dusted night and morning with equal oleate, zinc oxide and starch, or other drying powder. For three months before confinement the nipples must be washed daily with warm water and massaged with cacao-butter, lanoline, or pure vaseline. Astringent and spirituous lotions are injurious, for they harden the skin and make it liable to crack. Small and retracted nipples can be increased in size and drawn out by careful manipulation, and rendered more prominent by the gentle use of Bier's suction-cups. Swollen, prominent nipples must be protected by absorbent salicylic wool, frequently changed if there is much overflow of milk. By such general and local measures the mother will be fitted to bear a healthy child, and to bring it up on the breast.

The Mother or Wet-Nurse.—The perfect mother or wet-nurse is twenty-five to thirty-five years of age, healthy, active, intelligent, truthful, placid and equable in temper, unemotional or well controlled, cheerful, good-natured, and affectionate. She should have had one healthy child and experience in the mode of management. Many previous pregnancies in rapid succession impair the general health and quality of the milk. She should be temperate in food and drink, though not

necessarily a total abstainer. Above all, she must have plenty of patience, and must realize that for the time being nursing is her paramount duty, and that other duties and amusements must be put on one side if they interfere with the proper performance of her functions as a nurse.

On examination, she should present the appearances of perfect health and good nutrition. There is no advantage in being fat; some thin women are most excellent nurses. The teeth should be good, and free from caries and pyorrhea; the tonsils small, and not pitted; the appetite and digestion excellent; and the bowels open daily without the aid of medicine. She should be in the habit of taking daily exercise and sleeping well. Examination of a wet-nurse should reveal no evidence of past or present constitutional disease, more especially tuberculosis, congenital or acquired syphilis, gonorrhea, epilepsy, enlarged glands, and former rickets. She must not be pregnant nor menstruating when first employed. The mouth and throat must be carefully examined, and the skin and hair, to exclude rashes and vermin. The physical examination must never be omitted, but it is unnecessary to apply the tuberculin test and the Wassermann reaction. The nurse's child, if living, must be examined for evidence of disease, general nutrition, and cleanliness. It should be at least four to six weeks old, since congenital syphilis may not be evident before that age. It should be under six months of age, as the mother of an older child may not yield a good milk; but it is not necessary that it should be of the same age as the infant for whom the nurse is required. Take care that another child is not substituted for examination in place of an unhealthy one. Chemical and microscopical examination of the milk is not necessary.

The breasts vary in size, shape, and structure. Those of a primipara are generally conical or pyriform, not always large, and firm to the touch, with prominent nipples. In a multipara, and sometimes in a primipara, they are pendulous. In 80 to 85 per cent, one breast is larger than the other. The size is no measure of functional capacity, for the glandular structure may bear a small proportion to the amount of fat. A large, fatty breast diminishes little in size during suckling, whereas a well-developed gland becomes smaller and less tense. Even this is not an absolutely reliable test of functional value, for in some women the secretion of milk is only active during the process of suckling. The best test of quantity is to weigh the child on several occasions, before and after nursing. The best test of quality is the state of nutrition of the nurse's baby and the characters of its stools. The nipples should be prominent and erectile. Sometimes they are too large; more often they are too small, or flat and retracted to such an extent that suckling is a mechanical impossibility.

The *mode of life* must be regular and placid. Strict personal hygiene and a certain amount of exercise or physical work daily are essential. The diet should be generous, nutritious, and easily assimilable. Excess of food, especially indigestible food, strongly aromatic and highly seasoned foods, and strong tea, are to be avoided. Wet-nurses are often overfed. Having previously been in reduced circumstances, on a plain or insufficient diet, as soon as they get the chance, they drink too much milk, eat an excess of meat, and often take an undue supply of stout or other malt liquor, on the plea that it is essential to a good milk-supply. The usual result is that the quality of the milk becomes such that it upsets the digestion of the child. Many mothers take too much milk and nitrogenous food, which, combined with lack of exercise, leads to the secretion of milk rich in solids, and to indigestion in the child. During the early days of lactation, while the

mother is in bed, the diet should be light, digestible, and not too nutritious. After she is up, an extra pint of milk and the ordinary three meals in the course of the day are generally quite sufficient. Bread and milk, gruel, or cocoa, may be taken at bedtime. Fruit and vegetables can be given freely to counteract constipation resulting from an excess of milk. Assuming that the baby is obtaining a litre of human milk containing 20 grammes of protein, an extra 60 grammes of protein will be required in the nurse's diet. The surplus carbon is available for the formation of fat. As a general principle, alcohol is unnecessary. One or two glasses of burgundy or port, or $\frac{1}{2}$ to 1 pint of light ale or stout, may be taken daily by women who are accustomed to it. A small amount of malt liquor often assists digestion and assimilation. Personal tastes in diet must be consulted.

The wet-nurse's child, if alive, must be properly cared for. A moral obligation rests upon the employer not to allow the child to suffer whose mother he is paying to deprive it of its natural food, and the nurse should be free from anxiety. The mortality among children, especially under three months of age, is enormous. Therefore choose a nurse whose child has reached the fourth month of life, or has died from misadventure at an earlier date. If the nurse's milk-supply is superabundant, it must be drawn off or given to another child, so as to prevent stasis and loss of function. Under such circumstances allow the nurse to suckle her own baby, either at the beginning or at the end of the nursing period, according to which portion of the milk is wanted for the foster-child. A weak or new-born baby is extremely unlikely to be able to empty the nurse's breast.

The nurse must not be allowed to go home, for, apart from the risk of conveying disease, all control over her diet and habits of life is lost. She must be under constant supervision, lest she attempt to supplement a deficient milk-supply by cow's milk or other food, or to soothe with opiates an infant irritable from indigestion or insufficient food. The child must be watched, to see that it is not overfed, and the stools examined daily. A wet-nurse is always an experiment in feeding. If the infant is extremely weak or delicate, it may be less dangerous to rely upon carefully regulated substitute feeding, for there is no guarantee that the wet-nurse's milk may not be unsuitable, and even dangerous. The employment of a wet-nurse for a congenitally syphilitic baby, or one suspected to be thus infected, is inadvisable; for should the nurse contract the disease from any other source, she would be sure to ascribe it to infection from the child. In the Foundling Hospital at Moscow, Ginsburg noted that the wet-nurses in the syphilitic ward did not get this disease; and the general experience of physicians is that its infectivity is extremely slight in this stage. On the other hand, the presence of the *treponema pallidum* in the lesions of congenital syphilis renders it probable that the possibility of infection exists. If parents obtain a wet-nurse without the advice of the doctor in charge of the case, he should warn them of the risk they incur of an action for damages, if the nurse became infected; but it would be a breach of professional confidence for him to state the nature of the case to the nurse.

Lactation.—The secretion of milk is due to the metabolic activity of the secretory cells of the mammary gland, not a mere process of filtration. These cells manufacture the milk from the nutrient materials in the blood and lymph. Their metabolism is controlled and modified by the nervous system, and is influenced by the nature of the food-supply. An increase in one particular foodstuff does not necessarily produce an increase of the same constituent in the milk. Thus, fat in the milk is not derived invariably, if at all, from fat in the food. Carnivorous

animals secrete a milk rich in fat, and cows at grass yield much more butter-fat than can be accounted for by the fat in their food. As a general rule, the secretion of fat in the milk is increased by proteins, and diminished by fats, in the diet. Protein increases metabolic activity, whereas fatty food diminishes it. Possibly some of the fat is taken up by the gland cells from the blood directly.

Caseinogen and lactose are not found in the blood, and must therefore be formed by the metabolism of the mammary cells. Lactalbumin is closely allied to serum albumin of blood, but differs in several respects, and, if formed from it, is modified by the cells. Disturbance of normal secretion causes an increase in the percentage of lactalbumin and a decrease in that of caseinogen. Further, lactose is present abundantly in the milk of camivores, although little carbohydrate food is ingested.

Active lactation generally begins on the third or fourth day after confinement, and increases as the child grows older. In the later months of pregnancy milk can often be squeezed out in jets from the nipple, and may overflow without such pressure. That secreted in the first three days after delivery (*colostrum*) is scanty, laxative, and nutritive. Lactation lasts a variable period in different races, families, and individuals. A family tendency to normal lactation is undoubtedly transmitted. Unfortunately, the advances in the dietetic management of infants of women unable to nurse their babies is leading to the existence of a still larger number of mothers who are either unable or unwilling to do so. It may be laid down as an axiom that, when possible, the mother should nurse her baby for nine months, and wean it in the course of another month. The duration of nursing may be prolonged or diminished in accordance with the factors further discussed in the management of breast feeding.

The General Composition of Milk.—Milk contains the *præformæ principia* of diet—viz., proteins, fat, carbohydrates, salts, and water—in proportions which vary in different animals and in the same animal at different times and under different conditions. The proteins consist of caseinogen and albumin. Probably there is a small amount of hæmoglobin as well. The terms "caseinogen" and "casein" are often used to mean the same protein body. Halliburton's caseinogen is the substance which is converted into casein by the action of rennet ferment. It is identical with the casein of German writers and the "free casein" of Van Slyke and Hart—that is, it is equivalent to calcium casein or bicaseinate of calcium, for it is always combined with calcium. The casein of Halliburton is the same as the paracasein of the Germans.

Caseinogen combines with acids and alkalis in different ways which are not yet thoroughly understood. Possibly the reactions are of the nature represented in the following table:

CASEINOGEN COMBINATIONS.

Reagent.	Product.
1. Lime-water	Calcium caseinogenate (basic calcium casein).
2. Potassium or sodium hydroxide ..	Potassium or sodium caseinogenate.
3. Sod. bicarb.	Calcium caseinogenate and sodium caseinogenate.
4. Sod. nitrate	Sodium caseinogenate and calcium nitrate.
5. Dilute hydrochloric acid	Caseinogen hydrochloride.
6. Lactic acid	Caseinogen lactate (butter-milk); and casein lactate, if the acid is in excess (sour milk).

(All these products are coagulated by rennet.)

7. Rennet	Casein (paracasein), or curd, is a faintly acid medium; as used in an alkaline medium, curd the alkali is neutralized.
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It is probably due to such variations in the chemical changes that cow's milk is rendered suitable for particular infants. More observations are needed before insisting on the necessity for these refinements in somelacture.

The fats, lactose, and salts, vary to a less extent in different animals, though it is probable that, apart from differences in percentages, there are slight differences in composition.

THE DIETETIC NEEDS OF THE CHILD.—Proportionately to its weight, a child requires more food than an adult; in order to provide for growth and development, as well as for energy in the form of work and heat, the supply of secretions, the repair of waste, and the maintenance of general nutrition. Proportionately to weight, the superficial area of a baby is quite three times greater than that of the adult, and, seeing that more than two-thirds of the loss of heat is due to conduction, radiation, and evaporation from the surface, it is obvious that a relatively greater supply of heat-producing food is required. Moreover, in the first few months of life the heat production is deficient from lack of muscular activity.

The value of a diet can be estimated according to its caloric value, a large calorie being the amount of heat needed to raise 1 kilo of water 1°C . The number of large calories required per kilo of body-weight is called the *energy quotient*. The caloric value of 1 gramme of protein or carbohydrate is 4.1, and of fat is 9.3 (Biknes). These figures are obtained by combustion of the foodstuff in a bomb calorimeter, and it must not be forgotten that the caloric value of the food ingested is somewhat less, on account of imperfect digestion, absorption, and assimilation. Accepting these figures as a basis, the caloric value of an ounce of human milk is about 19, and of cow's milk about 20. One hundred calories per litre is equivalent to 45 per pound. From these figures the necessary calculations can be made. They are interesting, but not of much use in practice. An infant requires per kilo daily 100 calories during the first three months of life, 90 to 100 calories during the second three months, and 80 to 90 calories during the second six months. An adult of 70 kilos weight (11 stone) requires 30 to 35 calories per kilo when living an inactive life. An adult has a much smaller body-surface, and gives off only 10 calories of heat per 1 pound weight, whereas the infant gives off 30 calories. As it gets older the relative body area decreases, and the number of calories needed is less. For example, a 7-pound infant would require 210 calories. Add one-fifth, to allow for growth, and the total daily need is 250 calories, which would be supplied by about 12 to 13 ounces (300 to 400 c.c.) of human milk. Atrophic and premature infants may need 120 calories, or even more, per kilo.

Protein food supplies the nitrogen which is essential to the structure of protoplasm and every body cell. Waller has pointed out that, in proportion to weight, the infant requires more than the adult; but in proportion to body-surface the amount is approximately the same. An adult weighing 67 kilos takes about 100 grammes of protein daily. A six-months-old baby, one-tenth of the weight, takes a litre of milk, containing about 20 grammes of protein. Two grammes per kilo of body-weight is sufficient. A growing child requires relatively to its bulk more than an adult, in order to provide for growth. The development of young animals depends to a great extent on the percentage of protein in the milk. An infant doubles its weight in five months on a 1 to 2 per cent. protein diet; a calf doubles its weight in one to two months on 4 per cent. Possibly the caseinogen is the protein essential to growth, and the albumen merely nutritive. Vegetable

proteins, though free from crystalline extractives, are neither as valuable nor as digestible as animal proteins.

An excess of protein is inadvisable. Apparently nitrogen absorption varies little from day to day, and is much the same at different ages, although the intake per kilo varies. About one-third of the nitrogen in the food is retained, and this proportion does not appear to be affected by the percentage of either protein or fat in the diet. In high protein diets more nitrogen is absorbed and eliminated, more than half of it in the urine, and therefore more work is done by the infant. The excess is liable to set up indigestion, colic, and constipation, or simply loss in weight. Excess of uric acid in the urine, gravel, and renal colic, may occur. The stools are green or yellow, and contain curds if caseinogen is in excess.

Deficiency of protein food renders the child anæmic, listless, weak, and short of breath on exertion. The muscles are flabby and soft, dentition is delayed, growth ceases, and rickets may develop. In breast-fed infants and those on whey, the low percentage of protein may lead to the appearance of curds in the stools, disappearing when more protein is added to the diet. In these cases the protein is chiefly albumin, and the curd is either due to the abnormality in the relative proportions of fat and protein or to the formation of insoluble curd by the uncombated acid in the stomach. Caseinogen with a dibasic acid forms a loose precipitate completely soluble in solution of sodium chloride, but with an excess of acid an insoluble precipitate is formed. With a low percentage of protein there may be an excess of free acid in the stomach.

Fat is chiefly of value for the maintenance of body-temperature. There is no proof that it is stored up directly in the tissues from the food, but the stored-up fat is drawn upon if the diet is deficient in this respect. There is evidence that fat aids the absorption of inorganic salts, especially earthy phosphates, from the alimentary tract, for more of these salts are found in the stools if the diet is deficient in fat. Fat diminishes the secretion of gastric juice, lessens gastric peristalsis, and delays protein digestion. In the stomach it is partially split up by the gastric juice and the enzyme *lipase*; in the duodenum it is split up into fatty acids and glycerine. It is well absorbed, but there is often more in human milk and other diets than required, and from 4 to 5 per cent. escapes digestion, and appears in the stools in the form of fatty acids, neutral fats, and soaps.

Excess of fat interferes with the gastric digestion of proteins, and may set up "fat dyspepsia" or "fat diarrhoea." *Fat dyspepsia* causes frequent vomiting of curdled milk and mucus, with the odour of rancid cream, due to butyric acid. The vomiting comes on half to one hour after food, and the fat may resemble lumps of casein. In *fat diarrhoea* the stools are acid, loose or pultaceous, often large, yellowish-green or yellowish-white in colour, greasy, and mixed with much mucus. Colic, flatulence, intestinal catarrh, and loss of weight, are generally present. Other effects of excessive fat are the refusal of food; capricious appetite; constipation, with pasty, foul stools; ammoniacal urine; irritability; and possibly convulsions. Such children are often excessively fat, have an enlarged liver, and are apt to develop post-anæsthetic vomiting if operated on for even trivial ailments.

Deficiency of fat impairs digestion, causes constipation and the appearance of earthy phosphates in the stools, and leads to malnutrition and rickets. Possibly rickets is due to interference with the absorption of lime salts in the form of soaps. A deficiency of fat cannot be successfully replaced by additional carbohydrates.

Carbohydrates are a source of muscular energy and heat. Infants practically never suffer from a deficiency of this food, for the percentage in human milk is high, and varies within small limits. Sugar is constantly added to the diet of artificially-fed babies. Carbohydrates are divisible into monosaccharides (e.g., dextrose); disaccharides (e.g., lactose, maltose, and saccharose); and polysaccharides (e.g., starch and dextrine). Probably all are split up into monosaccharides in the intestine by the different ferments always present. Dextrose is the one of chief value; yet, according to Rouse (1911), grape-sugar is clinically the least suitable for infants.

Excess of lactose in human milk may render the baby *anally* fat. Excess of saccharose in condensed milk generally makes the babies fed on it fat, flabby, and rachitic. They suffer from dyspepsia, intestinal disturbance, and fermentation of the food in the alimentary tract, with flatulency, vomiting, thin and offensive stools, loss of weight, and sometimes fever.

Either lactose or saccharose, as cane-sugar or beet-sugar, is added to the diet of many bottle-fed infants. The comparative value and defects of these two carbohydrates are difficult to estimate. Commercial milk-sugar is often impure. It is not certain that it is identical with human lactose. It is converted into lactic acid by the lactic acid bacilli, of which there are several varieties, so perhaps there are several kinds of lactic acid. This acid acts on caseinogen in milk outside the body, and forms fine curds of caseinogen lactate, as in *butter-milk*. But if there is much of the acid in the milk outside the body or in the stomach a waste indigestible curd of casein lactate is formed. It may therefore cause indigestion. Weigert (1909) states that the use of 10 per cent. lactose solution has no influence on the weight curve, is no better than barley-water, and does not improve digestion. It also promotes the development of gas-forming bacteria, but does not seem to give rise to intestinal fermentation to the same extent as saccharose.

Cane-sugar is cheaper than lactose; beet-sugar still cheaper, but not as sweet. On account of their sweetness, they cannot be given as liberally as lactose. Given in proper amount, there is no objection to the use of cane-sugar in preference to milk-sugar. The digestive disturbances induced by its use are due to excess rather than to its chemical composition.

Maltose, as a substitute for lactose, is sweet and laxative. The percentage composition of malt extract is—Maltose, 50; soluble starch, 10 to 15; protein, 5; water and ash. It contains a diastase ferment which is useful in converting starch into sugar, if a cereal decoction is used as a diluent. Malt extract is given in the same quantities as milk-sugar, and is rather expensive—about three shillings per pound. Pure honey is as suitable, containing about 75 per cent. of invert sugar, and costs about ninepence per pound. Less of it is needed. Starchy foods are considered later.

Ferments of various kinds are present in milk, but their nature and functions are not accurately understood. Possibly the antiseptic action of fresh milk is dependent on a ferment. Unfortunately, the activity of the ferments is destroyed by heat.

Salts.—Many salts are present in milk and other foods, and, although in small amount, are of vital importance to the organism. According to Bangs, the percentages of salts in the ash of the newly-born animal are, with certain exceptions, practically the same as the percentages in the ash of the mother's milk. The exceptions are sodium, potassium, and iron. Animals can live on milk, but they

die if the salts are extracted, even if they are again added to the food after their extraction. This is an important fact, as it shows that the tissues are unable to utilize salts unless they are supplied in the form of organic compounds of protein. Some of the evil effects ascribed to a deficient protein diet may be due to an insufficiency of assimilable salts.

Potassium salts are present in greater, and sodium salts in smaller, quantity in the ash of milk than in that of the new-born animal. As the animal grows, there is a relative increase in the muscles rich in potassium, and a decrease in the cartilages rich in sodium. The maternal milk is of an appropriate composition to satisfy these requirements. There is more sodium chloride in cow's milk than in human milk, so the addition of salt to modifications of cow's milk is not necessary. It is somewhat advantageous in delaying rickett cordling, and making it less complete. Salt stimulates appetite and thirst, increases the secretion of hydrochloric acid, and assists digestion. When vegetables are taken, it is required for the neutralization of the excess of potassium salts present. Hence it is a useful addition to barley-water. Rice contains remarkably little potassium.

The percentage of lime in human milk is 0.0245; cow's milk, 1.51; and yolk of egg 0.38. Meat, cereals, and leguminous, contain very much less. The addition of lime-water to milk is unnecessary, if it is given with a view to supplying lime, for it contains less than cow's milk, and lime is probably only absorbed as an organic compound. The prolonged heating of milk makes the lime salts insoluble. Phosphates of lime and magnesium are most important for cell growth and bone formation.

The percentage of iron is much less in milk than in the ash of the new-born. Apparently the young animal stores up iron in the liver before birth, the iron being used up as the animal grows. The chlorosis of girls is possibly due to the storage of iron in the liver in preparation for pregnancy and for the supply of iron to the fetus. Dilution of cow's milk reduces the percentage of iron below that present in human milk, and may lead to anemia and debility. To counterbalance this, organic preparations of iron may be given, such as yolk of egg, raw-meat juice, and some of the proprietary preparations made from blood. The iron in hemoglobin is more firmly combined than that in the nucleo-albuminous compound in the yolk of egg. After the first year of life other iron-containing foods should be given. Cabbage and spinach contain a high percentage, but spinach has the disadvantage of being rich in oxalates.

IRON IN FOODS: MILLIGRAMS PER 100 GRAMMS OF DRIED SUBSTANCE.

(*The Hospital*, May 16, 1908.)

White bread	1.4	Potatoes	0.2
Sweet apples	1.7	Green peas	6.8
Pears	2.2	French beans	5.5
Cow's milk	2.3	Cassios	8.9
Goat's milk	2.5	Lentils	9.3
Brewer's yeast	2.5	Asparagus	16.5
Red currants	2.6	Yolk of egg	18.5
Rice	4.5	Green chesney	22.0
Barley	4.7	Cabbage	30.5
Black grapes	5.8	Spinach	40.0

Phosphorus is of value in the formation of bones, the nutrition of the nervous tissues, and perhaps in the prevention of rickets, scurvy, and polyneuritis. It has been estimated that a man requires 2 grammes daily. Yeast, orotate extract, and

pastures, are rich in phosphorus; cereals, leguminous, and potatoes contain considerably more than human milk; lean beef, yolk of egg, and cow's milk, contain six times as much. Lecithin and nuclein, rich in phosphorus, are present in considerable amount in brain and nervous tissues. Though it is uncertain whether they are digested and absorbed, it is harmless to give calf's brain and the hard roes of fishes to children. Nuclein is present in cow's milk, but Koplik states that it is not assimilated by the infant. Additional phosphorus is best procured as yolk of egg.

Sulphur is present in cereals, leguminous, potatoes, and certain fruits—*e.g.*, cherries and peaches. Lettuce and beets contain a high percentage. Its importance in the economy is not thoroughly understood.

The Management of Breast Feeding.—The babe should be put to the breast in six to twelve hours, as soon as the mother has recovered from the fatigue of labour. Stimulation of the nipples induces reflex uterine contraction, and lessens the risk of post-partum hæmorrhage. The child has a better chance of drawing out the nipples, if they are small and retracted, before the rapid swelling of the breasts, as lactation is fully established, makes it difficult or impossible to seize hold of them. A small amount of colostrum is obtained, and, besides being nutritive, acts as a laxative. No castor-oil, butter and sugar, or other purgative, is permissible. Such treatment may set up troublesome, and even fatal, gastric and intestinal disturbance. In addition, sucking stimulates the secretion of milk. In the first twenty-four hours the babe sleeps most of the time, and will only obtain $\frac{1}{2}$ to $\frac{1}{3}$ ounce in two meals; 3 ounces in four to six meals on the second day; and then 7, 10, 12, 14, and 16 ounces on successive days. Needless to say, these quantities are very variable. Pritchard, Carter, and Pitt, calculated, by weighing the infants before and after nursing, that the average amount obtained in sixty-one cases during the first ten days of life was $\frac{1}{2}$, $4\frac{1}{2}$, $4\frac{1}{2}$, $6\frac{1}{2}$, $6\frac{1}{2}$, $8\frac{1}{2}$, $8\frac{1}{2}$, $8\frac{1}{2}$, and $8\frac{1}{2}$ ounces. No other food should be given during the first three days of life. Colostrum is alone necessary. Lactation is not established until the third day, and often later. If food is given, sucking is less vigorous, and the stimulus to lactation is deficient. If the urine is concentrated and irritating, boiled cooled water, sweetened with saccharin, may be given. After the third day an insufficient milk-supply may be supplemented with a mixture of milk, water, and milk-sugar. It should be given alternately with the breast, or as a supplementary food after the child has been put to the breast and is not satisfied. If the child is weakly, premature, or has to be artificially fed from birth, begin with one or two teaspoonfuls of a 5 per cent. solution of milk-sugar, and gradually add small quantities of cream and milk.

Mode of Suckling.—The babe should be held partially on its side, with the head and back supported by the right or left arm of the mother, according as it is being fed from the right or left breast; and the mother must bend her body somewhat forward, so that her nipple falls easily into the infant's mouth. The breast should be steadied by the index and middle fingers of the disengaged hand, placed above and below the nipple. Pressure with these fingers enables the mother to prevent the child taking the milk too quickly, and to assist the flow by gentle pressure on the breast. During the first week the child is put to both breasts at each nursing, and after that to alternate breasts every other feed, unless it is weakly and the milk-supply scanty. It obtains the milk by compression of the sinus and the base of the nipple, not by suction, in the same manner as milk is obtained from cows

by milking. The mother should express a little of the milk first, to get rid of organisms present in the ducts.

Duration of Suckling.—The average child is allowed ten to twenty minutes for each feed. This time is modified by the strength of the child and its requirements, the rapidity of sucking, the quantity and quality of the milk, the frequency of feeding, and the state of the nipple. A healthy child, taking milk from a full breast, may nurse until satisfied. It should not be allowed to go to sleep with the nipple in its mouth. After the feed, the mouth, especially the corners, should be wiped, but the inside should be left alone, for fear of injuring the delicate mucous membrane.

Frequency of Feeding.—Give the breast every six hours on the first day, four-hourly on the second day, and then every two hours. Or let the infant be put to the breast every two hours while the mother is awake, during the first few days, or even every hour and a half if it is very feeble. The more frequent nursing in the first two days enables the child to obtain more colostrum, and stimulates the secretion of milk. As soon as lactation is fully established, put the child to the breast every two hours from 5 a.m. to 11 p.m. during the first month; every two and a half hours from 5 a.m. to 10.30 p.m. during the second month; and after that every three hours from 5 a.m. to 11 p.m. Some babies require feeding less frequently, but great circumspection must be employed in lengthening the intervals, and even more in shortening them. After the end of the fifth month it may be easy and advisable to feed the child every three and a half or every four hours—that is, reducing the number of feeds to six, and then to five, in the twenty-four hours. Now, although it is convenient and advantageous for the mother and nurse that the infant should be taught regular habits, and should learn to wake at 5 a.m. for its first meal, all babies are not so well behaved. It is a good plan to regard the second feed as the first feed of the day, and to allow the mother to nurse the child once before that and after the last feed at night. In no case should the interval after the last feed at night be less than four hours. Many infants are fed whenever they cry, on the assumption that they are hungry. Often the cry is due to indigestion, and the indigestible character of the milk results from too frequent nursing. Such a pain can be relieved by any hot fluid. Warm milk affords relief by virtue of its warmth, and induces fresh colic in a short time. Very feeble infants cannot go for six hours at night without food, and need one extra feed during this time; or perhaps they can do with two intervals of four hours each. In the later months of lactation the early morning feed may be omitted, if the child sleeps soundly until later. No hard-and-fast rule is necessary, or should be rigidly adhered to.

Regularity of Feeding.—Apart from the first feed in the morning, which may be given earlier than 5 a.m. if necessary, regular nursing is essential to successful nursing. If the intervals are irregular in duration, the quantity and quality of the milk vary considerably. Frequent nursing increases the protein content of the milk, makes the milk less digestible, and does not allow the stomach time for digestion and evacuation of its contents into the duodenum. The stomach rarely empties itself in less than two hours. Both the baby and the mammae should be trained to regularity. The intervals are calculated from the beginning of each feed and "by the clock." If the child is asleep, wake him up. If he is difficult to keep awake while nursing, shorten the duration of the feeds, so that in future he wakes up hungry at the proper time. If the interval is too long, he is too hungry,

suckle greedily, rapidly fills or overfills his stomach, and then regurgitates or vomits some of the milk, or gets indigestion.

If the mother is disturbed in the night by the child's cries, the temptation to give the breast is almost irresistible. When a nurse is available, the child should sleep in a different room from the mother; if not, in a cot by the side of the bed.

The Duration of Breast Feeding.—If the mother is strong and healthy, the milk-supply satisfactory, and the baby making satisfactory progress, suckling may be continued for nine months, and partial suckling for another one to three months. It should never be continued beyond the twelfth month, except under special circumstances, and rarely beyond the tenth month. Among the lower classes suckling is sometimes continued, generally partially, for two, or even three, years, with the idea of preventing another conception. Impregnation does not take place quite so readily during lactation as at other times. From 3.5 to 8 per cent. of suckling women conceive, although the catamenia have not returned. The reappearance of menstruation is the best indication that the mother may again conceive, and lactation has then probably little or no effect in lessening the chance of impregnation. Unduly prolonged lactation may give rise to headache, anemia, debility, muscular pains (the rheumatism of lactation), and increased susceptibility to disease. Anæmia, epilepsy, and insanity, are rare sequels. The child may suffer from a deterioration in the quality of the milk, a reduction in protein and total solids.

Most mothers can suckle their infants during the first three months of life. If possible, artificial feeding should be postponed until the fourth month, or later, when the child's stomach is more fully developed as a receptacle for food, its digestive powers are stronger, and the liability to infection is less. A maternal milk-supply can often be improved by alterations in diet, the mode of life, and the frequency of nursing.

Contra-Indications to Breast Feeding.—Both the mother and child have to be considered. General and local causes of ill-health in the mother may necessitate weaning. Lactation must not be regarded as a serious drain on the system, if the food-supply is satisfactory and nutrition well maintained. Often it appears remarkably beneficial to the mother.

Tuberculous women do not secrete good milk, but often nurse their infants satisfactorily. If there is active lung disease, nursing must be prohibited; if there is latent disease, it must not be continued long. Possibly the tubercle bacillus may reach the infant through the milk-supply; more probably it is acquired by direct infection, if there is actual lung disease. Past tuberculosis of glands, bones, or joints, does not contra-indicate nursing, provided the child gains weight regularly and the general health of the mother is maintained. Constitutional syphilis may impair the nutritive value of the milk, but the mother must nurse her baby on account of its debility, giving supplementary food if necessary. Immediate weaning is essential in acute diseases, especially those of the infective type.

The emotional temperament associated with a *neurotic heredity* is liable to cause alterations in the milk on very slight provocation. A neurotic heredity is also undesirable, in that the insanity of lactation may develop or melancholia ensue on prolonged nursing. Epilepsy is not a bar, if the mother is under observation, but injury to the child or an alteration in the milk may result from a fit. During

eclampsia, after the child's birth, the milk should be drawn off and discarded until the effects of the toxæmia in the mother have subsided. Albuminuria as a rule is no bar. If the "rheumatism of lactation" is not cured by a nutritious diet and tonic, the child must be partly or wholly weaned. In all doubtful cases nursing should be tried, and the effects on the mother and child carefully noted, for weaning must not be advised except after great consideration.

Fixure of the nipple may render suckling impossible. It is produced by abrasion or excoriation from want of cleanliness, constant moisture, too frequent suckling, or the use of astringent hardening lotions. Infection and suckling prevent the raw surfaces healing, and lead to adenitis. Preventive treatment consists in attention to the nipples during pregnancy, bathing with cold water before and after suckling, and rubbing in olive-oil or lanolin, or painting with raw white of egg. Treat excoriation with zinc ointment, or with balsam of Peru, dr. i., ung. sq. rose oil ung. rectificd. ad oz. i. For fissure, dry the nipple, and paint it freely with benzo acid gr. xx. ad oz. i. If this fails, touch it every other day with a finely pointed stick of silver nitrate; or wash the nipple with lot. ac. benzo. 5 per cent., anæsthetize with cocaine, paint on the fissure lot. argent. nitratis, 10 per cent., dry with absorbent wool, and paint on egg albumin. For the next few nursings use a glass nipple-shield. If the pain and bleeding from a fissure make suckling impossible, maintain the activity of the mamma by the systematic use of a breast-pump. Pain can be relieved by applying cocaine solution, 5 per cent., half an hour before nursing, the nipple being washed before the child is fed.

Lymphangitis, abscess and tuberculosis of the breast, prevent nursing. Even the breast-pump is not permissible, if there is mastitis. In early stages of mammary congestion, apply fomentations of hot oil, and massage gently from the periphery towards the nipple. It is painful at first, but milk soon flows freely and the child can be put to the breast. In later stages give a full dose of Epsom salts, and quinine, gr. ii., *sixtis horis*. Ung. belladonnæ, ext. bellad. and glycerine p.a., or a belladonna plaster, is applied locally. An incision is necessary if there is evidence of pus. Old mastitis is not a bar to nursing, but indicates lack of full physiological activity. Tuberculosis of the breast is rare. It takes the form of irregularly disseminated nodules, a confluent mass, or miliary tubercles, and sometimes leads to abscess and sinus formation. The axillary glands are enlarged, the affected breast smaller than normal, and the nipple often retracted.

The Effect on the Child.—The milk-supply may be defective in either quality or quantity. The only reliable evidence of an insufficient supply is a lack of gain in weight. Women anxious to suckle their babies rarely admit their inability to do so. Weighing the child before and after suckling will give the weight of the milk obtained by the child. Chemical and microscopical examination afford evidence of its quality. Observation of the child while suckling may show where the fault lies. If he sucks vigorously for a short time, and then drops the nipple with an angry cry, the quality is probably good, but the quantity deficient. If he nurses languidly and for a long time, the quality is probably poor and the quantity abundant. Occasionally both mother and nurse are deceived on account of the size of the breasts and the duration of suckling, although the baby is wasting rapidly. An insufficiency of milk can be supplemented by other food, and weaning is not indicated. Sometimes the milk is too rich in protein and sets up gastric disturbance or colic, disturbed sleep, fretfulness, crying, and abnormal stools. This can often be remedied by regulation of the mother's diet and mode

of life, less frequent nursing, or the administration of boiled water, or lime water to the infant immediately before it is fed.

The signs of satisfactory progress are an increase in weight, provided there is no oedema, satisfaction after food, absence of digestive trouble, normal stools, general contentment, and undisturbed sleep. A baby is not necessarily thriving because it is quiet and does not cry. The weighing-machine is not "a curse in the nursery," if the mother is instructed that babies do not always gain weight regularly, and that a loss in weight is not necessarily a sign of the approaching end.

HUMAN MILK.—A sample can be obtained for analysis by gentle suction with a breast-pump, or by means of a thin glass flask with a trumpet-shaped mouth, which is first filled with hot water; the water is poured out, the edges of the mouthpiece dipped in cold water, and it is then applied to the breast. As the flask cools the fall in pressure causes the milk to flow from the nipple. All apparatus must be thoroughly clean, and both the nipple and breast must be washed before it is applied. Take a portion of the milk from the middle of the nursing for analysis; that first poured out is watery and poor in fat, while that obtained at the end is rich in fat. Conclusions drawn from a single partial sample are not very satisfactory. It is very difficult to empty the breast with a breast-pump.

HUMAN milk is a thin, watery, bluish-white, sterile fluid with a peculiar taste and odour. It gives a faintly alkaline reaction with litmus, and an acid reaction with phenolphthalein. The specific gravity ranges between 1030 and 1035 at 60° F., and varies a little with the temperature. With a low percentage of fat and a high one of sugar it may reach 1042; with a high fat content it may fall as low as 1024. Milk containing 3 to 5 per cent. of fat and of average specific gravity may be regarded as good milk. These two factors are sufficient for a rough estimation of its quality.

The fat globules vary in size from 0.00015 to 0.005 millimetre in diameter. Their number is in direct relation to their size, averaging 5,000,000, and perhaps reaching 11,000,000 per c.c. The best milk contains a medium number of a medium size, and the worst a small number of small size. It is impossible to say from microscopical examination alone that the milk is of good quality. In general composition human milk resembles the mammary secretion of other animals; it contains ferments and anti-bacterial alexins, a yellow lipochrome, and minute quantities of nucleon, lecithin, cholesterol, and heurin. It does not curdle with rennet, unless a little hydrochloric acid is added, and then it only forms a fine, flocculent coagulum. It is by no means of constant quality. It varies in different women, in the same woman on different days and at different periods of the day, in the two mammae, and at different stages of each nursing. It is modified by the state of the health, mode of life, diet, exercise, menstruation, prolonged lactation, and other causes.

Colostrum differs from the milk of fully-established lactation. It is yellowish in colour, more alkaline, of higher specific gravity, and contains the colostrum corpuscles. These are large nucleated cells, epithelial in character, from the acini of the gland, enclosing granules and fat globules, but not yet disintegrated. Colostrum also contains lymphocytes, and mononuclear and polymorphonuclear leucocytes. The presence of these corpuscles indicates non-establishment or disturbance of equilibrium in the gland. If the mother is feverish they may be absent. They vary in number, and usually persist in the secretion for seven to ten days, disappearing more slowly in bad health, in puerperal affections, and in

a primipara than a multipara. They may reappear during ill-health and at the commencement of involution of the gland, and give rise to digestive disturbance in the child.

The percentage composition of colostrum is unimportant, since it is only a temporary secretion and may vary from day to day. The importance of the antigens or antibodies in it has been already mentioned. Colostrum is coagulable by heat, and may coagulate spontaneously. It contains a higher percentage of protein than normal milk, because of the residues present, and a lower percentage of sugar. The amount of fat and the size of the fat globules are variable.

The Clinical Examination of Milk.—Holt claims that by his method of examination he obtains results as exact as those obtained by the ordinary examination of urine. The method is based on the fact that the percentages of salts and sugar in human milk are nearly constant, that the percentages of fat and proteins vary, and that the average specific gravity at 70° F. is 1031. The specific gravity is lowered by an increased fat percentage, and raised by increased proteins.

Holt's apparatus consists of a small hydrometer graduated from 1010 to 1040, a pipette, and a glass-stoppered cylinder graduated in 100 parts and holding about 10 c.c. Half an ounce of milk is required. The specific gravity is taken by the hydrometer. To estimate the fat, the glass cylinder is filled by means of the pipette to the upper line exactly; it is then stoppered and allowed to stand at a temperature of 66° to 72° F. Generally the lower limit of the cream is sharply defined in that time, but if not, a further six hours may be allowed. Chemical examination shows that the ratio of fat to cream is very nearly 3 to 5, and for clinical purposes it may be so estimated. The protein content is then judged from the following table:

CLINICAL EXAMINATION OF MILK (HOLT)

	Specific gravity.	Percentage of Cream.	Protein (calculated).
Average	1031	7	1.5 per cent.
Normal variations . . .	1028 to 1029 1032	3 to 12 3 to 9	Rich milk. Fair milk.
Abnormal variations . .	Below 1028 Above 1032 "	Above 10 Below 3 Above 10 Below 3	About normal. Very poor milk. Very rich milk. About normal.

Many analyses of human milk have been recorded, showing great variations in the percentages of proteins and sugar, though comparatively little in the total of these substances. Some of the most reliable results are given in the following table. Meigs probably under-estimated the proteins and over-estimated the sugar. Elgood (1910) found the average of four cases was—fat 4.5, and other solids 8.75, per cent. Planche and Benda's analyses of 3,450 samples of milk of forty-six wet-nurses yielded an average of 3.4 per cent. fat; minimum 0.56, maximum 16.4 per cent. (1911):

THE PERCENTAGE COMPOSITION OF HUMAN MILK.

	Milgs.	Staples and Duffing.	Altimor.	Philly.	Carter and Rich- mond.	Leeds.	Average.
Protein ..	1.046	1.31	1.48	1.344	1.97	1.995	1.5 to 2.0
Fat ..	4.283	2.31	2.83	3.307	3.45	4.131	3.0 to 4.0
Sugar ..	7.467	7.01	6.22	6.303	6.35	6.030	6.0 to 7.0
Salts ..	0.181	—	0.17	0.192	0.28	0.201	0.2

Estimation of the relative percentages of caseinogen and albumin appears a matter of difficulty, for the variations are great, although in most cases the total protein content is about the average—*v.g.* :

THE RELATIVE PERCENTAGES OF CASEINOGEN AND ALBUMIN.

Observer.	Caseinogen.	Albumin.	Total Protein.
König ..	0.63	1.27	1.90
Hirt ..	0.63	1.50	2.13
Handrick ..	0.6 to 1.0	0.50	1.20
Lehmann ..	1.29	0.70	1.99
Tolmatschoff ..	1.28	0.34	1.62
Wyder Hirth ..	2.43	0.57	3.00

Although human milk may vary in quantity and quality, and in the relative percentages of its component parts, it must be regarded as normal if the infant takes it well, digests it satisfactorily, gains weight at a proper rate, and keeps in good health. Harrington found, in the milk of women whose infants were doing well, that the protein varied from 1.08 to 1.17, fat 2.02 to 5.16, sugar 5.68 to 7.30, and salts 0.12 to 0.21, per cent. More importance must be attached to the condition of the child than to the chemical or microscopical examination of the mother's milk.

Quantity.—The daily secretion of milk varies with the age and needs of the infant. It can only be estimated by systematically weighing the infant before and after each nursing for successive periods of twenty-four hours. To take one such weighing, and multiply the result by the number of feeds in twenty-four hours, may give a most inaccurate total. The amount obtained at the first morning feed, after the mother has had six hours' rest, is considerably greater than that yielded at subsequent feeds. A normal average amount at one to three months of age is 600 to 800 *cc.*, roughly 1 to 1½ pints, but this amount is often exceeded. It increases rapidly up to the end of the second month, and after that much more slowly. During the first two or three months, large babies take 15 to 20 per cent., and small babies 10 to 15 per cent., of their weight in milk; after that the proportions are smaller. Calculating from observations by Hæckner, Lamm, and Alfeld, the daily quantities are—

From the end of the first to fourth week ..	16 to 25 oz.
During the second month ..	20 " 30 "
From the third to the sixth month ..	25 " 35 "
From the sixth to the ninth month ..	30 " 40 "

The quantity varies with the appetite of the baby and the secretory power of the mother. It is no measure of the nutritive value. Some large babies thrive on remarkably little milk. Probably a high nutritive value is much superior to an abundant secretion.

Pritchard, Carter and Pitt obtained much lower figures by weighing babies before and after nursing at the St. Marylebone General Dispensary, the Kensington Infant Consultations, and the Queen's Hospital for Children. Their results are based on a large number of cases, whereas those of previous observers have been drawn from very few, and are given in the next tables. They show that there is a remarkably small increase in the amount of milk taken by the class of babies from which the figures were obtained. The babies at the hospital were ill and took less milk. Under the more favourable conditions of private practice the yield of milk is likely to be higher.

THE AVERAGE AMOUNT OF MILK CONSUMED BY A TEST FEED DURING THE VARIOUS AGES UP TO NINE MONTHS.

Age	Weeks.			Months.								
	2	3	4	2	3	4	5	6	7	8	9	
Marylebone figures	1.47	1.62	1.65	1.61	1.50	1.73	1.60	1.81	1.98	1.67	—	
Kensington figures	—	1.75	1.85	1.90	2.00	2.15	2.30	2.47	2.72	2.90	3	
Queen's Hospital figures	—	1.00	1.42	2.00	1.54	1.35	2.18	2.00	2.08	—	—	

THE AVERAGE AMOUNT OF MILK CONSUMED IN TWENTY-FOUR HOURS.

Age	Weeks.			Months.							
	2	3	4	2	3	4	5	6	7	8	
Marylebone figures	18.8	14.77	13.46	16.58	16.02	15.83	13.00	18.25	20.06	12.8	
Kensington figures	—	16.58	14.50	20.00	14.50	15.20	17.28	21.50	15.00	20.2	
Queen's Hospital figures	8.8	—	9.71	14.45	14.38	11.66	9.16	14.25	12.00	12.0	

The quantity of milk is increased by stimulation of the breast by faradization, the use of the breast-pump, suction by other infants, and massage for ten minutes three times daily; by massage of the abdomen in an upward direction; by the increased ingestion of fluid, preferably nutritious fluid; by extra food—e.g., malleine, senarose, and saratogen; by stimulants in moderation, and by drugs such as iron, arsenic, and strychnine. Of galactagogues, drugs which directly increase secretion, little favourable is known. Grisebach recommends tincture of cayenne

goat's rue (*Galaea officinalis*), 1 drachm five times a day, and tincture of the stinging-nettle in doses of $\frac{1}{2}$ to 1 ounce. Veterinary surgeons give infusion of aniseed freely, and apply it to the mamma as a fomentation. Thyroid extract and lactagol, an extract of cotton seed, are used. Injections of pilocarpin nitrate, $\frac{1}{2}$ grain, for a few successive days, may increase secretion or restore it when failing.

The quantity can be diminished by reducing the fluid ingesta and by giving saline cathartics. A full dose may entirely stop secretion for a time. It is rarely advisable to reduce the quantity. If the milk is poor in quality, it is better to try and improve its quality by measures which increase the percentage of solids. Preparations of belladonna or atropine, taken internally or applied locally, lessen the secretion. Camphor, 3 to 5 grains, taken three times daily for three days, may completely arrest the flow. Potassium iodide sometimes lessens it, and may induce atrophy of the breast. Fright or sudden shock may cause complete agalactia.

Variations in Quality.—Age has remarkably little effect. Menstruation may alter the milk to such an extent as to set up indigestion or diarrhea in the child. Usually a slight disturbance of digestion is the sole result, but occasionally the infant is seriously affected. Commonly there is no change in the milk and no effect on the child. Dyspepsia, colic and enteric catarrh, are more often coincident than due to this cause, and should be treated in the usual way, and not by weaning. Marked disturbance is generally due to an excess of protein, and should be treated by lengthening the intervals between the nursings, giving the child a little water or lime-water before nursing, and temperately modifying the diet and mode of life of the mother. Pregnancy does not affect the quality of the milk appreciably.

The solids in the milk, especially the proteins, are increased by increased protein diet and by increased frequency of suckling, with consequent indigestion and colic. An excess of nitrogenous food must be counterbalanced by an increase in the exercise taken. If indigestion is due to too frequent suckling, it is a simple matter to lengthen the intervals between the feeds.

The percentage of fat is increased by a liberal supply of protein food, malt extracts, and possibly alcohol. Vegetable diet diminishes the percentage of both fat and protein. The greater the yield of milk, the lower is the percentage of fat. A nursing mother must not fast. Deficiency of food is bad for her and for the child; her milk becomes weaker in protein and fat, and she has to draw upon her reserve stores of nutriment to provide even this unsatisfactory milk.

Alcohol is said to increase the proportion of fat, to slightly diminish the amount of sugar, and to have no constant effect on protein. A moderate amount is harmless, and none appears in the milk. As a general rule, alcoholic drinks should not be prescribed, for fear of setting up the alcoholic habit. In some cases a small amount will enable a mother to continue nursing, but to insist on prolonging it by means of considerable stimulation is bad for both mother and child. Good milk depends on good food, not on stimulants.

To convert a poor milk into a digestible rich one, order a liberal protein diet, and walking exercise morning and afternoon. More vigorous exercise, such as riding, cycling, and lawn-tennis, may be needed. The protein content is increased by increased protein diet, insufficient exercise, and frequent nursing, and is decreased by the opposite conditions. The fat content is increased by protein food, malt

extracts, and possibly alcohol, and decreased by deficient protein food, fasting, and excess of fatty food. The percentage of water is raised by increased fluid diet, and lowered by the lessened ingestion of fluids, profuse sweating, and saline cathartics.

Drugs taken by the mother may be partly excreted in the milk, and may affect the child. Such excretion is unreliable and uncertain. Citric and hydrochloric acids produce no change. Sodium salicylate, potassium iodide, iodoform applied externally, iodine injected intramuscularly, and colchicum, may all pass into the milk. Mercury is transmitted feebly and irregularly. Lead, arsenic, and antimony, have been excreted in this way. Opium and morphia have been known to produce deep sleep in the child when taken by the mother, so has chloral hydrate, while the effect of bromides is variable. Phenazone, phenacetin, urotropin, and possibly atropine and belladonna, may appear in the milk. Rhubarb, senna, and saline cathartics, may also be thus excreted, and may purge the child. Attempts to treat the infant through the medium of the milk-supply are foolish, and uncertain in their results.

WEANING.—The commencement of weaning should take place in the tenth month of life, partial suckling being continued for four or five weeks longer. Carried out gradually, the child's stomach and digestion become accustomed to the artificial food, and it is easy to ascertain the particular milk mixture which is suitable. During the first week one such feed is given at 8 a.m.; in the second week a second bottle is given at 8 p.m.; in the third week a third bottle is given at 2 p.m., or four bottles can be allowed daily; in the next week or two the remaining breast feeds are also replaced by the artificial food. The composition of each feed depends on the age of the child, and is described in the section on Artificial Feeding.

In sudden weaning the child is taken from the breast entirely, and put on a mixture suitable for its age. Generally it is advisable to begin with a somewhat weaker mixture. The child may refuse the food for some hours, or even a whole day, but will give in to the cravings of hunger if the mother persevere. Abrupt weaning may be necessary because the child refuses to take the bottle, or an artificial mixture, as long as he can get the breast.

It is advisable to begin weaning during cold weather rather than when it is very hot, and to postpone it if the child is actually teething. Weaning should be delayed if the child is very weak, and during or after an acute illness.

The indications for weaning are the same as those described as contra-indications to breast feeding (pp. 40, 41). Should the mother become again pregnant, the decision rests on several factors. If the child is gaining weight and is contented, and the mother's health is not suffering, suckling may be prolonged to the fifth, or even the sixth, month of pregnancy. The child must then be weaned entirely by the end of that month, and often at a much earlier date. If the baby is delicate or the weather very hot, suckling should be continued as long as possible, provided the progress is satisfactory. If the mother is delicate or the milk-supply insufficient, partial weaning may be begun at the end of the fourth month. The risk of reflex miscarriage being induced by suckling is so trifling as to be negligible, except in women particularly prone to miscarry. Conception is comparatively infrequent before the sixth month of lactation. At that age a baby can be weaned with little risk, if it is thought advisable for the sake of the fetus or the health of the mother.

2. ARTIFICIAL FEEDING.

THE artificial feeding of infants includes the consideration of the different varieties of milk and other foods which are given in place of, or in addition to, human milk. It may be necessary from birth or at any subsequent period, and is yearly becoming more generally adopted as the ordinary means of bringing up infants. No doubt artificial feeding, more or less in conjunction with breast feeding, has existed for an indefinite period. According to Ségala, Soranus of Ephesus, who practised in Rome during the reign of the Emperor Trajan, advised milk as the only food for the first six months. His directions for the bringing up of infants are somewhat similar to those of the present day. He recommends two baths a day, and that the water should be gradually reduced in temperature until cold water is used; the addition to the diet of crumb of bread or flour, dissolved in water or wine and honey, at six months of age; later on, soup, new-laid eggs, and bread soaked in wine and water; at eighteen to twenty-four months, less milk and more solids; and that an excess of linctus should be permitted at too early an age. *Farmaceuta* were usually added by Roman nurses after the fortieth day. Apparently the number of meals allowed was indefinite, the intervals irregular, and the food too indigestible.

In this country artificial feeding was begun in the eighteenth century. Water-pap, boiled bread and water, was given with human milk up to two years of age. Or baked flour was used in preference to bread. Such foods as beef-tea, broth, sugar, baked flesh, and various small birds, were given; also small light beer after the first year, and a little red wine when the child began to walk. Towards the end of the century milk was advocated as the proper substitute, and Underwood recommended that it should be boiled.

In the nineteenth century the duration of suckling became limited gradually to about nine months. The wet-nurse declined in favour and slowly disappeared, the sucking-bottle was evolved, and proprietary foods were produced. It is due to the evolution of a proper bottle, and the preparation of artificial foods under scientific conditions, that it has become possible to bring up infants successfully by artificial methods. Hence year by year, in spite of the opposition of the leading physicians in this branch of medicine, there is a steady increase in the number of hand-reared infants, and a steady increase in the number and quantity of proprietary foods. Of the various methods adopted, the use of cow's milk is the most general, for it is easily obtainable and reasonably cheap. We shall first consider its composition, its differences from human milk, and various methods of modification. Next we shall discuss the management of artificial feeding. And finally we shall consider the use of the milk of other animals, dried milk and other modifications of milk, and various proprietary foods.

Intolerance of cow's milk is exceptional. In rare instances even small quantities have induced vomiting and frequent liquid stools, sometimes rectal tenesmus, occasionally blood in the stools, fever, dyspnoea, and collapse. The acute symptoms are of short duration, and may be followed by prolonged spasmodic, with tetany and convulsions. Spasmodic has been ascribed to whey. Possibly this intolerance is of the nature of anaphylaxis. Finizio (1911) obtained a precipitin reaction in the blood with cow's milk in two such cases.

Cow's Milk.—In percentage composition cow's milk differs from human milk. In addition, it is crowded with micro-organisms when it reaches the consumer, and is a very suitable medium for their growth. On secretion it has an amphoteric reaction, turning litmus blue and tannic brown. With phenolphthalein it is acid. The amphoteric reaction with litmus is due to acid and alkaline sodium phosphates. Its specific gravity ranges between 1028 and 1035. Microscopical examination reveals extraneous substances, such as epithelial cells, particles of manure, hairs, etc. Its general composition is the same as that of other animals. Many ferments are present, and are readily destroyed by heat.

The total solids vary within considerable limits dependent on the breed of the cow, the period of lactation, and the nature of the food. The variation is in the fat content chiefly, for the other solids only vary between 8.5 and 11 per cent. The percentage of fat varies in different cows, in the same cow at different times, and during the same milking, according as the fore milk, middle milk, or stripplings, is taken for analysis. Each cubic millimetre contains in suspension from 2,000,000 to 3,000,000 fat globules. The fat is a mixture of neutral olein, palmitin, and stearin, and glycerides of palmitic, stearic, and oleic acids. Glycerides of volatile acids, chiefly butyric, caproic, and caprylic, are present in small amounts. Purin bodies, leithin, cholesterol, and mineral salts, are also present. Citrates are more abundant than in human milk, the amount being equivalent to 1.2 to 2 grammes per litre (Ostermeyer). The soluble bicitrate of calcium is converted into a less soluble tricitrate by boiling.

The average composition obtained by different analysts does not show nearly as great discrepancies as in the case of human milk. Protein is estimated by Kjeldahl's process, fat by Babcock's apparatus or Gerber's acido-butyrometer, and sugar by Fehling's process. The percentage composition is given below in the section in which cow's milk is compared with human milk.

The Milk Supply.—The milk should be that of healthy cows which do not react to the tuberculin test. It should be the mixed milk of a large number of cows, for such milk maintains a much more uniform standard of quality than that of one cow. The best milk-cows are Shorthorns, Ayrshires, Kettys, Devons, and Red Polls. The milk of Jersey, Alderney, and Guernsey cows is much richer in fat, but the fat globules are large, and the cows are more liable to disease. The milk of newly calved cows must not be used, for the colostrum corpuscles are likely to disagree. The milk of old cows, and of cows which have calved a long time previously, is apt to be thin, watery, and deficient in fat.

Cows should have a liberal supply of clean water, plenty of food, a ration of nitrogenous food, and comparatively little exercise. A diet of hay and oats, or grass, is the best. They must not be fed on fermented food, such as brewers' grains, nor allowed to drink stagnant water. Their pastures should be free from noxious weeds. The milk of cows fed on turnips and linseed or cotton-cake may disagree with infants.

Cowhouses should be light, well drained, scrupulously clean, and lime-washed, with roomy stalls and good ventilation, but not draughty. A temperature of about 60° F. is the most suitable. The bedding must consist of clean straw, peat moss, sand, or sawdust, fresh twice a day. The floor ought to be swept an hour before milking. On no account should the cows be hurried or frightened before being milked.

The cow must be kept clean, and, before milking, the udder and teats should be

gently washed with warm water and dried with a soft towel. The milker must wash his hands, wear a clean overall, and use a pail thoroughly scalded out with boiling water. The milk should be aerated by pouring it in a thin stream from cow to can in the open air, or by pumping in air with a suitable machine. It must be then cooled to 60° F., or under ideal conditions down to 40° F. If sent by rail, it ought to be put in cans fitted with lids to exclude dust, and in vans built on the principle of the refrigerator. It would be still better if the milk were cooled, bottled and sealed at the dairy in the country, and delivered direct from the station terminals to the consumer. The shorter the distance the milk has to travel and the fresher it is, the better for the infant. Even in towns it ought never to be more than twelve hours old. No chemical preservatives are admissible. Borax, boric acid, salicylic acid, and formalin, are the ones chiefly used. Hydrogen peroxide is harmless, except for enabling milk not properly fresh to be employed.

Boddé's Process of preserving Milk.—Add about 15 c.c. of a 3 per cent. solution of H_2O_2 to each litre. Heat it to 51° to 52° C. (124° to 125° F.) for at least three hours. The H_2O_2 is decomposed, and the nascent oxygen acts as an efficient germicide. The milk is then bottled, and will keep for eight to ten days in hot weather. There is no change in its flavour or appearance, and cream rises as usual. Non-spore-bearing organisms are destroyed.

Test for Boric Acid.—Put 20 c.c. of milk in a beaker, add a drop or two of phenolphthalein solution, and then drop in a solution of caustic soda of about normal strength until a faint pink colour appears. Pour the mixture into two test-tubes. To one part add an equal quantity of distilled water, to the other an equal bulk of a neutral 50 per cent. solution of glycerol in water. If boric acid is present, the mixture in the first tube will become pink, while that in the second will turn pale or white.

Test for Formalin.—If a few crystals of anisidol are sprinkled on slightly diluted normal milk it becomes pink or salmon colour. If formaldehyde is present it becomes canary yellow (Manget and Marien). Whey containing formaldehyde turns yellow on the addition of anisidol. Schiff's reagent (fuchsin decolorized with sulphurous acid), impregnated with sulphurous acid gas, is also used as a test. Precipitate casein and fat, and add the filtrate to the reagent; it turns pink if formalin is present. Formalin (40 per cent. formaldehyde) is added as a preservative in the proportion of one drop to each ounce of milk. It delays the action of rennet, gastric digestion, and the pancreatic digestion of fibrin and starch.

Test for Salicylic Acid.—Make an ether solution, evaporate it, dissolve the residue in alcohol, and add ferric chloride. A violet colour is produced.

CREAM.—The chief fraud in milk-dealing is the removal of cream. This raises the specific gravity, which is again brought down by the addition of water. To detect such a fraud, place 100 c.c. in a graduated vessel, or fill any glass cylinder graduated in 100 parts. Take the specific gravity. Let the milk stand for twenty-four hours, and read off the proportion of cream which has risen to the surface. If the specific gravity is within the limits of 1028 to 1035, and the percentage of cream not less than 9, it may be considered good average milk. The lower the specific gravity and the higher the percentage of cream, the less likely is the milk to have been tampered with.

Cream merely differs from milk in its percentage of fat. Gravity cream is obtained by skimming milk after it has stood for twenty-four hours. Centrifugal cream is that obtained by means of a separator. The latter is the fresher if it is

made from fresh milk. To a slight extent the emulsion is broken up by the centrifugal force. It contains from 40 to 50 per cent. of fat, but can be made of lower percentage. Much that is sold contains only 20 per cent. Proper centrifugal cream is separated from the mixed milk of many animals. The milk is filtered, cooled down to 40° F., pasteurized, centrifugalized, and again cooled to 40° F. The fat globules may become coherent and indigestible, especially if the bottles have not been kept cool or have been much shaken in transportation. Such cream gives rise to the vomiting of water, fat, and curd, successively.

Gravity cream is very variable in quality, and usually contains about 16 per cent. fat; sometimes it is merely rich milk or strappings. It does not keep well, and is often preserved by means of ice or chemical preservatives. It should be obtained from the mixed milk of a herd, not from a fancy cow. The milk should be allowed to stand at the same temperature, for the same length of time, and be skimmed by the same person.

Top Milk.—Because of the liability to variation in the quality of gravity cream and the richness of centrifugal cream, it is generally preferable to use the upper portion of milk into which the bulk of the fat has risen while the milk has been standing in the cold. Here again we are met with variations according to the quality of the milk and the duration of standing. Such estimations as the following may be quoted:

PERCENTAGE OF FAT IN THE MILK.

Duration of Standing	The Top—						Quantity
	Half.	Third.	Fourth.	Fifth.	Sixth.	Eighth.	
Six hours	8	10	12	—	14	20	Chapin.
Five to eight hours	7 to 8	—	10	—	—	—	Townsend.
Four to five hours	7	10	12	15	—	—	Holt.

Chapin's results were derived from a 4 per cent. fat milk. Holt also states that if a pint of mixed milk, freshly drawn and cooled rapidly, is stood for four hours, the upper 4 ounces contains 20 per cent. fat. For practical purposes we may reckon that the upper half of milk, which has stood four hours, contains 7 to 8 per cent. of fat. There is an advantage in having food modifications on unduly high fat estimations, in that babies do not digest high percentages of fat very well.

The Comparison of Cow's Milk with Human Milk.—Certain biological and chemical differences make it impossible to prepare from cow's milk a fluid identical with human milk, even though on analysis the two fluids appear the same. The proteins in the milk of different animals are not homologous, and their digestibility appears to depend partly on special biochemical characters. Although human caseinogen is essentially the same as that of cow's milk, and produces the same proportion of amino-acids, it is not coagulated by rennet. The proportion of caseinogen is much greater in cow's milk than in human milk. Caseinogen is precipitated more readily by magnesium sulphate, and with greater difficulty by acetic acid, from human milk than from cow's milk; and the curd formed by the addition of acid to human milk is finer, more flocculent, and more digestible. According to Biedert, cow's milk coagulates like human milk in the stomach if the ratio of fat to protein is the same. Schlossmann states that the coagulum is the same if albumin is

added. Certainly it is mainly a matter of dilution, for on the addition to cow's milk of four to five times its bulk of water, the curd obtained by the action of dilute acetic acid closely resembles that of human milk. This implies that there is four to five times as much caseinogen in cow's milk as is present in human milk. The following table shows the average differences between the two milks, and the relative proportions of the two proteins. In human milk caseinogen : albumin : : about 2 : 3. In cow's milk the proportions are about 4 : 1. My own analyses of cow's milk yielded an average of albumin 1.4 to caseinogen 2.6.

THE COMPOSITION OF HUMAN AND COW'S MILK.

	Human Milk.	Cow's Milk.
Protein	1.66 2	2.5 to 4.0
Caseinogen	0.6 .. 1.2	2.5 .. 3.7
Albumin	0.5 .. 1.2	0.3 .. 1.0
Fat	3 .. 4	2.5 .. 4.0
Sugar	6 .. 7	4.4

Apart from the protein differences, the other constituents are not identical. Much of the indigestibility of cow's milk is due to the fat, the globules being ten to fifteen times larger than those of human milk. The fat of human milk contains a higher proportion of olein, and less stearin, palmitin, and volatile fatty acids, and has a lower melting-point. There is a certain amount of evidence that the carbohydrates in the two fluids are not chemically, physically, and physiologically identical, though probably the differences are trivial and negligible. The inorganic salts differ in quality and quantity, Na_2O , CaO , MgO , and P_2O_5 being relatively less, while K_2O and Fe_2O_3 are more plentiful in human milk (L. Findlay, 1906). Possibly the good effects of dilution are due to a reduction in the percentage of salts present. Apparently they are injurious unless in organic combination with protein.

It is necessary to make use of an average standard composition of cow's milk and human milk as a basis for the preparation of a modified cow's milk as nearly as possible identical with human milk—e.g. :

AVERAGE STANDARD COMPOSITIONS OF MILK.

	Human Milk.	Cow's Milk.
Protein	2.0	4.0
Fat	4.0	4.0
Sugar	6.6	4.4
Salts	0.2	0.6
Water	87.2	87.0

The effect of adding water can be easily calculated. After such dilution the addition of fat and sugar are obviously needed. Unfortunately, on account of the differences in the proteins, mere dilution is quite incapable of rendering the proportions of these proteins identical, though the total percentage is the same. On the other hand, the baby's stomach can be gradually trained in the digestion of

artificial mixtures, so that probably too much importance is attached to these differences.

The Modification of Cow's Milk.—In the modification of milk it is advisable to make use of the simplest possible methods, and to avoid all unnecessary mechanical interference. Once the initial difficulty of obtaining a clean, superfatted milk of fairly constant fat percentage is overcome, it is easy to devise a suitable weak mixture and to gradually increase its strength. The top half of a good mixed milk, which has stood from four to six hours, contains approximately the percentages—Protein, 4; fat, 8; sugar, 4. A mixture of top milk, 5 ounces; lime-water, 1 ounce; sugar, 1 ounce; water, ad 20 ounces, has an approximate composition—Protein, 1; fat, 2; sugar, 6. By gradually increasing the proportion of top milk up to eventually 10 ounces, and reducing that of water, the total mixture remaining at 20 ounces, the approximate composition of the mixture finally reaches—Protein 2, fat 4, sugar 7, per cent. If the percentage of sugar is too high, add only 4 or 7 drachms to the mixture. Instead of adding sugar, a sugar solution of lactose, 10 drachms; lime-water, 1 ounce; water, ad 20 ounces, can be used; it contains 6.25 per cent. sugar. Adding the top milk to this solution, the first mixture contains about 6, and the final mixture about 5, per cent. sugar. If a higher proportion of fat is desirable, allow the milk to stand a similar length of time, and use only the top third or top fourth.

The home modification of milk according to this method makes it unnecessary to know the exact percentages of protein and fat in the milk used, provided always that the same amount of top milk is taken after the same period of standing at approximately the same temperature. From the effect on the child, as judged by the digestion, character of the stools, and the gain in weight, it is easy to decide whether the food should be made richer in fat or protein, or in both.

Laboratory Methods—The Percentage System of Feeding.—To secure accuracy of composition, to minimize the chances of contamination, and to provide a food suitable for a particular infant, milk mixtures are made up in milk laboratories by various companies according to the prescription of the doctor—e.g.:

A MIXT. PERSOENNEN.					
II. Casein	0.55
Albumen	0.75
Fat	2.50
Sugar	6.00

Amount per feed: 4 ounces. Number of feeds: seven. Heated at 167° F. for twenty minutes. Alkalinity: 5 per cent.

The component parts of such a mixture are derived from the efficiently cooled milk of cows, which ought to be free from tuberculosis. The cows are kept under hygienic conditions and carefully fed; and special precautions are adopted during milking for the sake of cleanliness, and subsequently in keeping the milk at a low temperature and free from contamination. Cream of definite fat percentage, fat-free milk, whey, 20 per cent. sugar solution, lime-water, and distilled water, are the materials used. Begin with a weak mixture, and gradually increase the strength or modify the percentages according to the results. Both the doctor and nurse are saved trouble. The milk is supplied in bottles containing enough for each feed, and merely needs warming. As a temporary expedient the method has certain advantages, but extensive trial has shown that it is not very satisfactory. It

would be particularly useful among the lower classes, who are unable to obtain clean milk, to keep it under proper conditions, and prepare suitable mixtures, were it not for the expense. For other parents simpler measures are generally successful and easily carried out. Many babies do badly on such milk, possibly because of its constant composition, lack of freshness, or deficiency in some constituent. Pasteurization is not an unmitigated advantage, as will be shown later, and may prove injurious. Occasionally deleterious organisms get into the milk in spite of the care taken.

The strongest argument against percentage feeding is that Nature does not provide milk of unvarying chemical composition. It has been shown that the mother's milk varies at different times of the day in either breast, and at different stages of each nursing. The milk first secreted is poor in fat, while that withdrawn last from the breast is almost pure cream. Hence the child may be said to get a series of meals of different composition. It is scientifically unsound to keep rigidly to definite percentages of the various constituents, and still more so to provide the same percentages in each feed. The repetition of meals identical in flavour, quality, and quantity, is absolutely opposed to Nature's method of feeding. The child's stomach is not a test-tube, nor is feeding merely a chemical experiment.

Concentrated milk of two strengths is supplied by *Clay, Paget and Co., Ltd.*, viz.:

1. Protein, 4; fat, 10.5; sugar, 12.5.
2. Protein, 4; fat, 8; sugar, 14.

From these suitable milk mixtures can be prepared by simple dilution. They are useful for young infants while travelling.

The Management of Artificial Feeding.—Successful artificial feeding depends on attention to details, suitable apparatus, and appreciation of the following factors:

1. The size of the child's stomach; the age and weight.
2. The quantity of food for each feed.
3. The number of feeds to be given in twenty-four hours.
4. The regularity of feeding.
5. The composition of the substitute food.
6. The mode of preparation of each feed.
7. The temperature of the food and the mode of administration.
8. The preservation of the food.
9. The cleanliness of all apparatus.

The Apparatus.—Although simplicity is to be aimed at as far as possible, it is convenient to have a refrigerator, two siphons, a pasteurizer or a proper milk-saucepan, thermometer, two to four feeding-bottles, bottle-brushes, absorbent and non-absorbent wool, straining muslin, a mixing pitcher, 8-ounce and 20-ounce graduated measures, a bottle-warmer, rubber teats, bicarbonate of soda, and loric acid.

Use boat-shaped bottles, as simple as possible in structure, with a glass stopper and valve at one end, and at the other a large mouthpiece for the attachment of a single rubber teat, which can be inverted easily. The valve is not essential, and rubber valves should be avoided. Bottles with long tubes are untrustworthy because of the difficulty in keeping the tubes clean; even glass tubes are objectionable for the same reason. Feeding-cups without a strainer are sometimes used,

but it is not particularly easy to clean the spout. A rubber teat is attached to the mouth. Both the feeding-cup and the boat-shaped bottle are advantageous, in that each feed must be supervised by the mother or nurse. The present feeding-bottle is the result of gradual evolution from the cow's horn, with parchment or leather on the worn-off tip, used in 1783. This was succeeded by a glass bottle of like shape in the early part of the nineteenth century, and after that by the pap-bottle and papspoon.

The Size of the Stomach.—The stomach capacity is more or less proportionate to the size of the child, but varies at different ages and in different children of the same age. It also varies in children of the same age and weight, so only a rough average can be ascertained. Most of the results have been obtained by weighing babies before and after nursing. This is not an accurate measure, for some of the food passes through the pylorus during feeding. Estimations must be based on many such weighings, not on one alone, for both the child's appetite and the amount of milk obtainable vary. The estimations based on amounts taken in artificial feeding are also unreliable, since the stomach often becomes dilated. So, too, measurements after death are untrustworthy, because of the liability to overdistension.

At birth the stomach is very small, and its capacity may be estimated as equivalent to 1 per cent. of the child's weight. Allow a daily increase in capacity of 1 gramme up to the end of the first month. Thus, a child weighing 6½ pounds (3,000 grammes) has a stomach capacity at birth of 1 ounce; at one week, 1½ ounces; at two weeks, 1¾ ounces; at three weeks, 1½ ounces; and by the end of one month, 2 ounces. A larger child may have an extra drachm or two for each feed.

In the second month the gastric capacity amounts to 2½ ounces. After this the stomach increases more slowly in size, and during the next four months holds 3 to 4 ounces, sometimes 5 ounces; and from six to nine months of age, 5 to 6 ounces, and occasionally more. In artificial feeding the gastric capacity is enlarged by increasing the amount of food allowed at a meal. The smaller measurements are the more accurate, and more consistent with those obtained by the weighing of breast-fed babies.

The Quantity of Food for a Meal.—This is based on the gastric capacity. Fix a maximum amount, but do not insist on the baby taking it all. The best indication that the food is satisfactory in quantity and quality is a regular weekly gain in weight, combined with contentment, normal sleep, satisfactory stools, and the usual indications of health. Quantity cannot be considered apart from quality. A bulky food is not necessarily a nutritious one, and too rich food may cause digestive disturbance. A child grows by the aid of the food it digests and assimilates. Either the quantity of the food or its quality may be at fault, sometimes both. In healthy infants the weight is a better indicator than the age in estimating the food requirement, but neither age nor weight alone is a sufficient guide. A wasted baby may need much more food than its weight indicates. No hard-and-fast rules can be enunciated. Some infants need remarkably little food. Others take an apparently excessive amount before making good progress. Each child must be carefully studied, and its food-supply apportioned to its needs and digestive capacity. Overfeeding is indicated by irritability, constant crying, indigestion, colic, vomiting, and unsatisfactory stools.

The Number of Feeds in Twenty-four Hours.—Just as if breast-fed, the child should be fed at the same intervals or less frequently. The breast-fed child empties

its stomach in one and a half to two hours, while a bottle-fed one takes about half an hour longer. During the first month of life he should have ten feeds, every two hours, from 5 a.m. to 11 p.m.; eight feeds at intervals of two and a half hours in the second month, and subsequently seven feeds at intervals of three hours. Often after six months of age, occasionally before, the number of feeds can be reduced to six at intervals of three and a half hours, or even to five at intervals of four hours. Prolonged intervals must not be encouraged, because hunger leads to the food being taken too rapidly, and may result in vomiting or gastro-enteric disturbance. It is a good plan to calculate the time-table from 7 a.m. or 8 a.m., and to allow one feed to be given subsequent to the last feed at night, preferably two to three hours before what may be called "the first meal of the day." Very delicate infants, especially during illness, may require feeding two-hourly night and day, only small quantities being allowed at each meal. Stronger children, who wake up hungry in the night, should be given richer food if they are digesting their diet satisfactorily. It is essential to distinguish between restlessness due to indigestion and that of hunger.

Regularity in Feeding.—Although animals, such as calves running with their dams, suckle whenever they wish to, both in maternal nursing and bottle-feeding the best results are obtained by feeding the infant at regular times "by the clock." The intervals between the feeds are calculated from the beginning of the feed. Each feed should take ten to twenty minutes. If the child is asleep wake him up, and if he takes little feed let him go without the remainder, and he will then wake up ready for his next meal.

The Substitute Food.—Use fresh cow's milk, delivered twice a day. It is rare for any infant, unless extremely marasmic or suffering with gastric disturbance, to be unable to digest cow's milk properly diluted, given in suitable quantities at the usual intervals. Exceptionally it may have to be diluted at first with even seven to eight times its bulk of water. Gradually the stomach is trained to deal with stronger mixtures. Theoretically it is unsound to increase the strength of the food with the increasing age of the child, for a maternal milk-supply increases in quantity, not in quality, as the child gets older. At two months of age a baby can generally be trained to digest a milk mixture containing 2 per cent. of protein. Strong healthy children who have reached six months of age, and are taking 6-ounce feeds, but appear to need more food, can be given a richer mixture. As a general rule the mixtures recommended below suit the average baby. If more food is required, I prefer to increase the proportions to milk 2 parts, diluent 1 part, rather than to increase the bulk. I am strongly opposed to giving undiluted milk. It is such a frequent cause of indigestion and constipation in infancy. Parrot (1887) advocated undiluted milk. Brien and Chavane have obtained excellent results by feeding infants on sterilized, undiluted milk. Prolonged heat renders milk less coagulable by rennet, and thus counteracts the effect of excessive casein curds in the stomach; but there is no reason to suppose that an excess of casein is an advantage to the child. I have seen many infants fed by this method develop troublesome gastric and enteric disturbance.

Satisfactory mixtures can be made according to the simple method recommended on p. 53, or in accordance with the following tables. These suit the average child, and can be easily modified if necessary.

FOOD DURING THE FIRST MONTH.
The quantities are given in drachms.

					Month of Life.			
					First.	Second.	Third.	Fourth.
Milk	2	3	4	5
Cream	1	1	1	1
Water	5	6	7	8
Lime-water	1	1	1	1
Milk-sugar	1	1	1	1

Do not begin with the addition of cream, but add it gradually if the diluted milk is digested. Gravity cream, containing 15 to 20 per cent. fat, is preferable to the richer centrifugal cream, which must be used in amounts of one-third. Butter (80 per cent. fat), margarine, and cod-liver oil, are useful substitutes.

FOOD AFTER THE FIRST MONTH.

					Month of Life.		
					Second.	Third to Fifth.	Sixth to Ninth.
Milk	1 oz.	1½ to 2 oz.	2½ to 3 oz.
Water	1 "	1½ " 2 "	2½ " 3 "
Cream	1 dr.	2 " 4 dr.	2 " 4 dr.
Lime-water	2 "	4 dr.	4 dr.
Sugar	1 "	1 to 2 dr.	2 "

It has been already pointed out that the use of top milk is preferable to the addition of cream. If cream is used, it is advisable to prepare it in the home from milk which has been allowed to stand in the cold. In the second table it is noticeable that the kind of sugar is not stated. This must be decided on in accordance with the considerations stated on p. 56. As a rule it is best to give lactose in the first two months, equal parts of lactose and cane-sugar in the next two to four months, and then cane-sugar. Maltose or honey can be given instead of lactose, and in the same quantities. After the second month thin barley-water can be used as a diluent, and lime-water omitted. In the sixth month it is often necessary to allow the larger one of the feeds recommended in the table, and at all ages it may be necessary to give a weaker or a stronger food.

The Preparation of Each Feed.—Measure in a graduated glass the quantities of cream, milk, and water, required for twelve hours. Spoons differ greatly in size, and are unreliable measures. Dissolve the sugar in the water. Mix thoroughly, and keep the mixture in a clean jug, covered with muslin, or in a tightly sealed glass jar, in a refrigerator or in a cool place surrounded by ice. To prepare a feed, thoroughly stir the mixture with a clean spoon or glass rod, and measure out the amount required. Pour it into a milk saucepan, and heat it until it just begins to boil. Cool it down, add lime-water if prescribed, and feed the child.

Or prepare the food for twenty-four hours, divide it into the requisite number

of feeds, and put them in separate bottles, stoppered with sterilized wool, and sterilize them in a suitable apparatus, such as Soxhlet's or Hawkley's. After sterilization the bottles are kept in the cold until required. The chief disadvantage of this system is the sterilization.

It is of comparatively little value to boil the milk when it is first delivered, and then allow it to cool down gradually while merely covered with muslin to keep off dust. Such milk goes sour almost as quickly as if unboiled. Infective organisms and many lactic acid bacilli are destroyed, but the spores of dangerous organisms may escape destruction, and develop freely as the milk cools.

The Temperature of the Food.—Neglect of attention to this point is a common source of failure of artificial feeding even in hospitals. Let the temperature be 100° to 102° F. at the beginning, for it quickly falls. Warm the food up again after half the bottle has been taken. The temperature must be ascertained by a thermometer, not by taste or dipping in the finger. The food can be warmed by standing the bottle containing it in hot water, or by means of a food-warmer.

Mode of Feeding.—The child should be held half reclining on the nurse's lap, with the head and back supported. Give the food slowly, keeping the teat full to prevent air being swallowed. Each feed should take ten to twenty minutes. The hole in the teat must be big enough to allow the food to pass through slowly, without undue exertion in sucking. Wipe the baby's mouth gently afterwards, especially at the corners, but not inside, and put him quietly in his cot to go to sleep. Sucking at an empty bottle or a "comforter" should be prohibited. The latter often falls on to dirty floors, etc., is perhaps cleaned (?) by being sucked by the nurse before it is replaced, and conveys dirt and infective organisms to the child. Both are sources of flatulence and indigestion.

Preservation of the Food.—Milk and cream must be fresh, and kept in sweet-smelling, cool places, such as a cellar or refrigerator, or in a tightly corked bottle in cold or iced water. A window-ledge with a north aspect is often a convenient place. Food must not be kept in a nursery, bedroom, or any place where it can be contaminated by smells from housemaids' sinks, drains, or decomposing food.

Cleanliness of Apparatus.—Wash each bottle after use in hot water, and then in soda and water, a bottle-brush being used. Then rinse it out several times in clean boiled water, and finally put it in a basin of clean cold water or boric acid solution, 1 drachm to the pint. Before use, rinse it out again in pure boiled water. The teats are thoroughly cleaned and kept in a similar way. All apparatus which has contained milk or cream must be well washed in hot water and soda and water, and finally in clean water, to get rid of the soda. The nursery, the cot, the nurse, and the surroundings of the child, must be kept scrupulously clean. An unexpected visit by the doctor to the nursery is more efficacious than yards of directions in procuring proper attention to details.

SHOULD MILK BE BOILED?—Heat converts milk from a fluid with many characteristics of a living tissue and various biochemical properties into one devoid of all semblance to life. It destroys ferments and micro-organisms, expels odours and gases, alters and precipitates some of the salts, reduces its antiscorbutic properties, renders it less coagulable by rennet, and affects its nutritive value.

Biochemical Character.—Fresh milk has an inhibitory power over the growth of organisms. Myer Coplan (1907) states that human milk inhibits bacterial growth absolutely for an hour, and almost absolutely for a second hour, the milk being digested in the stomach in the meantime. New cow's milk possesses inhibitory

powers for six hours. As an alternative to human milk in preventing infantile diarrhoea, give cow's milk, kept at 32° F. immediately after milking. Such milk retains half its inhibitory power at the end of twenty-four hours, but loses it at blood heat in one to two hours. Even under ideal conditions cow's milk has only one-fourth of the inhibitory power of human milk. This property decreases in milk which is kept, is damaged by preservatives, and is completely destroyed by boiling.

Bacteriology.—Ordinary milk as it reaches the consumer contains from half to ten million or more organisms per c.c., according to the external temperature, the cleanliness and purity of the milk-supply, and the care devoted to its management. "Certified milk" can be obtained in America containing not more than 30,000 organisms per c.c., 4 per cent. fat, and 0.02 per cent. acidity. There are present three main groups of organisms:

1. Non-spore-bearing—e.g., *B. coli*, *B. acidii lactici*, and *B. proteus* groups and various cocci. These are easily killed at 100° C., at 90° to 95° C. or in milk "brought to a boil," and at 70° C. (158° F.) for thirty minutes.

2. Spore-bearing and anaerobic—e.g., *B. acidii butyrici* and *B. enteritidis* sporogenes (Klein), organisms which cause acid decomposition and coagulation. The spores may be killed at 100° C. after one to one and a half hours' exposure, or with absolute certainty after three successive periods of such exposure.

3. Spore-bearing and aerobic—e.g., *B. subtilis* group or "peptonizing" bacilli, liquefying the faintly acid coagulum formed. Flügge isolated twelve species from milk. They grow rapidly at 25° C. (77° F.) and upward, and are very resistant to heat, the spores escaping destruction after an exposure to 100° C. for six hours. Like the *B. enteritidis* sporogenes, they give rise to infantile diarrhoea. The toxin is intracellular, and is destroyed by boiling.

These three main groups of organisms are those which give rise to lactic acid, butyric acid, and peptonization. Souring is commonly due to one or more of the numerous types of lactic acid bacilli. The degree of acidity is a fair test of the number of bacilli present. They are not only harmless, but are actually beneficial by crowding out and preventing the growth of other organisms. Moreover, sour milk is obviously not fresh milk. Delaying the process of souring by heat, preservatives, etc., simply gives other and more dangerous organisms a better chance of development, whereas sour milk does not go putrid for a long time. Both lactic acid and the bacilli act as intestinal antiseptics by preventing the growth of injurious intestinal flora.

The organisms of diphtheria, cholera, specific fevers, and tuberculosis, are readily destroyed by pasteurization, boiling, and sterilization. The heat does not destroy the products of the organisms; so the fresher the milk is at the time it is heated, the less is the risk from toxins. Unfortunately, although such processes destroy organisms, many spores, especially those of the most dangerous organisms, escape destruction.

Methods of Heating.—In pasteurization milk is heated to 158° F. (70° C.) for twenty to thirty minutes in a special apparatus. The character of the milk and its taste and smell are not much altered. Rapid cooling after exposure to heat is an important part of the process. I cannot recommend commercial pasteurized milk. It may not be fresh or clean before it is prepared. In "scalding" milk, "milk brought to a boil," the temperature is generally about 200° F., while in boiling the temperature is raised somewhat above that of boiling water. The taste

and smell are much altered. During cooling a thick tenacious scum forms on the surface, which consists of casein, albumin, fat, and salts. The milk is devitalized. In sterilization, a special apparatus or a home-made one being used, milk is exposed to superheated steam for twenty to thirty minutes. Properly prepared sterilized milk is better than the home-made article, in that dirt and foreign bodies are removed by the separator, and the milk is fresher when it is sterilized. The risk involved in its use is that unclean or unclean and stale milk, imperfectly sterilized, may be sold under this name. It is quite a different article from fresh milk.

The General Effects of Heat.—About 90 per cent. of the carbonic acid gas and 50 per cent. of the oxygen and nitrogen are driven off. Consequently the acidity falls, and earthy phosphates are precipitated. Some of the citric acid is precipitated as tricalcium citrate. Other salts are changed. The fat-splitting ferment is destroyed at 147° F., and the proteolytic ferment at boiling-point. Changes begin at a temperature of 140° F. Exposure at this temperature for thirty minutes destroys tubercle bacilli and reduces the percentage of soluble albumin. Exposure at 175° F. for ten minutes destroys the soluble albumin, partially caramelizes the milk, and changes its taste.

The antiscorbutic quality of the milk, highest when it leaves the udder, diminishes with the age of the milk, deteriorates on exposure to heat, and may be actually destroyed. Possibly this quality is connected with the citric acid present.

Heat is also said to render fat assimilation more difficult, to partly destroy the sugar, to convert organic phosphorus into inorganic phosphates, and to destroy nuclei and lecithin. No cooked milk is as nutritious as uncooked milk, and the nutritive value diminishes in proportion to the degree and duration of heating.

It is unsafe to use unheated milk except under special circumstances and in cold weather. Yet heated milk is not absolutely safe. Flügge has shown that the spores of twelve different kinds of aerobic bacilli survived in milk kept at boiling-point for two hours. These organisms are liable to set up diarrhoea. No degree of heat will destroy toxins previously formed, or convert bad milk into good milk. A clean milk is of greater importance than the use of methods to counteract the defects of imperfect milk. Unfortunately such methods are at present necessary. What is required is a supply of clean milk from healthy cows, cooled down to at least 60° F., unadulterated, unheated, free from preservatives, and containing 3.25 to 3.50 per cent. of fat.

Tests for Raw and Cooked Milk.—Some dairies send out the milk pasteurized, and it may be important to know whether the milk has been heated. Fresh unheated milk turns red on the addition of guaiac and 1 per cent. solution of hydrogen peroxide, added drop by drop. *Saul's Test:* To 10 c.c. of milk add 1 c.c. of an aqueous solution of orthomethylaminophenyl sulphate, or of acrid, and 1 or 2 drops of commercial H_2O_2 solution (about 3 per cent. strength). Raw milk gives a vivid red colour within thirty seconds. The colour is destroyed by alkali, but not by acid. The test will show the presence of 1 per cent. of raw milk. Milk kept at 158° F. (70° C.) for one hour reacts readily; kept at 167° F. (75° C.) for half an hour it does not react. Boiled milk and milk heated above 158° F. remains uncoloured, or becomes faint pink on standing.

The Effect of Heat on Coagling.—On heating milk at various temperatures up to 129° C. for periods of a quarter to one hour, and testing its coagulability with acetic acid and rennet ferment, after it had been cooled down to 40° C., the following results were obtained:

1. *Acetic Acid Reactions*.—Heat alone did not render cow's milk similar to human milk in its reaction. On prolonged exposure at a high temperature, the curd with acetic acid was more coherent. If the milk were diluted with water before being heated, the size of the curd depended on the degree of dilution. The finest curd, very like that of human milk, was obtained by dilution with 2 parts of water, and heating at 100° C. for a quarter of an hour. Weak or thick barley-water and lime-water as diluents gave similar results, according to the degree of dilution. The finest curds were obtained with thin barley-water as diluent, finer even than those with plain water. Up to 80° C. lime-water did not prove as satisfactory a diluent as plain water. At higher temperatures the milk was altered in colour by the action of heat on the lime-water, and the results were valueless.

2. *Reactions with Rennet*.—One drop of rennet solution coagulated fresh milk in two minutes. If milk were sterilized by exposure to 100° C. on three successive days for one hour daily, it required a large quantity of rennet to produce coagulation. Even at the end of forty-five minutes only a soft curd was produced. The effect of dilution by the large amount of rennet solution added must be remembered. The amount of rennet required to curdle heated milk increased with the height of the temperature and the duration of exposure. If the milk were diluted before being heated, the softness and degree of cohesion of the curd depended on the degree of dilution rather than on the heat applied. Barley-water had no more effect than plain water on rennet-curdling. These experiments tend to show that the essential factor, in rendering the reactions of cow's milk with acetic acid and rennet like those of human milk, consists in simple dilution with water to such an extent as to reduce the percentage of caseinogen to that present in human milk. They clearly prove that heat modifies caseinogen, possibly by action on the lime salts, so that it is less coagulable by rennet.

The Effect of Alkalies and Citrate of Soda.—It has been pointed out (p. 33) that caseinogen enters into a variety of combinations with acids and alkalies. The addition of alkalies reduces the coagulability with rennet, or even completely destroys this reaction. Sodium citrate inhibits rennet-curdling in the test-tube and in the stomach. One grain or more is added to each ounce of milk. It is generally said to precipitate some of the lime salts; but Varlet states that it increases their solubility. Citrate of soda is a neutral salt, very soluble in water, cheap, and more effectual than citrate of potash. A drop or two of chloroform, or impregnation with CO₂ gas, is necessary to prevent a fungus growing in weak solutions. Undiluted citrated milk is often a suitable diet.

Probably the beneficial effects of sterilization, citrate of soda, and alkalies, are closely allied. By preventing coagulation with rennet, the milk is prevented from curdling in the stomach, and passes on readily into the intestine. Gastric digestion is lightened, and is replaced by pancreatic digestion.

Home Modifications of Milk.—The use of mechanical diluents, the peptonization of milk, and the preparation of whey and butter-milk, are the chief modifications available. Decoctions of barley, oatmeal, or rice, are the common diluents; occasionally egg-albumen, gelatine, or dextrinized foods. Oatmeal is richer in fat than the other cereals, and is somewhat laxative. Rice is practically devoid of fat.

Peptonized Milk is prepared by means of Fairchild's *zymose* powders or the *Allenbury peptonizing powders*. Stale powders are apt to curdle the milk. Sometimes such curdling is due to staleness of the milk. Directions are supplied with

the products. The milk is diluted with one-fourth part of water. The duration of peptonization varies from ten to twenty minutes, and the mixture is then rapidly brought to a boil to prevent further digestion. If the peptonization is prolonged the mixture becomes bitter. "Fairchild's peptogenic milk powder" can be used. It contains pancreatin, bicarbonate of soda, and milk-sugar. A mixture of milk 10 ounces, cream 2 ounces, water 10 ounces, and the milk powder 1 measure, is heated slowly, with constant stirring, so that it is brought to a boil at the end of ten minutes, or is heated quickly to 190° to 140° F. for six minutes. The resulting fluid, often called *humanized milk*, contains protein 2.0, fat 4.5, sugar 7.0, per cent. (Leeds), and the proteins are in a minutely coagulable and digestible form. "Commercial humanized milk" varies greatly in composition and mode of preparation. It should not be recommended unless its mode of preparation and percentage composition are known to the prescriber.

Partial peptonization does away with the necessity for attenuating the curd, though it may be advisable to lower the percentage of proteins by dilution with water. The amount of peptonization is reduced by shortening the duration of the heating process, and a return to the ordinary milk diet can be thus effected. If one feed of unpeptonized milk is given in place of one peptonized feed, and if this agrees the other peptonized feeds can be gradually replaced. Used reasonably, and for not more than three to four months, peptonization does not seem to injure the digestive powers of the child. It is chiefly of value for delicate or wasted children with digestive powers weakened or in abeyance, and in febrile disorders interfering with digestion. Full directions must be given about the process of preparation, the amount for each feed, and the number of feeds to be given.

Whey is made by curdling whole or separated milk. As the curd contracts the whey is squeezed out. Sometimes the curd is broken up mechanically with a fork, and squeezed through muslin or a fine hair sieve, thus breaking up the casein particles, which do not again become blended into coherent masses in the stomach. Junket tablets or various preparations of rennet are used. The latter are made with glycerine, are variable in strength, and may be impure. Beauchamp Hall advises the use of tablets of purified and standardized rennet, sufficient to curdle 10 ounces of milk. Each tablet is made up with soda bicarb. 1½ grains, calcium phosphate ¼ grain, and lactose 5 grains. Ten ounces of milk yield seven of whey.

Whey is devoid of casein, practically fat-free, and contains about the same amount of sugar and salts as the original milk. It is enriched by casein particles and fat globules if the curd is squeezed in muslin. The albumin may be reconstituted at 0.85 per cent.

VARIOUS ANALYSES OF WHEY.

				Maximum.	Maximum.
Proteins	0.82	1.00
Fat	0.05	0.18
Sugar	6.63	5.45
Ash	0.49	0.70

It is an easily-digestible, weakly-nutritive fluid, useful as a temporary food and in the first few months of life. As a diluent of milk it has the advantage of reducing the percentage of caseinogen, but not that of albumin. The excess of salts may

give rise to intestinal toxemia or gastroenteritis. A mixture of cream and whey is more nutritious. The addition of 1 drachm of cream, 16 per cent. fat, to 2 ounces of whey, is equivalent to adding 1 per cent. of fat. When cream or milk is added to whey, the whey must be first heated to 150° F. to destroy the rennet ferment, or curdling may result. This is not essential if only small quantities of cream are added or if centrifugal cream is used. Minute curds are unimportant.

Sweet Whey Powder.—On account of the difficulties and trouble involved in making whey, a useful substitute in the form of "sweet whey powder" can be employed. Its percentage composition is—Protein, 14.25; fat, 0.27; lactose, 74.45; ash, 9.80; moisture, 1.28. One teaspoonful in 2 ounces of water yields a fluid apparently identical with whey.

Tartared Whey is recommended by Still and Myers (1907) as a cheap substitute. Add tartaric acid, 8 grains, to half a pint of milk brought to a boil. It is then kept simmering for five minutes, and strained through butter muslin. The specific gravity of the whey is 1000. It contains protein 0.38 per cent., fat 1.2 per cent., and is only faintly acid to litmus. It can be added to milk without further curdling.

White Wine Whey.—For this Still recommends a cheap cooking sherry, about one shilling per bottle, because it contains more acetic and tartaric acid and less alcohol than the better wines. Ten ounces of milk are brought to a boil, 2½ ounces of sherry added, and the mixture is again boiled up. It is then removed from the fire, allowed to stand for three minutes, and strained through butter muslin. One ounce of this whey is about equivalent in alcoholic strength to 25 drops of brandy.

Citric Acid Whey.—Boil a pint of milk with lemon-juice, 2 drachms, and strain off the curd.

Butter-Milk is made from sour milk or cream, preferably from sour skimmed milk, and should be less than twenty-four hours old. One pint is churned for fifteen minutes in a glass churn, of 1 quart capacity, at a temperature of 60° to 70° F. In the process of preparation the calcium casein is converted into casein lactate, and the sugar is decreased in consequence of lactic acid fermentation. Its acidity is about 0.5 per cent.; caloric value, 9 to 12 per ounce; and percentage composition, protein 2 to 3, fat 0.25 to 1.5, sugar 3.0 to 3.5. It is generally given sweetened and sterilized. Add one teaspoonful of flour and four of granulated sugar to a quart of butter-milk, with constant stirring, and heat it to boiling in a double saucepan. If heated too much it will curdle, unless it is stirred constantly or is made slightly alkaline by the addition of carbonate of soda. It must be cooled rapidly and kept in bottles. It contains a considerable amount of lactic acid and, if unheated, crowds of living lactic acid bacilli. "Nutricia" is a condensed butter-milk.

Lactobacilline Milk is a commercial product, but can be prepared at home. It is analogous to butter-milk, and is made by inoculating milk with huge numbers of lactic acid bacilli. It is then sweetened and sterilized; or given unsterilized, if it is desired to give large quantities of lactic acid bacilli as well as lactic acid. *Kepair*, *Matsou*, and *Koumiss* are derived from the milk of the cow, goat, mare, or ass, by fermentation with yeast or kephair grains. They are quite unnecessary. Alcohol, if required, can be given in simpler forms. These foods are useful in enteritis and colitis, and are supposed to limit intestinal putrefaction.

Dialyzed Milk is useful in infantile dyspepsia, sepsitis with oedema, and in the exudative diathesis. Lehndorff and Zak advise the following mode of preparation: Suspend 500 c.c. of milk in a parchment bag in 10 litres of water. Change the water hourly, and keep it at 115° to 131° F. (45° to 55° C.). Add 0.1 c.c. polydial

to prevent decomposition, and again in two hours' time. In four to five hours the sugar is reduced to about half, and the salts are also reduced. It tastes more like distilled water than milk, and coagulates at 176° F. unless a minute quantity of sodium carbonate is added.

Albumin.—It is sometimes advisable to increase the percentage of albumin in the milk by the addition of albumin. This is a greyish-white powder of neutral reaction, and percentage analysis (Lancet, 1911), albumin 85.00, mineral matter 6.00, moisture 10.40. It is milk albumin in a soluble form, an albuminate. Bergell states that it is a pure uniform salt of albumin, soluble in water. About 3 grains is added to each ounce of diluted cow's milk, and prevents the formation of large curds on acidification. "Sweet whey powder" can be used in a similar manner.

Condensed Milk.—A full account of "condensed milks" is given in the Report to the Local Government Board, 1911, by Dr. F. J. H. Cottis. Borden obtained a patent for his process in 1865. The milk was used in the American Civil War, and has been used in rapidly increasing quantities ever since. In addition to the quantity made in this country, a yearly amount of about 1,000,000 hundredweight is imported. It is usually condensed to about one-third of its original bulk.

Varieties (Cottis).—1. Full Cream.—(a) Fully sweetened by the addition of 35 to 40 per cent. of cane-sugar (*i.e.*, sucrose from any source, probably derived from beet).

(b) Partially sweetened by added sugar, less than 18 per cent.

(c) Unsweetened.

2. Machine-skimmed.—(a) Fully sweetened by 38 to 45 per cent. of added sugar.

(b) Partly sweetened, by less than 18 per cent. sugar.

(c) Unsweetened.

Thus, they are prepared from skimmed or separated milk, whole milk, or whole milk with added cream. About half the imports consist of sweetened, skimmed or separated milk. Unfortunately, this is often given to babies, whereas it should be reserved for the use of older children and adults.

Of the sweetened condensed milks, Nestlé's and the Milkmaid brands, prepared by the same firm under admirable precautions, are most satisfactory as regards quality, purity, and cleanliness. Similar brands are the Rose, Full Weight, Anglo-Swiss, and Peacock. All these contain approximately protein 10, fat 10, and sugar 60, per cent. They must be diluted with eight or more times their bulk of water to reduce the percentage of sugar, and cream added to raise the fat content.

Unsweetened condensed milk, such as the Ideal, First Swiss, Viking, and Hollandia brands, contain protein 8 to 11, fat 2 to 12, and lactose 13 to 18, per cent. Of these, the Ideal is richest in fat. It must be diluted with water, and cream and lactose added to bring its chemical composition up to that of human milk. Such milk soon turns sour when the tin is opened. Cane-sugar acts as a preservative of the sweetened varieties.

It is often supposed that condensed milk is sterile. Gordon and Elmadfa, in an addendum to the report quoted, state that, of fifteen brands examined, none was sterile. The organisms were chiefly *saccharae*, protease, spore-bearers of the *Bacillus subtilis* group, and other organisms which had no recognizable source in the milk or in human contamination. Streptococci, probably of fecal origin, were absent from only four brands. The abundant organisms were such as are abundant in the air. Unsweetened milk was the most sterile. Bacilli of the colic group were

all destroyed in the process of condensing. Although the milk was not sterile, the number of organisms was infinitesimal in comparison with the huge number present in cow's milk used by the poor in hot weather.

Its Advantages.—Both varieties are valuable while travelling, during hot weather, in malarial states, and if good cow's milk is not obtainable. The unsweetened brands are chiefly of use in the treatment of infantile diarrhoea and other alimentary disorders in which a large amount of cane-sugar is contra-indicated. It is often necessary to add a little cane-sugar to make them palatable. The sweetened varieties are most used. As they are generally freely diluted and sweet, they are readily taken, digested and absorbed easily, and are quickly prepared. Machine-skimmed condensed milk, sweetened or unsweetened, is a cheap food suitable for children taking a mixed diet and getting fat in other forms. It is much superior to jam because of its protein content. In practice, only those milks condensed from whole milk, or from milk with added cream, should be allowed in the feeding of normal infants. Used under medical advice they give excellent results, and certainly save the lives of many malarial infants. A good condensed milk is greatly superior to dirty, inferior cow's milk. It should not be continued as a permanent diet for more than a few months. The tendency to scurvy is counteracted by means of fruit juice, the deficiency of fat is made up for by giving cod-liver oil emulsion, and extra protein can be added in the form of egg albumin. The addition of yolk of egg to the diet is of great benefit.

Its Disadvantages.—Babies fed only on condensed milk almost always develop rickets, frequently become anæmic and sometimes scorbutic, and generally get fat and flabby. Its defects consist in the deficiency of protein and fat in the dilutions given, and the relative excess of sugar. It is, however, an exaggerated statement to make that every baby so fed becomes rachitic. I have seen several who have taken only this food for months at a time, and yet have cut their teeth at or even before the usual dates, and have shown no signs of rickets. Such babies are generally unduly fat. They are liable to bronchitis and gastro-intestinal derangements, and possess little vital resistance. The methods of counteracting the evil effects of a condensed milk diet have been already indicated. Some of these ill effects should be ascribed to an insufficiency of nutritious food rather than to the milk, for in many cases the baby is fed on a very dilute mixture, and is practically starved. The composition is not invariably the same as that stated by the manufacturer. Sometimes chemical preservatives are added. The milk may be stale or dirty before condensation, and individual tins may deteriorate or be imperfectly sterilized.

Dried Milk.—One of the most satisfactory methods of preparing dried milk is the Just-Balmaker process. The milk is mixed with warm water for use. It is cheap, convenient, palatable, and easily digestible. Dried milk is sold under the names of Glaxo and Lactinum, and desiccated skimmed milk as Lactogen. Glaxo contains protein 22.2, fat 27.4, sugar 41.5, ash 3.4, and water 6.0, per cent. Lactinum contains protein 28 and fat 29 per cent. They are useful temporary foods in hot weather, but must not be given for long, on account of their liability to cause scurvy and the deficiency in fat if they are much diluted.

Many preparations of pure casein are on the market, and are most valuable foods for growing children. The chief ones are Biegene, Casein, Casmen, Placmen, Proteze, and Tilia. Casein is combined with ammonia as Eucasin, with sodium

as Nutrow, with sodium glycerophosphate as Sanatogen and Vi-casein, and with albumose (20 per cent.) as Sanose.

Ass's Milk is obtainable from Welford's Dairy Company, London, at 3s. per pint in sealed bottles. One ass supplies enough milk daily for three infants under three months of age, two during the next two months, and one only of an older age. It is given at 100° F.

THE COMPOSITION OF ASS'S MILK.

	Dextrose- Saccharose.	Glucose.	Casein.	Whey- Myelin.	Fat.
Protein	1.23	1.4	1.7	1.88	1.95
Fat	2.01	0.8	1.4	1.02	1.07
Sugar	6.33	5.6	6.4	5.38	6.48

It contains about the same amount of proteins of both kinds and of sugar as does human milk, but is deficient in fat. Babies do well on it for a short time—say up to two to three months. Cream can be added. It has slight laxative properties, counteracted by lime-water and destroyed by boiling.

Goat's Milk resembles cow's milk on chemical analysis, though it is said to be more digestible. It has a strong odor and unpleasant smell if the animal is ill, fed on garlic, &c., or is milked into dirty pails. Its chief advantage lies in the fact that anyone with a small plot of grass can keep a goat, feed it carefully, and obtain fresh pure milk. Abroad, it may convey the organism of Malta fever.

Proprietary Foods.—In many of the proprietary foods recommended for infants there is a large amount of carbohydrates, frequently in the form of starch. In some the starch is partly or entirely converted into dextrins, dextrose, and maltose. Others contain much cane-sugar. Many have a basis of condensed milk. Seeing that there is no substance analogous to starch in milk, it is unlikely to be a suitable food for young infants.

The Value of Starch in Infant Feeding.—It is sometimes maintained that no starchy food is permissible for an infant under six months of age. Practical experience shows that weak barley-water can be used as a diluent of cow's milk during the first few days of life, as well as later, often without injury to the child and frequently with decided benefit. Probably a small amount of starch can be digested at any age, and the infant's stomach can be trained to digest increasing quantities. Successful artificial feeding of infants depends greatly on the power of accommodation in the digestion of what is really an abnormal food, for, as has been shown, artificial milk mixtures are not identical with human milk, though possessing the same composition according to careful analysis. It has also been shown that it is not essential to use lactose, and that cane-sugar is often just as suitable. Starch is even better in some respects. It is not so sweet, and is less likely to set up catarrh of the mucosa.

An amylolytic ferment is secreted in the saliva during the early months of life. Ibrahim (1909) found it present in the submaxillary and parotid glands of twenty-two premature and twelve new-born infants. It gradually increases in activity and quantity as the infant gets older; but salivary secretion is very scanty and rarely appreciable before the age of two months. About 1 c.c. is secreted in

twenty minutes at the end of the first month, and in two minutes at the end of the third month of life.

Both the mæconium and the stools of early infancy contain a diastasic ferment. It is found in the pancreas of the premature and new-born, and rapidly becomes stronger as age advances. Possibly another ferment, capable of inverting starch, is secreted by the epithelial cells of the buccal and intestinal mucosa. Heat and moisture will also invert small quantities of starch without the aid of a ferment. Experimental observations on the amount of unchanged starch in the stools have shown that a considerable quantity of the starch ingested is absorbed, the amount varying in different infants. The degree of digestibility varies with the kind of starch. Potato starch is most readily digested, while next in order come the starches of wheat and oats, barley, maize, and rice. It is digested with difficulty unless previously converted by heat into "soluble starch," as in making barley-water, the granules swelling and rupturing the cellulose envelope of the starch grain.

From these considerations it is a just conclusion that starch can be digested from birth onwards, and that the glands can be trained to increased diastase activity in order to deal with increased quantities of starch. There is no inherent reason why this training should not be begun shortly after birth, instead of waiting until the age of six months, an age chosen on theoretical grounds. Barley-water, as usually made, contains about 2 per cent. of starch. Mixed with an equal quantity of milk the percentage is halved. Such an amount of starch is non-injurious, and may be beneficial by encouraging the growth of lactic acid bacilli and the formation of lactic acid. These organisms assist in preventing the growth of proteolytic bacteria. If a starchy diluent is used from birth, begin with a milk mixture containing 0.5 per cent. or less of starch, and gradually increase the amount as the child gets older. At all ages adopt this course, slowly increasing the percentage up to as much as 3 to 5 per cent. It is often advisable not to begin the use of starchy diluents until the third month, sometimes later, because of the deficiency of diastase ferment and salivary secretion. The evil effects of starch in early life result from (a) excess; (b) its administration in the form of a more or less insoluble emulsion instead of as soluble starch; (c) the substitution of starch for the necessary proteins, fat, and salts. In other words, the mischief is due to a deficiency in the proximate principles of diet rather than the presence of the starch. In many instances infants get on better if water is used as a diluent instead of barley-water. It is not necessary to add starch to the diet before the age of six to nine months. At this age it is advisable to begin to educate the child gradually in the digestion of starch. For this purpose either ordinary household foods can be used, or recourse had to various proprietary and more expensive articles of diet.

Disadvantages of Proprietary Foods.—As a rule they are unnecessary, for the child can be brought up on modified milk. Extra fat is supplied in the form of cream, butter, margarine, olive oil, or cod-liver oil. Extra protein can be given by using whey or egg-albumen water as a diluent, or by adding a dried casein preparation. The percentage composition of the foods is sometimes misleading, and is not always constant. The different elements may be combined in unamiable forms, the salts insoluble and not in the form of organic compounds, and the fat imperfectly emulsified. Excess of carbohydrates, especially when previously converted into maltose, etc., produces obesity rather than health and strength. The mortality among infants brought up on artificial foods is very high, although some babies take these foods with impunity, and even advantage. The magnificent

labels of manufacturers' advertisements only exist in the imagination, or, when seen in the flesh, are generally fat and rachitic. Fortunately, the disadvantages of these foods are largely discounted by giving them with fresh milk. Rickets and scurvy often ensue.

Classification of Proprietary Foods—1. *Condensed Milk*:—Sweetened and unsweetened, skimmed and unskimmed.

2. *Condensed Milk plus Carbohydrates*.—(a) *Containing Malted Starch*—e.g., Allenbury No. 2 Food, Horlick's Malted Milk, and John Bull No. 1 Food; foods practically devoid of starch.

(b) *Containing Partly Malted Starch*—e.g., Maltin Food, Mido Food, and Theri-lactin's Infantina.

(c) *Containing Unchanged Starch*—e.g., Anglo-Swiss, Franco-Swiss, and American-Swiss Foods.

Benger's Food may be included in this group. It consists of cooked wheaten meal and extract of pancreas. On preparing it with milk the ferments partially digest the proteins and the starch. The fat is unaffected. It is valuable for insensate and delicate infants. The addition of half to one teaspoonful to a feed is only about equivalent to using barley-water as a diluent. It must not be increased in quantity at the expense of the essential cream and milk. Carrick's Soluble Food is comparable with the foods made from condensed milk and partly malted flour. It is made of desiccated milk, malt and wheat flour, lactose, and extract of pancreas. It is inferior to Benger's Food in not being prepared with fresh milk.

3. *Carbohydrate Foods*—(a) *Completely Malted Foods*.—Mellin's, Horlick's, Page's Malted Farina, and Hovis Babies' Food. Cheltine Maltose Food, Diastase Farina, and Racia, are of a like nature. Malted food can be prepared at home by Terrier's process: Make a gruel of rice flour 80, milk 200, and boiled water 600, grammes, and add an infusion of germinating malt. Maltine or Kepler's Malt Extract can be used instead. Let it stand for quarter of an hour to half an hour at 160° F., and then add sugar 50 grammes.

(b) *Partly Malted Foods*.—Allenbury No. 3, Cuisine Infants' Food, Comb's Malted Food, Hovis No. 2, John Bull No. 2, Hoveley's, and Savory and Moore's Food.

(c) *Practically Unchanged Starch*.—Neville's, Ridge's, Opman, and Albany Foods, Robinson's Patent Barley and Groats, Scott's Oat Flour, Chapman's Flour, etc.

Basasee Flour, recommended by Pritchard, contains starch, sugar and gum 77.5, and albuminoids 2.5, per cent. One tablespoonful is added to a pint of water and boiled for five minutes. It is then mixed with an equal quantity of milk.

Malted foods can be given at a very early age in small quantities instead of cane-sugar, provided they are not unduly laxative. It may be advisable to use partly malted foods before the age of six months, but those composed of unchanged starch are never really necessary. To train a child gradually in the digestion of starch, begin with a purely malted food—e.g., Mellin's—pass on to a partly malted food—e.g., Savory and Moore's—and then give one of unchanged starch. It is simpler, cheaper, and generally satisfactory, to begin with thin barley-water, and gradually make it thicker and thicker.

4. *Meat Preparations*, such as various brands of meat teas, extracts, juices, and jellies, peptones, and peptoids, and meat powders. Most of these consist of extractives which have no direct nutritive value, but possibly stimulate the

digestive functions and throw an extra strain on the liver and kidneys. The high proportion of salts and extractives is liable to induce thirst and diarrhoea. Some meat jellies, though expensive and unwholesome, have a pleasant flavour, and, being readily taken by a feverish infant who refuses other food, may encourage appetite. Hot fluids, like beef-tea, are pleasant to the febrile tongue and in disordered digestion. In small quantities they do no harm, and their administration comforts an anxious mother, who fears her child will die of starvation and has a profound faith in the nutritive value of such foods. As ordinary articles of an infant's diet they must be condemned, for they are apt to spoil the appetite for simpler milk and carbohydrate foods. Sometimes they are the cause of an irritable, neurotic state and a tendency to night terrors. There is no serious objection to giving home-made chicken tea, mutton broth, and similar foods, in the last quarter of the first year of life, especially if it is desired to reduce the nutritive value of the diet. A daily meal of chicken broth, thickened with pounded vegetables and a cereal is useful for fat, plethoric, overfed babies, who have been brought up on an excess of malted foods, cream, and milk.

No meat preparation containing alcohol or kola is permissible for infants. Of the various meat extracts in the market, Bovril is the most trustworthy and nutritious. Of some of these preparations ox-blood appears to be the basis; and egg-albumin appears to be the chief protein constituent of others. Liebig's Extract is free from protein and fat; and Valentine's Meat Juice, according to Chittenden, contains only 0.55 per cent. of coagulable protein and gelatine. If extra protein food is required, it is preferable to use white of egg, the juice of fresh meat, freshly scraped raw or slightly grilled meat, casein foods, or a dried meat powder such as can be prepared at home by slowly drying minced boiled beef in the oven and then grinding it up in a coffee-mill. Meatox and Brand's Nutrient Powder are commercial meat powders. Predigested foods are apt to induce diarrhoea because of the albumoses and peptones present in them. The richest of these is Somatose, containing 80 per cent. of soluble proteins. It is a grey powder, made from meat, soluble in water, and almost devoid of taste and smell. A milk somatose is made from milk.

5. *Vegetable Proteins* include Aleurone and Aleuronat, made from wheat; Legumin, or vegetable casein, made from pulses; Plantose, from rape seed; and Roborat, from cereals. Roborat is the best, and contains 83 per cent. protein; it is tasteless and the most soluble variety.

6. *Fat Foods*.—Pichard oil, Cottonseed oil with 6 per cent. oleic acid, various cod-liver oil emulsions, and Zipatin, olive oil with 6 per cent. oleic acid, are used as substitutes for cream and other fats. Pancreatic Emulsion consists of emulsified lard, pig's pancreas, and oil of cloves. Petroleum Emulsion is a hydrocarbon, not a fat, and is useful as a lubricant. Everton Toffee consists of fat and carbohydrates; Cremalto is a mixture of Devonshire cream and malt; Chocolate contains fat 20 and carbohydrate 50 per cent.; Virel is made from malt extract, marrow fat, yolk of egg, and lemon-juice, and is said to contain 20 per cent. fat. Virevin is much the same. These are partly foods and partly medicines, and are often of value in the dietetic treatment of delicate children.

Diet after Weaning.—When a child is digesting satisfactorily a mixture of cream, milk, barley-water, and sugar, and has reached the age of nine months, or even earlier if artificial feeding has been begun at a young age, the next step is a gradual increase in the percentage of starch in the feeds and a change in the variety

used. Such foods as barley or oatmeal jelly, or a thin gruel made with cornflour, ground rice (1 drachm to 3 ounces milk), fine oatmeal, tapioca, or arrowroot ($\frac{1}{4}$ drachm to 12 ounces milk), may be added to the milk and well boiled. The next step is the addition of half to one nursery rusk or Robb's biscuit to one or two feeds daily of milk and water equal parts, or milk two parts to one of water. Malted rusks are preferable if the child is asthenic. If the child is at all anæmic or rachitic, give one or two teaspoonfuls of yolk of egg, raw or lightly boiled, in two of the daily milk feeds, or the yolk of egg and raw-meat juice on alternate days.

It is difficult to get quite fresh meat, and both meat and meat-juice keep badly in hot weather. Beef-juice may give rise to tapeworm, so the juice of mutton is safer. Meat-juice is best made by means of a meat-press, such as the Hercules meat-press, but it can be prepared by mincing the meat, pounding it up in a mortar with a pinch of salt, and standing it in a cup with just enough cold water to cover it for two to three hours, and then squeezing it through muslin. It must be kept in a closed bottle surrounded by ice in a refrigerator.

During the eleventh and twelfth months purées of peas and chestnut meal, and mashed potatoes, may be given, if the child wants variety and digests the other foods, but it is often advisable to postpone these foods until the first year of life is ended. A hard crust of bread may be given to the child to chew as soon as the first tooth is cut. Bread is rarely advisable as food before the end of the first year, and never until the child is digesting starch in other forms. It is somewhat glutinous, and is apt to be swallowed in indigestible lumps. The so-called Standard Bread is not necessary. Its coefficient of digestibility for protein, fat, and carbohydrate, is lower than that of white bread. More nutriment is obtained from an equivalent quantity of white bread than from Standard bread of 85 per cent. flour. Possibly, among children fed chiefly on bread, the proteins, nucleo-proteins and basic proteins in the germ present in Standard bread have some nutritive value, but the removal of the germ does not take away much protein.

As a rule too much milk is given after weaning and until the child is on a full mixed diet. Excess of milk gives rise to constipation, anæmia, pallor, and inadequate nutrition, from lack of other foods. Stomach-eructation is present as consequence of dilatation, but disappears when the condition of the child gets worse from milk dyspepsia, vomiting, and diarrhoea. During the weaning period five or six meals a day are sufficient.

Diet from Twelve to Eighteen Months of Age.—The simple milk and carbohydrate diet can be continued if the child and mother are satisfied and the child progresses favourably. I prefer to give the milk diluted with one-third of diluent, unless it is very well digested and the child's appetite good. A more mixed diet is generally required—e.g.:

FIRST MEAL (at 6 to 7 a.m.).—Six ounces of whole or diluted milk, hot or cold. A slice of stale bread or a rusk broken up and soaked in milk, or a slice of thin bread with dripping or butter.

SECOND MEAL (at 8.30 to 9.30 a.m.).—One of these daily.—A small basin of rusk or bread and milk. A little fine oatmeal porridge with cream or milk. A basin of thick milk gruel. A cup of thin cocoa made with milk, and thin bread and butter.

THIRD MEAL (at 12.30 to 1.30 p.m.).—*First Course:* One of these—Mashed baked cold potato moistened with milk, chicken or mutton broth, the red gravy of

undercooked meat or meat-juice, 1 to 3 ounces. Lightly boiled or poached egg yolk, mixed up with stale breadcrumbs or with mashed potato and gravy. Stale breadcrumbs soaked in gravy or meat-juice.

Second Course: One of these—A large tablespoonful of mustard, tapioca, comfort, ground rice or semolina pudding, blanchmange or junket. Cold water, or milk and water, to drink.

FOURTH MEAL (at 4.30 to 5 p.m.).—The same as at the first meal; or thin cocoa, with bread and butter.

FIFTH MEAL (at 8 to 9 p.m.).—A large cupful of milk gruel made with rice, sago, tapioca, or hominy; or a rusk or sponge finger soaked in milk.

Many other foods can be given at this age, especially those of a carbohydrate nature, preparations of egg, small quantities of jellies and fruits such as the pulp of grapes and oranges, baked apples, and even mashed banana if it is thoroughly ripe. Proprietary foods—e.g., Mellin's, Savory and Moore's, and Benger's—are very useful for delicate children when given as the last meal of the day. A healthy, strong child may require a more nitrogenous diet, and can have pounded-up fish, chicken, or red meat, at the midday meal when fifteen months old, and exceptionally at an earlier age. Occasionally the following diet, suitable for an older child, can be begun for a strong, well-nourished baby three months younger. It is important to make all changes slowly, and, when a new article of diet is tried, to begin with small quantities.

Diet from Eighteen to Thirty Months of Age—**FIRST MEAL** (at 6.30 to 7.30 a.m.).—A cupful of milk with rusk, stale bread, or milk biscuit, broken up and soaked in it, or some bread and butter.

SECOND MEAL (at 8.30 to 9.30 a.m.).—One of these—A basin of rusks or bread and milk. Oatmeal porridge with cream, milk, or golden syrup. Hominy grits or Quaker Oats and milk. Cocoa or milk, with bread and butter. Milk, a lightly boiled egg, and bread and butter or dripping.

THIRD MEAL (at 1 to 1.30 p.m.).—*First Course*: One of these—Boiled fish, chicken, turkey, roast or boiled mutton or undercooked beef minced up finely, and for the younger children pounded up in a mortar into a paste; one tablespoonful mixed with mashed old potato or with stale breadcrumbs and gravy, and vegetables such as spinach, young green peas, and young asparagus-tips, passed through a sieve. Lightly boiled or poached egg with mashed potato or stale breadcrumbs and gravy. A large cupful of broth or soup (purée) with mashed potato or stale breadcrumbs.

Second Course: One of these—Custard or plain milk pudding, blanchmange, comfort or ground-rice mould, junket, plain sweetened jellies, baked apple.

FOURTH MEAL (at 4.30 to 5 p.m.).—Milk, or thin cocoa made with milk, with bread and butter, rusks, plain biscuits, sponge fingers, and bread and honey. Or a basin of milk gruel, rusk and milk, or bread and milk.

FIFTH MEAL (at 8 to 9 p.m., if desired).—One of these—A cup of milk gruel. A drink of milk and a biscuit, rusk, or piece of bread, soaked in it. A feed of Mellin's, Savory and Moore's, or Benger's Food, if the child is badly nourished.

Water or barley-water should be given as a drink at dinner, and, when wanted, between meals for the relief of thirst, especially in hot weather. Milk should be diluted if the child is overstimulated. Tea, coffee, and stimulants, are not permissible.

Many vegetables—e.g., cabbage, broccoli, turnip-tops, and cauliflower—may be given if passed through a sieve. Carrots, potatoes, artichokes, peas, and lentils, can be allowed in the form of vegetable purées if they are thoroughly cooked. The juice of fruit and fruit jellies, such as those of apple, blackberry, and currants, are also useful.

Diet after the Age of Thirty Months.—Four meals a day are sufficient, but a fifth one is permissible.

BREAKFAST (at 7.30 to 8.30 a.m.).—One of these—Bread or milk and milk porridge with milk or golden syrup, honey grits, Quaker Oats, or other cereal food. An egg boiled, poached, or scrambled. Bread and butter, dripping, or fruit jelly. Milk or cocoa to drink.

It is often advisable to give a first course of some carbohydrate food in small quantity, and a second course of egg or bread and bacon fat. After the age of four years there should be a definite second course of egg, plain omelette, fat bacon, or ham, fish, or home-made potted meat.

LUNCH (at 11 a.m.).—A cupful of milk and a slice of bread and butter or dripping, or a biscuit. If the child takes a poor dinner, omit lunch, or merely allow a little fruit—e.g., some grapes or a ripe banana—and a drink of water.

DINNER (at 1 p.m.).—The *First Course* is selected from the following: Boiled fish, poached egg, brains, sweetbread, minced beef, mutton, chicken, or turkey, vegetable purées. Up to the age of four years allow red meat twice a week.

Second Course.—Plain boiled puddings, meat dumpling, milk puddings, tarts, cream soups, blancmange, junket, stewed fruit.

TEA (at 4.30 to 5 p.m.).—One of these—Bread and milk, milk gruel, honey and milk. Or milk or weak cocoa with bread and butter, honey or fruit jelly, sponge fingers and plain cakes.

SUPPER (at 8 to 9 p.m., if desired).—Milk and biscuit.

Modifications of Diet according to Age.—After the age of four years meat need not be minced if the child eats slowly and masticates well. Rabbit and veal are permissible if well boiled, minced, and pounded, and pheasant or partridge if freshly killed. If the diet is plain and the child eats slowly, he may have as much as he wants. Breakfast and dinner are the chief meals.

After the age of five years, three meals a day are generally sufficient. Red meat can be given four or five times weekly, and even daily after the commencement of the second dentition. Potted meat or egg sandwiches and sardines can be given at tea-time after the fourth or fifth year, and fish, meat rissoles, or eggs, after the seventh year. Milk and biscuit or fruit may be allowed for lunch, and milk and biscuit at bedtime, are often needed by a growing child. Avoid highly seasoned made-up dishes, fried foods, and indigestible foods, such as pork, tough steak, duck and goose, and nuts, candied fruits, and pineapples. Currants if well cooked, jams containing seeds, and marmalade, are now permissible. If not too new or too decayed, cheese can be given. Salt should be the only condiment. These diets are such as suit the majority of children. They can be extended or modified in the direction of simplicity in accordance with the needs and digestive capacity of the particular child. Some children are made ill by excess of carbohydrate foods, especially those which ferment readily—e.g., jams and sugar. Others suffer ill effects from too nitrogenous a diet, notably one containing meat extractives. Red meat appears to have a stimu-

lating effect on metabolism and the nervous tissues, and must be given in moderation to nervous and excitable children. Many children dislike fats, and can only be got to take them in the form of butter, margarine, dripping, and cream. It is of importance in successful feeding to pay attention to the idiosyncrasies of the child, and not to compel him to take food which he apparently loathes. On the other hand, it is a fatal mistake to give way too readily to the child's fancies, and to tempt him with other foods when he refuses simpler ones which he ought to take. With judgment and discretion children can be taught to take the simple foods, and yet not end with such a distaste for them—e.g., milk puddings—as to loathe them in later life.

DISEASES OF CHILDREN

CHAPTER I

DISEASES OF THE NEW-BORN

F. S. LANGHEAD

INTRODUCTION.

ASYPTYX NEONATORUM.

LOTION NEONATORUM.

EMERY NECTON AND MARTIN.

UMBILICAL INFECTION.

GENERAL SEPTIC (SEPTICEMIA AND
PYEMIA).

TETANUS NEONATORUM.

OPHTHALMIA NEONATORUM.

HERPES.

PERFORATION NEONATORUM.

SCARF-EDINA.

SCLETERA.

TRICHO-SCLETERA.

SCLETEREMIA.

INTRODUCTION.

THE first days of life are a period of change. A new circulation and respiration are established almost suddenly at birth. Thereafter, little by little, the organism adjusts itself to its new and very different environment. Birth itself has its attendant difficulties, which may cause temporary or permanent damage to the newly-born child. Transition from the fetal to the extra-uterine form of respiration is not always accomplished with ease, as the occurrence of *urphyria neonatorum* denotes. Moreover, signs are not wanting that the mechanism does not always run smoothly in its new surroundings.

The pulse-rate still approximates to that of the fetus—being about 130 per minute—and varies with the slightest excitement. *Respirations* are rapid also. At birth they are about 35 to 50 per minute, and afterwards gradually lessen, reaching 30 per minute in a few weeks. They do not become slower until after about twelve months. Besides being quick, the breathing is irregular both in frequency and depth, and often cyclical in rhythm. Even asymmetry of movement may be acquired imperfectly, so that air enters now one part, now another, of the lungs. A systolic murmur is heard not infrequently over the precordia for the first weeks of life, to disappear completely later.

The heat-regulating apparatus is not yet efficient, and the temperature fluctuates widely for slight causes. Exposure to cold soon induces collapse. A rise of temperature to 101° F. or more, known as *infection fever*, is apt to occur during the first week. It is roughly proportional to the loss of body-weight, and is connected with deficient nourishment, for it abates when the flow of maternal milk begins.

The urine at first is clear and of low specific gravity, but almost directly becomes cloudy, and remains so for four or five days. Nucleo-albumin is passed in quite 50 per cent. of all newly-born infants. Its amount is never very great. Often epithelial and hyaline casts are found. Usually after about the third day, but

sometimes earlier, a brick-red sediment is seen, which may stain the diaper. It has been shown to consist of ammonium urate, and is probably associated with deposits of a combination of ammonium urate and a proteid substance, which are found at the apices of the renal pyramids of children who die soon after birth, and which are known as *uric acid infarcts*. The red deposit seldom lasts for longer than a week. There is also a very high percentage of uric acid in the urine of the newly-born (see also Chapter XII., p. 604).

ASPHYXIA NEONATORUM.

Asphyxia neonatorum is usually due to premature stimulation of the respiratory centre. This leads to abortive attempts on the part of the foetus to breathe while still unborn. In the more serious cases exhaustion of the centre supervenes. Commonly the incentive to premature respiration is a deficiency or absence of the placental blood-supply to the foetus during delivery. It occurs, therefore, more frequently when labour is difficult. Twisting or nipping of the umbilical cord, detachment of the placenta, tonic contraction of the uterus, and prolonged abnormal pressure on the foetus in the uterus or genital canal, may each act in this way. Another factor is feeble circulation in the mother, either from heart or lung disease or exhaustion during delivery.

Asphyxia neonatorum is apt especially to follow breech presentation, for in such cases there is often considerable delay in delivering the after-coming head, and respirations are liable to be excited prematurely by cutaneous stimulation of the exposed body of the foetus, and by nipping of the cord by the head. It may be symptomatic of congenital heart disease or of serious intracranial lesions, either produced by injuries inflicted at birth by forceps or prolonged pressure, or due to defective development of the brain.

It is customary to describe two varieties of asphyxia in the newly-born: (a) blue asphyxia (*asphyxia cyanotica*); (b) white asphyxia (*asphyxia pallida*). The former is less serious than the latter, by which, however, it is replaced if treatment is of no avail. The latter is usually fatal. Although stages of transition from one to the other are recognizable, the distinction conveyed by these terms is important, for the treatment of the two forms is not the same.

Asphyxia Cyanotica.—As the name implies, the baby is blue at birth. Its features are bloated and its eyes are closed. It lies with its limbs flexed. Manipulation shows that there is no loss of tone in the muscles. The reflexes are not abolished. Respirations are either absent or shallow, irregular, and ineffectual. Jerkings may be seen. The heart is still acting well, and forcible pulsations can be seen and felt in the umbilical cord. If the skin be stimulated, occasional gurgling and gasping noises accompany attempts at deeper respiration, but unless the condition is relieved by treatment these soon cease, or again become very shallow, and *asphyxia pallida* follows. Air is prevented from entering the lungs by mucus, perhaps accompanied by meconium or amniotic fluid, in the throat and upper air-passages.

TREATMENT.—No attempt must be made to encourage respiration until the obstructing mucus has been removed, for unless this is done it will be drawn into the smaller bronchi with the first inspiration. The cord should not be tied until it ceases to beat. First the mucus must be sucked out of the throat by a catheter.

If no catheter is available, the baby must be held up by its legs and the throat swabbed out with a strip of linen wrapped around the little finger. When all the mucus has been removed, the infant may be made to cry by smacking its buttocks or sprinkling cold water over it. With the advent of normal respiration the colour steadily improves, and the pulsations in the cord gradually stop. The latter can then be tied and cut. If these measures are unsuccessful, artificial respiration must be employed without delay.

Asphyxia Pallida.—The baby has the pallor of death. Respiratory movements have ceased or are scarcely perceptible. The muscles have lost their tone. Reflexes are absent. The heart beats slowly and feebly, and pulsation in the cord is either absent or very slow and weak. The sphincters are relaxed.

TREATMENT.—There is no time to lose. The cord must be tied and cut without delay. The utmost gentleness must be observed lest the heart stop altogether. The throat must then be cleared of mucus, and the baby immersed up to its neck in hot water at 104° F. Whilst in the bath its body should be rubbed thoroughly and its thorax rhythmically compressed. If the heart is still beating, the baby should be taken out of the water, rapidly dried with a warm towel, and artificial respiration started. After six or eight movements of artificial respiration the baby should again be immersed in warm water, its throat freed from mucus, and its body actively rubbed as before. If the heart has not stopped beating, the baby must be dried rapidly, and artificial respiration employed again. These methods of resuscitation—hot baths, drying in a warm towel, and artificial respiration—should be continued in rotation until the child cries or its heart stops.

The question of the best method of artificial respiration is still *sub judice*. The wringing movements devised by Schultz, which are chiefly in vogue on the Continent, and have been adopted by the Dublin School, have been severely criticized by English obstetricians. If carefully and properly carried out they are very efficient. Many accidents, such as rupture of the liver or spleen and fracture or wrenching of the vertebral column, have attended its use, but probably these should more properly be ascribed to lack of skill rather than to any defect in the method itself. If the operator has not become expert in Schultz's movements by practice with a dummy or dead foetus, it is better for him to resort to Sylvester's method, such as is commonly employed to resuscitate the apparently drowned. Labarde's device by rhythmical traction of the tongue is difficult to employ satisfactorily, because the tongue is too small. Insufflation is practised by some. It must be done by inserting into the larynx a catheter, through which air can be pumped by a bellows. As a means of expelling mucus it is very efficacious, but does not prove a satisfactory method of promoting oxygenation, and readily produces emphysema.

DIMENIONS AND PROGNOSIS.—The chief difficulty in diagnosis is in distinguishing simple asphyxia from that which is due to cerebral lesions. Cerebral injury and compression may be detected by signs of damage by forceps or by cranial depression. Frequently it is suspected only because treatment proves useless, although applied while the asphyxia is slight.

If the child survives, local cerebral paralysis or mental defect are regarded by most authorities as indications that a cerebral lesion existed, but, according to Schultz and Jacobi, asphyxia alone may lead to idiocy. Little also enumerates it among the causes of infantile spastic paralysis.

A natural result of imperfect respiration is the persistence of more or less extensive areas of the foetal form of lung (congenital steleotasis). This may cause prolonged respiratory embarrassment (see also Chapter VII., p. 338).

PATHOLOGICAL ANATOMY.—The changes found after death resemble somewhat those characteristic of death by drowning. The right side of the heart and the great veins are engorged. Petechiæ and large hæmorrhages may be found in the serous membranes and viscera, and hæmorrhagic effusions in the serous cavities. The lungs are hyperæmic, and, if the baby has not breathed, are foetal in form and size. If, on the other hand, respiration has partly been established, areas of expanded lung stand out from the darker, firmer, and more sunken patches of steleotasis.

The respiratory passages contain inhaled mucus, and sometimes mucous and amniotic fluid. Often similar material is found in the stomach, into which it has been swallowed during the irregular inspiratory efforts which the foetus made before its birth.

ICTERUS NEONATORUM.

A large number of infants, who are otherwise in perfect health, develop jaundice shortly after birth. Its frequency has been variously stated. Thus, Holt found it in 33·3 per cent., and Cruse in 84·4 per cent., whilst Brochet contends that it is never absent. Usually it begins in from twenty-four to forty-eight hours, seldom after the fifth day. The face is affected first. A faint yellow tinge of the skin and sclerotics is all that can be seen in most cases, but more rarely a deep yellow coloration follows. In the majority the urine remains free from bile pigments as far as can be determined by the usual tests, but examination microscopically reveals clumps of dark pigment, the *mucous powder* of Parrot and Robin, which have been proved to be bilirubin by Cruse. The urine of the newly-born appears to be incapable of keeping bilirubin in solution. Bile salts have been detected in the urine by Halberstam. Simple icterus neonatorum seldom lasts for longer than a week or ten days, and if the colour deepens after this time some more serious form of jaundice is to be suspected. Occasionally, however, as Still has pointed out, jaundice indistinguishable from simple icterus neonatorum persists for two months, or even longer. Apart from sleepiness, no toxic symptoms accompany it, the pulse-rate is not slowed, and the child remains healthy. It is more common in premature infants, and more intense in those who are wasted and undernourished.

PATHOLOGICAL ANATOMY.—*Icterus neonatorum* is not a fatal disease, so that post-mortem examinations have only been possible when some complication has been present. The serous membranes and interior of the vessels and body fluids are stained a yellow colour. The organs participate in this change to a less degree, the spleen and kidneys excepting, as far as can be seen. The liver is little altered, often being only slightly yellow or yellow in patches. Orth has found clumps of bilirubin crystals at the apices of the pyramids of the kidneys, in which situation they are visible to the unaided eye, and also similar crystals in the blood, adipose tissue, and brain and other viscera. That the developing teeth may be pigmented by bile is shown in a case of the writer's, where jaundice dating from soon after birth entirely disappeared, but as each tooth erupted it was seen to be deeply impregnated with bile pigment.

Ætiology.—Many conjectures have been made as to its cause. Some of these have now been proved untenable by the work of Städelmann, Nannyn, and Minkowski, who have shown that all jaundice is hepatogenous. These need not be discussed. Silbermann and others have ascribed the jaundice to a rapid destruction of red corpuscles during the first days of life. This fitted in with the falling blood-count of erythrocytes which is known to exist after about the third day, but it has since been shown that the total number does not diminish; the decrease is apparent only, and is due to an increase in the blood-plasma. There is no evidence of disintegration of red blood-cells. The view of Knöpfelmacher is quite comprehensible. He holds that the bile capillaries become blocked by tenacious bile, the product of hyperactivity of the liver cells immediately after birth, in consequence of their rich blood supply. The newly-formed bile cannot flow out through the overfilled bile capillaries, and therefore passes into those of the blood. Pacchioni believes that the fundamental cause is the loss of water sustained by the infant in the first days after birth. This produces a condensation of the fluids of the body, including the blood, and, if severe, leads to the formation of so viscid a bile that it cannot flow freely along the bile channels, and is absorbed by the blood and lymph.

Diagnosis.—The diagnosis is from the more severe forms of jaundice which occur at this period. Such are pathological, of which there are three specially important—*septic jaundice*, jaundice due to congenital *stenosis or absence of the gall-bladder and bile-ducts*, and jaundice due to *syphilitic cirrhosis of the liver*.

In the case of *septic jaundice* the baby is seriously ill, with considerable fever. The umbilicus is usually the site of infection. Pyogenic micro-organisms may be grown from the septic focus, and sometimes from the blood. In chronic cases *sepsis* sometimes produces jaundice, not by intoxication or blood-infection, but by spread of inflammation along the umbilical vein to the portal fissure, and occlusion of the bile channel by newly-formed fibrous tissue. This was clearly the sequence in a patient on whom the writer made an autopsy. The symptoms were those of obstructive jaundice.

In *congenital malformation of the gall-bladder and bile-ducts*, jaundice is often postponed for one or two weeks, but, being developed, gradually increases in intensity until death. The stools are white from absence of bile. The urine contains bile pigment, but no urobilin, as can be demonstrated by the spectroscope. The liver, and often the spleen also, can be felt to be enlarged. This condition is more fully described in the chapter on Diseases of the Liver (Chapter V., p. 229).

Syphilitic cirrhosis is an uncommon cause of jaundice at this age. It may be suspected if other signs of syphilis are present, especially if this is corroborated by a positive Wassermann reaction.

Other rare forms of *intra-uterine* must be considered.

Glaister has described a form of *hereditary jaundice* which appears soon after birth. In the instance which he recorded a woman had eight children, of whom six died of jaundice within a short time of birth. Her mother had twelve children, all of whom were jaundiced after birth, but recovered. Several of her brother's children were also jaundiced during the first few days of life.

Certain authors (Arkwright, Auden, Busfield) have reported cases of *familial jaundice* in which no history of jaundice in previous generations was obtainable. The principal features of this condition are thus summarized by Auden: (a) The appearance of jaundice in successive children of the same parents; (b) the rapid development, within a few hours of birth, of jaundice, which quickly becomes

profound; (c) a drowsy condition, which ends in convulsions in the fatal cases; (d) a severe anemia following the jaundice, and persisting for a long time in the children who survive; and (e) the hæmatogenous character of the jaundice and absence of hæmorrhages. This variety of jaundice is very fatal, only eight recovering out of thirty-one cases recorded by these observers.

By the title *Kernikterus*, Schmeil, Flannestiel, Bracke and others have denoted a fatal form of jaundice in the newly-born, in which after death the basal-cerebral ganglia are found stained deeply yellow, whilst the rest of the brain is only faintly tinged; the yellow areas show necrosis of the ganglion cells, which is probably the cause of their ready absorption of bile pigment. Most of the cases have been familial.

Congenital family cholemia (congenital arthritic jaundice) sometimes dates from birth also. It is a familial and hereditary disease characterized by fluctuating jaundice, anemia, and enlargement of the spleen, and to a less extent of the liver. The blood-serum contains bile pigment, which, however, is absent from the urine. Bouts of irregular pyrexia occur. The red blood-corpuscles have been shown to be abnormally fragile (see also Chapter V., p. 232).

H. D. Rolleston has recorded a case in which jaundice began at the end of the first week, due to *apylætic inflammation of the walls of the bile ducts*. Death occurred two weeks later.

Jaundice is also symptomatic of the hæmorrhagic diseases described by Winkler and by Böhl (see section on Hæmorrhage, pp. 91, 92).

The severe forms of jaundice may be accompanied by hæmorrhages.

TREATMENT.—Simple icterus neonatorum requires no treatment. The treatment of the more serious pathological varieties is dealt with under their respective causes.

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BREAST SECRETION AND MASTITIS.

Every child, whether male or female, develops more or less swelling of the breast. It begins before birth, but is not noticed usually until the second or third day. For about nine or ten days it increases, and then gradually lessens, to disappear altogether at the end of the third week. Milky fluid (known as "witch's milk") can be expressed from the nipple, and corresponds chemically and microscopically to colostrum. The secretion is unaccompanied by constitutional disturbance, and must be regarded as physiological. Probably the same agent which calls forth the mammary activity in the mother, and which is circulating in the blood, is responsible for a similar reaction in the foetus. According to Halban, this substance is a secretion of the chorionic epithelium of the placenta, whilst others hold that it is a hormone derived from the corpus luteum. The condition requires no treatment.

Sometimes the distended gland becomes infected by micro-organisms. It is said that these may already be lurking in the lactals, but more probably they are obtained from without, gaining admission through the nipple. Cracked or sore

nipples increase this danger. Trauma is often the direct antecedent of infection, especially that injury which is produced by ignorant mothers and nurses in their endeavour to perform the rite of "breaking the nipple strings," a practice which should be forbidden absolutely.

The child is feverish, restless, refuses the breast, and may suffer from diarrhoea or vomiting. Its breast is swollen, red, tender, and oedematous. Suppuration may take place, and one or more abscesses be formed.

PROGNOSIS.—The outlook is favourable, and recovery may be expected in most cases in a few days. The breast has been destroyed to a greater or less extent, and consequently in later life the gland may prove inefficient for suckling.

TREATMENT.—If abscesses form, they must be opened by incisions radiating from the nipple, so as to damage the lacteal ducts as little as possible. Otherwise compresses soaked in lead lotion or weak solutions of alcohol (40 per cent.) may be applied. Manipulation must be avoided.

UMBILICAL INFECTION.

Normally the umbilical cord separates by mummification on about the fifth day, but infection leads to delay and disorder in the process. Before separation could progress is not uncommon, the cord becoming greenish, swollen, and offensive, and leaving a ragged ulcer at its base. After separation a grey granulating area discharging a little serous fluid may persist for a few days. This condition—*omphalitis*—is of little importance. *Umbilical ulceration* is another result of local infection. The umbilical folds are slightly oedematous and reddened, and on separating them an ulcer can be seen, discharging pus or covered by a fibrinous exudate. It may extend to the abdominal wall.

More serious is *umbilical cellulitis* (*omphalitis*). The navel is swollen, inflamed, and often projects in a cone-shaped fashion. Pus is discharged freely. The oedema and redness, sometimes striated by the scarlet lines of lymphangitis, may spread in the abdominal wall as far as the symphysis pubis. Abscess formation is likely to occur. Usually the navel is ulcerated. It is accompanied by considerable constitutional disturbance—fever, restlessness, anaemia and wasting, and vomiting or diarrhoea—and frequently leads to septicæmia. In a few instances the cellulitis gradually subsides.

Umbilical gangrene is, fortunately, very rare. It occurs only in feeble infants who are exposed to the worst hygienic surroundings, and is usually preceded by umbilical ulceration or cellulitis. The destructive process rapidly spreads into the deeper tissues, and may perforate the abdominal wall, permitting protrusion of coils of intestine. Focal fistula and general peritonitis may follow by extension into the gut and general peritoneal cavity respectively. It is almost invariably fatal.

Local infection of the umbilicus may be complicated by inflammation of the umbilical arteries or vein. On the other hand, these vessels may be affected while the navel is normal in appearance. *Umbilical arteritis* or *periaarteritis* may involve the whole length of both arteries, or may be confined to one or a part of one, either continuous with or remote from the navel. The infection spreads usually along the perivascular tissue. In many cases the condition is wholly unsuspected; in others pus is discharged from the navel, and can be shown to emerge from the arteries by examination with a probe. The arteries may be felt like firm cords

through the abdominal wall. The local suppuration sometimes leads to pelvic abscess or general peritonitis. Blood-infection occurs in a few cases.

Unilateral phlebitis and periphlebitis is a less common but more serious condition, and is almost always fatal. Often there are no local signs at the umbilicus. The inflammation spreads rapidly along the vein to the liver, which may become the site of multiple abscesses. Signs of general septicæmia are soon manifest. Jaundice develops, and gradually deepens, until the skin becomes browned. Death follows in a few days.

Hæmorrhage from the navel is usually caused by umbilical infection (see *Hæmorrhage in the Newly-Born*). The umbilicus is also the common portal of entry for the organisms responsible for sepsis in its various forms, for erysipelas, and for fetid necrotiform.

TREATMENT.—Efficient prophylaxis is the most essential treatment. Until the navel has healed, it must be treated with as strict aseptic precautions as a surgical wound. When local infection is present, all that is possible is to apply antiseptic dressings and lotions, to keep the navel covered, to incise areas of cellulitis, to open abscesses if they occur, and to establish free drainage. Collapse must be combated by stimulants. (See also Chapter XX., p. 1077.)

GENERAL SEPSIS (SEPTICÆMIA AND PYÆMIA).

The newly-born child is very prone to septic infection; for not only is its natural immunity low, but there are many unguarded portals by which micro-organisms can gain admission. The chief of these is the umbilicus. The skin and mucous membranes are also particularly vulnerable, because they desquamate soon after birth. Other less common channels of infection are the lungs, conjunctival sac, and middle ear. It is probable that occasionally the septicæmia is perinatal, the infection being communicated from the mother to the child through the placenta.

Among the bacteria which cause sepsis in the newly-born, the staphylococci and streptococci occupy the first place. Others are the pneumococcus, *B. coli commensis*, *B. pyocyaneus*, *B. lactis aerogenes*, Guertner's bacillus, the gonococcus, and rarely a bacillus of the influenzal group of organisms. The modes of infection are various. During birth it is due to contact with vaginal discharges or imperfect surgical cleanliness on the part of the attendants. After birth it may be derived from the hands of the midwife, from the dressing applied to the navel, from cow's milk, and more rarely from breast milk, from dirty teats and bottles, and from the water of the bath. In lying-in hospitals it occurs in epidemic form, being conveyed from one child to another by dirty hands or utensils. It is more common in children fed artificially, probably because the protective substances in which the mother's milk is rich are not available, and because cow's milk so often contains a large number of bacteria.

SYMPTOMS.—In the most virulent forms death takes place in a few hours or days from collapse, without any of the usual symptoms of septic infection having been noticed. In some the disease is clinically gastro-enteritis, vomiting and diarrhoea being associated with a high and variable temperature. Other forms are meningial and purpuric. In the former the child is lethargic, restless, irritable, and subconscient; there is often general rigidity, and convulsions may occur. In the latter the respirations are rapid, irregular, and shallow, and patches of discoloured, with or without bronchial breathing, can be detected in the lungs.

The most characteristic form is that which is sufficiently protracted to display the constitutional symptoms of sepsis, accompanied by metastatic suppurating foci. In these cases the child has a high irregular temperature, is drowsy and apathetic, and becomes gradually thinner and more anæmic. Umbilical suppuration may be obvious. The skin is grey in colour, and often ulcerated over points of pressure such as the sacrum or occiput. Abscesses are formed in the skin or subcutaneous tissues. Suppuration may occur in the serous cavities, and thus empyema, pyopericardium, meningitis, pyogenic arthritis, or peritonitis, may develop. Epiphyseitis may cause separation of one or more epiphyses. The lungs usually contain many foci of broncho-pneumonia, some of which have softened to become abscesses. Septic thrombosis of the umbilical vein may cause multiple abscesses in the liver also.

The urine generally contains albumin, and often pus, blood, and epithelial and hyaline casts. The blood shows a great diminution in erythrocytes, deficient coagulability, and sometimes a polymorphonuclear leucocytosis.

Three symptoms which are characteristic of septic infection at this period are oedema, jaundice, and hæmorrhage. The latter may occur in the skin in the form of petechiæ or large ecchymoses, from the mucous membranes and into the viscera. It may be widely distributed or localized. Hæmorrhage from the umbilicus is to be explained in this way in many instances, or the clinical picture may resemble *melena scæstrorum*. The rare conditions described by Bühl and Winkel are probably to be reckoned as varieties of sepsis also.

DIAGNOSIS.—A focus of suppuration—such as discharging navel—jaundice, and hæmorrhage, offer the best guides to the diagnosis of sepsis. If these are absent, its existence cannot be more than problematical, unless organisms are grown from the blood. This, however, is not possible in most cases.

PROGNOSIS.—The prognosis is always grave. Signs of collapse without local suppuration are omens of fatal import. The younger and smaller the infant, the worse is the outlook.

TREATMENT.—The most important treatment is prophylactic. Scrupulous cleanliness must be observed both at the birth of the child and subsequently. The navel dressing must be sterilized. All utensils must be boiled. Breast feeding must be insisted upon when possible, and the nipples carefully washed before and after each feed. In artificial feeding strict precautions must be taken to keep the feeding-bottle and teats aseptic. The milk should be scalded.

Little can be done when infection has already taken place. Stimulants, such as alcohol, digitalis, and caffeine, may be used to counteract heart failure. Collapse is best treated by injection of normal saline subcutaneously or by the bowel. Abscesses must be opened and empyemata or other collections of pus in serous cavities evacuated and treated by the usual surgical methods.

TETANUS NEONATORUM (TRISMUS NEONATORUM).

Tetanus neonatorum is almost invariably an umbilical infection. It has frequently occurred in epidemics. Osler mentions that in certain of the West Indian islands more than half the mortality among negro children has been due to this cause. In St. Kilda, one of the Western Hebrides, of 120 children 84 died within fourteen days of birth. Routine dressing of the navel with iodoform was employed, and the disease was stamped out (Turner).

SYMPTOMS.—It begins usually between the third and tenth days of life, and may end fatally in a few hours or days. Of thirty-one fatal cases collected by Lewis Smith, twenty-four died within two days of the onset. The earliest symptom is difficulty in sucking due to trismus, which, at first induced by putting the child to the breast, soon becomes persistent. In mild cases this may be the only symptom, but in more severe infection the picture of tetanus as it affects adults is closely simulated. Rigid sardonicus and tonic contraction of muscles causing cyanosis, retraction of the head, opisthotonos, and fixation of the limbs in the flexed position, are all present, at first spasmodically as the result of external stimulation, but afterwards continuously. The muscular spasm makes crying difficult and swallowing impossible. The temperature is not always raised, but is often high and irregular. The navel may be ulcerated or suppurating, but frequently there is only a slight serous discharge or none at all. Even in the first day or two it may not be possible to isolate tetanus bacilli. If the child recovers, the spasm gradually becomes less intense, until it passes off in two or three weeks. The mortality is high, for between 70 and 80 per cent. die.

As in the case of adults, no specific lesion is found after death.

DIAGNOSIS.—The condition is usually easily diagnosed. Spasmodic contractions of muscles may occur in intracranial disease, such as haemorrhage, meningitis, and encephalitis. Knipfelmacher mentions a case where congenital hydrocephalus in a child three weeks old was associated with intermittent tonic spasms. These all occurred in the extremities, and were provoked by handling and moving the child. Recently Sutherland has recorded a case of cerebral hypoplasia in a baby whose symptoms very closely resembled those of tetanus, and included trismus.

PROGNOSIS.—The onset of symptoms within the first week proclaims a fatal issue; the later the appearance of the disease, the greater is the chance of recovery.

TREATMENT.—Unfortunately, the onset of symptoms is an indication that certain of the nerve cells have already entered into affinity with the toxins generated at the umbilicus, but an effort should be made to neutralize those which are still unattached by the injection of antitetanic serum. It is better to divide the dose, and inject part subcutaneously, and part into the subdural space, as recommended by Roux. The most practicable way to reach the subdural space in the first days of life is by lumbar puncture.

The navel should be actively treated by scraping and antiseptics. Tizzoni advocates silver nitrate as the best germicide for the tetanus bacillus. For the rest sedatives, especially chloral and bromides, are beneficial. From half to one grain of chloral hydrate may be given by the mouth or 2 to 3 grains by the rectum every two hours. Chloroform may be used as a temporary measure to allay the spasms.

The patients are difficult to feed, and nasal feeding has to be resorted to in nearly every case.

OPHTHALMIA NEONATORUM.

Ophthalmia neonatorum is the chief single cause of blindness. Cohn has shown that in Germany 31 per cent. of the inmates of asylums for the blind owe their loss of vision to this disease. In the United States, according to Fox, it accounts for 30 per cent. of the blind. A Commission in 1889 found that in the United Kingdom some 7,000 people suffered from blindness so produced. In the great majority of

cases it is due to the gonococcus, and infection occurs during delivery, the eyelids being bathed in the maternal discharges, from which the organisms gain an entrance into the conjunctival sac when the eyes are opened. More rarely the pneumococcus, one or other variety of streptococcus, or *Bacillus coli*, is the offending organism. Other modes of infection are from the hands of the physician or midwife, either while the child is still unborn or after delivery. It may become infected later indirectly from the mother or from other children.

SYMPTOMS.—Generally the disease begins on the second or third day. In about three-fourths of the cases both eyes are affected. The eyelids become more and more oedematous and reddened, until they remain closed. At first the discharge is slightly tinted and contains a few flocculi, but after a day or two it is more abundant, creamy and yellowish-green in colour. On opening the eyes this often spurts out, having obviously been under considerable tension behind the apposed lids. The conjunctiva, both palpebral and globular, is seen to be reddened and oedematous, and after the first few days is granular. Unless blood-infection occurs, the general condition remains good and there is no constitutional disturbance.

In from two to four weeks the discharge diminishes, and, after becoming serous, stops. The conjunctiva loses its granulation and oedema, and the lids return to their normal size.

COMPLICATIONS.—The complication most to be feared is corneal ulceration, since it often causes perforation, and may lead to synechiae, staphylomata, cataract, or panophthalmitis, and destruction of the eyeball. Occasionally multiple ulcers are formed, which by fusion may affect the whole cornea. Other complications are cellulitis or ulceration of the navel and gonorrhoeal arthritis and septicæmia.

DIAGNOSIS.—Suppurative conjunctivitis can scarcely be mistaken, and the only question to be answered is, To which organism is it due? This should be decided by bacteriological examination of the discharge.

TREATMENT.—The prophylactic measures recommended by Crêde have done much to reduce the frequency and severity of the disease. At the Central Blind Asylum of Munich the percentage of cases of blindness due to ophthalmia neonatorum has fallen from 42.9 to 28.4 since this procedure has been in use. It consists in bathing the eyelids of the infant directly after birth with sterilized water by means of pledgets of cotton-wool, and then applying 1 drop of a 2 per cent. solution of silver nitrate to the conjunctiva by a glass rod. Infection after birth can be avoided by proper observance of the ordinary rules of surgical cleanliness. When one eye only is affected, the other must be carefully bandaged after it has been bathed and treated with silver nitrate solution. The baby should lie on the affected side.

Therapeutic measures should be directed to lessening the inflammation and removing the discharge as quickly as possible. Cold compresses made of six or eight layers of gauze wrung out of iced water may be applied, but need to be changed frequently, as they soon become sodden with pus. The discharge may be removed by douching every half-hour or hour with weak antiseptic lotions or boiled water. Boracic acid (3 per cent.), permanganate of potash (1 in 1,000), or perchloride of mercury (1 in 5,000), are suitable solutions for the purpose.

Once daily the conjunctiva should be anasthed over with a 2 per cent. solution of silver nitrate. Atropine sulphate (0.5 per cent.) must be dropped into the eye immediately haziness of the cornea is detected.

HEMORRHAGE.

For purposes of description, the hemorrhages to which the newly-born are prone may be classified thus:

- A. Those mechanically produced at birth:
 - (i.) Cephalhematoma.
 - (ii.) Intracranial hemorrhage.
 - (iii.) Hematoma of the sterno-cleido-mastoid muscle.
 - (iv.) Visceral hemorrhage.
- B. Vaginal hemorrhage.
- C. Umbilical hemorrhage.
- D. Hemorrhage due to infection:
 - (i.) Syphilitic.
 - (ii.) Septic.
 - (iii.) Epidemic hemoglobinuria (Winkler's disease).
 - (iv.) Bñhl's disease.
- E. The "hemorrhagic disease" (*Morbus neonatorum*).
- F. Hemorrhage due to severe jaundice.
- G. Hemophilia.

Two of these conditions, sepsis and jaundice, have already been considered; the others will now be described.

A. Hemorrhage produced mechanically at Birth.

(i.) **Cephalhematoma.**—A cephalhematoma is an effusion of blood beneath the periosteum of the bones forming the cranial vault. It may be produced during delivery, but more often is detected first two or three days after birth. Its usual situation is over the right parietal bone, but it may sometimes be seen over both parietals, or over the frontal, occipital, or temporal bones. The tumour is firm, and its outline may be obscured by oedema of the soft tissues lying over it. When this has been absorbed its situation may be readily demonstrated. The scalp over it can be lifted up and shown to be separate, whilst its borders are strictly limited by the cranial sutures, where the periosteum is more firmly adherent, and thus escapes detachment by the effused blood. In this way it forms a rounded tense swelling, whose sides slope downward with more or less abruptness to the edge of the bone over which it lies. At first it increases, reaching its maximum usually in about a week, when it varies in size from that of a hazelnut to that of a hen's egg, but occasionally is considerably larger. The scalp may show signs of injury, such as laceration or hemorrhage, but is often quite normal, except for the early oedema.

A few days after its formation softening occurs near its centre, and fluctuation may be detected. The contrast between the fluid central area and its margin is often so great that on palpation it appears that a hole in the skull exists—an impression which may lead to a mistake in diagnosis if the position of the tumour is overlooked. This bony ring is easily explained if we consider that hemorrhage occurs into the osteogenetic tissue between the periosteum and developed bone. With the separation of the periosteum, the most superficial part of the bone-forming

layer is also detached, and, continuing its function in its new situation, produces a bony shell, thickest at the peripheral part of the tumour, and thinning gradually towards its summit. It may ultimately form a complete covering for the smaller hematomata. In some cases the effused blood is quickly absorbed without new bone formation, and no trace of the hematoma remains. In others the process takes several weeks, or even three or four months, and a definite ridge marks the site it occupied.

DIAGNOSIS.—Caput succedaneum may cause difficulty in diagnosis during the first few days, but usually is easily distinguished by the readiness with which it pits on pressure and by its position, for it is not limited by the cranial sutures. Meningoceles can be emptied by pressure, become more tense when the infant cries, and, those produced by trauma excepted, are found in the region of the sutures and fontanelles.

COMPLICATIONS.—Cephalhematoma may be accompanied by evidence of injury during delivery, such as cranial deformity or fracture of cranial bones, laceration of, or hemorrhage into the scalp, and meningeal or cerebral hemorrhage. Not infrequently asphyxia neonatorum occurs. Very rarely blood is also poured out between the cranial bones and the dura mater, producing a condition which has been called *cephalhematoma internum*. This blood communicates with that under the pericranium by a fissure in the bone, either resulting from fracture or developmental in origin. Occasionally the blood-tumour becomes infected and converted into an abscess. If this occurs, the condition, which is usually of little moment, becomes very serious, and may cause death by spread of the inflammatory process to the bones or meninges, or by septicæmia.

ÆTIOLOGY.—The most acceptable view is that cephalhematomata are due to rupture of the fragile vessels of the subperiosteal tissue, which are in a condition of hypoxæmia and stasis. This is in keeping with the fact that they are usually found over the right parietal bone, for this is the presenting, and therefore least supported, part in the majority of births. They are commoner in first-born babies, and especially in the first-born of elderly mothers, indicating that the resistance of the maternal soft parts is a contributing factor. They are also common in male infants and in infants delivered through small pelvis. Additional evidence of the importance of congestion as a cause is the frequent association of asphyxia neonatorum.

In a few cases the position of the effused blood corresponds to a local injury, such as that produced by forceps, or pressure against the maternal pelvis, but in the majority the labour has been devoid of special difficulty. Frisch is of opinion that the hemorrhage is not due so much to congestion as to dragging on the scalp. He believes that during labour the scalp adheres closely to the soft parts of the parturient canal. In the intervals between the pains the head recedes, but the scalp, being held by the soft parts, is pulled away from the bone. Frequent repetition leads to a mechanical loosening of the pericranium, and rupture of the vessels beneath it.

TREATMENT.—A simple pad and bandage to protect the skin and prevent injury is all that is required as a rule. Very large hematomata may be opened, emptied of blood-clot, and closed again by suture, but the risk of infection makes this procedure unjustifiable in any but exceptional cases. Puncture and aspiration add an element of danger which it is quite unnecessary to run. Compression

bandages are such only in name, unless they are adjusted so tightly as to endanger the vitality of the scalp, and are better avoided.

If suppuration occurs, free incision and drainage must be employed without delay.

(ii.) **Intracranial Hemorrhage** is occasionally produced at birth, but is rare. The bleeding is usually meningeal, scarcely ever into the brain itself. Hemorrhage into the lateral ventricle may occur. It is recorded by Spencer that, among 6,088 deliveries, death of the foetus during delivery or soon after birth was caused by meningeal hemorrhage in fifty-three cases. Hemorrhage into the brain tissue occurred in only one normally-formed foetus, and intraventricular hemorrhage in seven. Such hemorrhages, whether meningeal or cerebral, follow difficult and protracted labours, and are often accompanied by signs of pressure by forceps and by consequences of congestion or injury, such as cephalhematoma and asphyxia.

Many of these children are born dead, or the signs of intracranial hemorrhage are obscured by fatal asphyxia neonatorum. In others cerebral compression is denoted by a slow pulse, shallow, grouped, or Cheyne-Stokes respirations, bulging and tense fontanelles, convulsions, and paralysis. These symptoms are usually manifest soon after birth, but in some cases are delayed for four or five days. The convulsions may be general, unilateral, or confined to one limb or part of a limb. They may be only temporary, perhaps giving place to paralysis, or, if the child lives, may persist. Cerebral diplegia is ascribed to hemorrhage produced at birth, but the first signs often fail to appear for several weeks after birth. Finkelstein has diagnosed a case of meningeal hemorrhage in the newly-born by finding altered blood in the fluid withdrawn by lumbar puncture. When the condition is not rapidly fatal, and localizing signs are present, the skull should be trephined and the blood-clot removed. Cushing has recorded the cases of four infants in whom the operation was performed; two recovered (see also Chapter XIV., p. 797).

(iii.) **Hematoma of the Sterno-Cleido-Mastoid Muscle** occurs as the result of rupture of its fibres by injury during delivery. Blood is extravasated, and finds its way under the muscle sheath and between the muscle fibres, especially between those which are torn. The right sterno-mastoid muscle is the more often affected; rarely the condition is bilateral. A small tumour, the size of a marble or a walnut, forms in the muscle at the site of the injury, which is generally at the junction of its upper and middle thirds. It is rather indefinite in outline, and almost cartilaginous in consistence. It is not painful, but at first may be tender on pressure. Usually the tumour is not noticed until a week or ten days after birth, and disappears in about twelve months.

Torticollis may occur, the chin being directed in some cases to the same side, in others to the opposite. It is usually temporary, but permanent torticollis may be caused by shortening of the muscle. If the tumour be examined microscopically in its early stages, young connective tissue is seen, and torticollis dating from early childhood, and associated with a mass of fibrous tissue in the usual position of a hematoma, undoubtedly occurs. In one case seen by the writer there was a history that during infancy a "lump" had been present at the same site.

Apart from this rare sequel, its course is entirely favourable, and complete recovery follows almost invariably.

ETIOLOGY.—As a rule the muscle fibres are ruptured by being mechanically overstretched, and this can only be brought about, in unassisted labours, by extreme rotation of the head. In *artificial* delivery the same factor is chiefly responsible.

Rupture occurs most frequently in occipito-posterior and breech presentations. Most cases cannot be ascribed to direct violence, the result of manipulative interference, but forceps, the obstetrician's fingers, and the umbilical cord, have each been blamed.

TREATMENT.—No treatment is necessary in most cases. Gentle massage with a lubricant may be employed after the first few weeks. If torticollis threatens, passive movements may be tried, to stretch the affected muscle.

(iv.) **Visceral Hemorrhage.**—Hemorrhages are usually found in one or more of the important viscera of children who are still-born or die shortly after birth. Herbert Spencer has made 130 autopsies on such children, and found that, although visceral hemorrhage may occur under any condition of labour, it is more common and severe in those who are subjected to much pressure by the parturient canal or by the instruments or hand of the attendant. It affects especially those who are delivered by the lower extremity. Cerebral hemorrhage excepted, hemorrhage into the viscera is more frequently met with in pelvic than in cephalic presentations. In most instances it is probable that the hemorrhage is accountable for the death of the child. In 136 post-mortem examinations, Spencer found hemorrhage into the brain or meninges in 63 cases, into the spinal cord in 39, into the liver in 37, into the lungs in 25, into the testes in 15, into the kidneys in 34, into the suprarenal glands in 27, and in a few instances into other viscera.

In the *liver* the hemorrhage is usually found on its anterior surface, beneath the capsule, which it strips up, flaps containing blood being formed. Hemorrhage into the *spleen* may occur beneath the capsule or into the cortex or medulla, but is commonest in the loose tissue of the hilum. Many deaths in the first few days after birth are due to hemorrhage into the *lungs*. Such children never breathe well, but are cold and blue, with a subnormal temperature. Dullness may be detected over the lung, accompanied by weak breath sounds. Generally the blood is poured into the pulmonary bases, but the greater part of a lobe or lung may be affected.

Suprarenal Hemorrhage in early life is particularly interesting. Hamill was able to collect ninety cases of children in whom this condition occurred. These include twenty-eight still-born infants. From careful analysis he divided them into three groups: (1) Those in which death occurs during or before delivery, due to manipulation and trauma; (2) those in which it occurs between birth and detachment of the cord, due chiefly to infection of the cord; and (3) those dying afterwards, in which the cause is unknown, but is probably of an infectious or toxic nature. By far the greatest number fall into the first group.

The right suprarenal is more often affected than the left; occasionally hemorrhages are found in both. If the hemorrhage is slight, it is limited to the medulla, but sometimes the glands are converted into sacs containing fluid blood. This may burst through the cortex and peritoneum, and be found in the peritoneal cavity. Purpura is a common accompaniment. Death may be hastened by convulsions.

Examples after the first few days of life are rare, but form a definite clinical group (see Chapter X., p. 577).

The chief causes of visceral hemorrhages in the newly-born are mechanical squeezing of the blood into certain areas during the act of birth, and external violence by rupturing vessels at the points pressed upon. The delivery of the vessel walls is a contributing cause, for the hemorrhage occurs chiefly in those

organs which contain many thin-walled vessels, and in parts where they are less supported. Spencer disparages the importance of asphyxia as a cause.

Visceral hemorrhages after the first day or two of life may be due to infection of the cord, to syphilis, or to the "hemorrhagic disease" of infants (*melena neonatorum*). Matter explained the frequency of hemorrhage into the suprarenal bodies by their situation behind the liver. He believed that the large heavy infantile liver is pressed back against the posterior abdominal wall during labor, and so squeezes the inferior vena cava against the vertebral column, damming back the blood into the suprarenal glands, and causing it to burst its bounds.

B. Vaginal Hemorrhage.

Bleeding often occurs from the vagina of newly-born girls. It is not comparable to other hemorrhages at this period, for it is unaccompanied by any disturbance of their general health. As a rule it begins on the fifth day, and seldom lasts less than twenty-four or thirty-six hours, although it may recur. It is slight in amount, not enough to appear as drops, and in only a few cases soils the diaper. Examination of the vulva reveals a little blood-stained mucus, or perhaps some small clots, which may be traced to the hymeneal orifice.

Halban speaks of the condition as a "pregnancy reaction." He has demonstrated that newly-born girls, with few exceptions, show congestion of the uterus, accompanied by subepithelial hemorrhages, and sometimes hemorrhages into the uterine cavity—changes similar to those found in menstruation. In adults these are excited by a function of the ovaries; in the newly-born, according to his view, by substances circulating in the blood during pregnancy, derived from the placenta. Soon after birth the mucosa of the uterus returns to its normal state, and the uterine body shrinks, postpartal involution being completed in about three weeks. Regarded in this way, it is analogous to the signs of prostatic activity in newly-born boys, in whom Schlichta has described hyperemia, secretion, and sometimes hemorrhages and infiltration in these glands. The secretion of colostrum and milk by newly-born children has been explained in the same way.

Quite apart from this condition, vaginal hemorrhage is very rarely one of the manifestations of the hemorrhagic diseases of infancy.

C. Umbilical Hemorrhage.

With Knöpfelmacher, we may divide the hemorrhages from the umbilicus into three groups. In the first the bleeding is arterial, and occurs from the stump of the cord immediately, or at most a few hours, after birth, in spite of ligation. It is evidence that the normal occlusion of the umbilical artery has failed, and is due to an unusual blood-pressure in it. This results from imperfect establishment of the adult circulation, such as occurs in asphyxia neonatorum, pulmonary atelectasis, and congenital heart disease. Those cases which occur a few hours after birth are explained by shrinking of the cord and loosening of the ligature.

The second group of cases is much smaller, and comprises those in which bleeding occurs from the arteries at the level of the umbilicus during separation of the cord. It begins usually towards the end of the first week, and is due to local sepsis.

In the third and largest group, that of *perichthysium umbilicale* hemorrhage, the blood comes from the smaller vessels and capillaries at the junction of the skin and umbilical cord. It may occur before or after separation of the cord, but is

uncommon after the second week. It usually indicates some severe constitutional disorder, such as sepsis or syphilis, and may be part of the condition known as "melena necrotorum." For these reasons it is often associated with bleeding elsewhere, and is generally fatal. The mortality, according to Grandjéan, is 83 per cent.

TREATMENT.—The application of another firm ligature is usually sufficient to stop the hemorrhage which comes from the cord. In the other cases a 1 in 20 solution of supracornal extract or saturated solution of calcium chloride may be tried, combined with pressure by a firm pad. If this is unsuccessful, a long needle may be passed under the umbilicus from side to side, and a coarse silk ligature wound round this in a figure-of-eight manner, so compressing the umbilical vessels:

D. Hemorrhages due to Infection.

(i.) **Hemorrhage due to Syphilis.**—Although most cases of hemorrhage in the newly-born cannot be attributed to syphilis, there is little doubt but that it accounts for a few. Abt thought that two out of a series of twelve were so caused, and Wilson records ten in which syphilis was present in either the parents or the infant. The bleeding starts at birth, or very soon afterwards, and occurs from the navel and mucous membranes, and into the skin. Jaundice is often present. The bleeding is extensive and usually fatal, and on post-mortem examination hemorrhages may be found in the viscera and serous cavities, associated with syphilitic changes. Kundrat has pointed out the occurrence of syphilitic cirrhosis of the liver, with bleeding from the bowel, in the newly-born, probably due to portal congestion.

The researches of Mrazek afford an explanation of some of these cases. In hereditary syphilis he describes an endarteritis of the small vessels, and changes in the walls of the capillaries, which may lead to complete occlusion of their lumina: Venous stasis is so produced, and hemorrhages follow. Both he and Esser record cases in which this condition was found in the smaller vessels of the intestine in infants.

(ii.) **Hemorrhage due to Sepsis** has already been dealt with.

(iii.) **Epidemic Hemoglobinuria** (Winckel's Disease).—This very rare disease was first fully described by Winckel in 1879, but attention had been drawn to it previously by Charrin (1873) and Bigelow (1875). It occurs in epidemics usually in lying-in hospitals, and is very fatal, almost invariably causing death in a few days from coma, convulsions, or collapse. It begins on the second to the fourth day, and is ushered in by restlessness, vomiting, and diarrhoea. The pulse and respiration become rapid, and the child bronze-coloured from a combination of jaundice and cyanosis. The temperature is only slightly raised. Prostration follows, often accentuated by convulsions. The urine is passed with straining, and usually contains methemoglobin, but sometimes red blood-corpuscles. Albumin and blood-casts may be present also.

PATHOLOGY.—Fatty degeneration and hemorrhages have been found in the various organs. The spleen is enlarged and shows granular masses of blood-pigment. The liver and kidney are usually swollen also. The blood is much thickened and contains numerous granules.

The etiology of the condition is unknown, but its epidemicity and symptoms make it almost certain that it is due to a very virulent infection, capable of producing rapid destruction of the blood-cells. No local source of infection has been discovered.

(16.) **Buhl's Disease** is also exceedingly rare. Feeble respirations and cyanosis may be present from birth. At about the fifth or sixth day edema develops, accompanied by increasing feebleness, anoxia, jaundice, and hemorrhages into the skin or viscera, and from mucous membranes. Death from collapse ensues in about two weeks.

The changes found after death resemble those found in acute yellow atrophy or phosphorus-poisoning. Fatty degeneration is far advanced in the liver, kidneys, and heart. The walls of the pulmonary alveoli and the voluntary muscles are also affected. The viscera show acute parenchymatous necrosis.

Like Winkler's disease, it seems to be due to an infection, possibly through the umbilicus, but its cause has not yet been elucidated.

E. The "Hæmorrhagic Disease" (*Melena neonatorum*)

When other varieties of hæmorrhage in the newly-born have been considered, there still remains a small group to which the somewhat inappropriate title of *melena neonatorum* has been given. Forty-five cases occurred in 5,700 births (Townsend). Twice as many male infants are affected as female. The aetiology of this condition is obscure.

Bleeding usually begins about the third day, but may date from birth, or be delayed for a week or more. The commonest source of bleeding is the bowel, from which it proceeded in twenty of a series of fifty cases collected by C. W. Townsend. At first it is slight in amount, the meconium being stained with dark blood or containing a few small clots, but in a short time the stools may consist of blood only, either fluid or as a mass of clots. Hæmatemesis occupies the second position in point of frequency. It occurred in fourteen of Townsend's cases. It usually accompanies melena, but may exist alone. In forty-two cases reported by Silbermann, hæmatemesis and melena were both present in 23 per cent., and hæmatemesis alone in 16 per cent. Blood may also come from the mouth, the nose, or the ear, and hæmorrhages may be found in the serous spaces, including the meninges, and in the viscera, especially the suprarenal bodies. The temperature is raised at the onset, but collapse soon follows, denoted by subnormal temperature, feeble, rapid pulse, shallow respirations, coldness, pallor, and suppression of urine. A fatal issue is to be feared on the third day. Jaundice is frequently detectable, though not severe.

The condition is always serious, usually fatal. Of Townsend's fifty cases, thirty-one died and nineteen recovered. However, Still records seven cases, of which four recovered.

PATHOLOGY.—Nothing except hæmorrhages is found in most instances. Sometimes hæmorrhagic, hyperæmic areas have been seen in the gastro-intestinal mucous membrane, which occasionally has shown some superficial erosion. Punched-out ulcers, similar to the peptic ulcers of adults, have been noted in the stomach, duodenum, and œsophagus. Moynihan was able to collect sixteen examples from the literature in which the duodenum was the site of such an ulcer. It is possible that these account for the bleeding, but more probably they mark the site of hæmorrhages, which have led to necrosis, the necrosed area being digested and absorbed, and an ulcer remaining. This is borne out by the occasional association of hæmorrhages elsewhere.

DIAGNOSIS.—Symptomatic hemorrhage, such as that due to sepsis, syphilis, or local gastro-intestinal disorder, must be considered. Hemorrhage which originates by sepsis will not appear directly after birth. The condition of the umbilicus may supply the clue to the diagnosis. Unless a bacteriological examination of the blood can be made during life, it is impossible to exclude sepsis in any particular case, so that it is more than likely that many cases of so-called "hemorrhagic disease" are really examples of septic infection in the newly-born. Syphilis may be detected by the Wassermann reaction. An enlarged liver may be felt in that form of bleeding ascribed by Krunder to syphilitic hepatic cirrhosis. Other signs of syphilis should be looked for. The blood, vomited or passed by the bowel, may have been swallowed, and have its source in ulceration of the mouth or lips, syphilitic rhinitis, or lacerations of the mouth or pharynx produced during delivery. Sometimes it is derived from the mother's breast if her nipples are cracked. Such sporadic hemorrhages are usually only slight, and are unaccompanied by constitutional disturbance.

TREATMENT.—Satisfactory results have followed the use of gelatine. A drachm of a 5 per cent. solution may be given by the mouth every hour, or an absolutely sterile preparation injected subcutaneously. For subcutaneous injection a 10 per cent. solution may be employed, 2 drachms being injected two or three times daily. Friswald recommends the following formula:

Gelatin, alba (Merck)	gr. xxx.
Sol. chlor.	gr. ii.
Aq. destill.	℥ss.

One ounce should be given hourly by the mouth. Adrenalin solution, ergot, and perchloride of iron, are also used.

Recently J. E. Welch has advocated the use of normal human serum, 10 c.c. being injected subcutaneously three times a day. This dose may be increased or given more frequently in the more severe cases. The blood is collected from the median basilic or cephalic vein in a sterile flask, and the serum is drawn off as soon as it separates. It is then ready for use. He reports twelve cases in which this treatment was employed; all recovered.

F. Hemorrhage due to Severe Jaundice.

This is considered elsewhere, and needs no special description here (see Chapter V., p. 208).

G. Hemophilia.

Hemophilia is now regarded as a very rare cause of bleeding in early infancy; indeed, it is open to doubt whether it occurs at this period. Grandisier reported 576 bleeders, of which only twelve had bleeding from any source during the first few days of life. In the past the term has been used to cover all the varieties of hemorrhage in the newly-born for which no satisfactory explanation is forthcoming.

It may be suspected if a family history of hemophilia is obtainable.

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PENPHIGUS NEONATORUM.

Cases of pemphigus in the newly-born may be divided into syphilitic and non-syphilitic groups. The former is prone to affect the palms and soles (where it causes coarse desquamation), parts which in non-syphilitic pemphigus always escape. In the milder and non-syphilitic forms a few bullæ appear either synchronously or in crops, and are scattered widely over the body. Occasionally they occur on the mucous membrane of the mouth and palate, rarely on the scalp. In a few days the bullæ shrivel or rupture, and if they have not become infected a dry crust is formed, covering a superficial ulcer. When the crust has peeled off, a red circular area of skin remains. Sometimes the eruption is thicker and more diffuse, leading to almost general desquamation. This is particularly so in the syphilitic cases. The bullæ may suppurate.

It is probable that non-syphilitic pemphigus is not always evidence of the same infection. In some cases the baby is happy and well-cared for, and seems little the worse for its eruption; in others it is piny and wasted; and in yet others the temperature is high and irregular, and severe constitutional symptoms are present, with or without signs of local sepsis. The disease sometimes occurs in epidemics, and frequently one midwife may infect a series of children. *Staphylococci* and *streptococci* have been obtained from the bullæ on many occasions.

Syphilitic pemphigus is detected by the distribution referred to above, by other evidences of syphilis, and by the Wassermann reaction. *Spirochæta* have been found in the blæbs. Subsequently the skin may be pigmented in the characteristic "rusty" manner.

TREATMENT.—Soaking the blæbs and dusting them with equal parts of calomel starch and boric acid is the most effective way of dealing with pemphigus which is due to sepsis. In the marasmic cases cod-liver-oil and iron are indicated, and attention should be directed to the feeding. For syphilitic pemphigus anti-syphilitic treatment is required. For other cases arsenic may be tried; but its effect is not so striking as in the pemphigus of adults or older children (see also Chapter XXIII, p. 1153).

SCLERO-CEDEMA, SCLEREMA, PSEUDO-SCLEREMA, AND SCLERODERMIA.

There is no doubt that much confusion still exists concerning the manner by which the several members of this group of diseases may be distinguished the one from the other. Since the work of Denis and Billard, Continental authorities have adopted, for the most part, the sharp differentiation which they drew between sclero-cedema and sclerema, conditions which had been considered similar hitherto. On the other hand, quite recently, Comba at a congress of the Italian Pediatric Society would admit no distinction between them, and even those who hold that they are different agree that both may occur together in the same patient. English authors have been slow to accept the conclusions of Denis and Billard, and generally describe one condition only, which they designate "sclerema." Some of the cases which they have recorded under this title do not conform to the clinical picture of either sclerema or sclero-cedema. Lushien regards them as examples of scleroderma occurring in early life.

To avoid further misunderstanding, I shall describe *sclero-odema*, *sclerema*, and *sclerodermis* as separate diseases, according to the view which is held by most authorities, and for the present shall keep apart the anomalous English cases under the title "pseudo-sclerema," as has already been done by John Thomson.

TABLE SHOWING THE CHIEF DIFFERENCES BETWEEN *SCLERO-ŒDEMA*, *SCLEREMA*, *PSEUDO-SCLEREMA* AND *SCLERODERMIS*.

	<i>Sclero-Œdema</i>	<i>Sclerema</i>	<i>Pseudo-sclerema</i>	<i>Sclerodermis</i>
General condition	Premature, weakly, or wasted	Premature, weakly, or wasted	Not necessarily weakly	Not necessarily weakly
Season	Winter	Summer	Any	Any
Age	First week	Birth to sixth month	In first weeks	Any age
Distribution	Spreads from extremities; leaves connective-tissue spaces	Spreads from calves; confined to parts where fat is present	Backs, back, etc.; patchy and circumscribed	General, symmetrical, or localized
Consistence	Swelling; which pits	No swelling; does not pit	Slight swelling; does not pit	Begins as oedema; later hard, and does not pit
Appearance of skin	Cyanotic, mottled, or white	Yellowish or white	Bluish-pink, livid, or white	White and shiny
Temperature	Subnormal	Extraordinarily low	Normal	Normal
Constitutional disturbance	Considerable	Profound	Slight or none	None
Course	Rapid; frequently fatal in a few days	Rapid; nearly always fatal in a few days	Slow; usually recovers	Slow; always recovers

Sclero-Œdema (*Sclerema Œdematosum*) is not a very uncommon condition. It occurs particularly in premature infants, twins, and those who are weakly and undernourished or syphilitic. It is more prevalent in the winter months and in cold climates.

It is confined to children in the first week of life, usually beginning on the second to fourth day, very rarely after two weeks. Exceptionally it is congenital.

SYMPTOMS.—After a short period of restlessness and refusal of food, *sclero-odema* makes its appearance usually on the dorsa of the feet, in the lower part of the abdominal wall, and on the cheeks. These parts become swollen and tense, and can be pitted by firm pressure, but their consistence is harder than that of simple oedema. The underlying muscles also are infiltrated with a fluid exudate. Rarely the tension is so great that pitting is impossible. The skin varies in colour according to the age of the infant. If *sclero-odema* occurs before desquamation has taken place, it has the parboiled appearance characteristic of the first day or two of life. In others it is cyanotic, waxy, or mottled. It can be raised from the deeper tissues.

The swelling rapidly spreads upwards, and may affect the whole body except the front of the chest. Œdema of the penis and scrotum is nearly always present.

In severe cases the general swelling causes the child to be stiff and motionless, and its skin is cold to the touch. The temperature is subnormal, being about 95° F. in mild cases, but sinking as low as 90° F., or even lower, in those which are more severe. The respirations are slow and the pulse weak. The urine is small in amount, but seldom contains albumen. Death may take place in a few days, the child becoming more and more stuporous, and its heart's action increasingly feeble until collapse follows. Frequently the course is more protracted and ends in recovery.

MORBID ANATOMY.—After death the skin, subcutaneous tissues, and muscles, are seen to be soddened with a watery exudate. The mediastinal tissue may be affected similarly. Venous stasis and congestion of the organs, especially those of the abdomen, is obvious. Not infrequently the lungs, besides being congested, are the site of hemorrhages, which are usually small, but occasionally extensive.

Microscopical examination reveals no specific lesion. The epidermis is often shallow and ill-developed, but this is only to be expected in premature or feebly-nearly-born infants. The subepidermal tissues are swollen, and their elements separated by the exudate. The lymphatic spaces and vessels are dilated.

Occasionally the heart muscle shows some fatty change.

PATHOGENESIS.—It is questionable whether sclero-odema should be considered a distinct and independent disease. Luithlen holds the contrary view, and believes the peculiar form of the oedema is to be explained by the age and feebleness of the child, for the skin and subcutaneous tissues, and especially the vessels, are not yet fully developed. Feeble circulation and respiration with deficient oxygenation are also influential in producing the condition. Another factor which is of first importance is cold. Given a feeble or premature infant and exposure to cold, it is probable that any condition which produces vascular congestion may cause sclero-odema.

TREATMENT.—Warmth is essential, and is best afforded by the use of an incubator. Hot baths and massage may be tried. Radaboni recommends injections of glycerine to which 10 per cent. of iodide of ammonium has been added. Breast feeding must be employed if possible, but it is often necessary to feed nasally or by the bowel.

Sclerema (Sclerema Adiposum).—According to Luithlen, the earliest reference in the literature to sclerema is by a doctor at Ulm, who wrote "*De Febre Rigida et Frigida*," in 1718. The first exact description of the condition we owe to Denman, a physician of the Middlesex Hospital, who is quoted by Underwood in a "*Treatise on Children*," in 1784.

It is a very rare affection, and is much less common than sclero-odema. It occurs not only in the first few days after birth, but at any time up to the sixth month in weakly, premature, and marasmic infants. Very seldom is it congenital. A majority of the cases follow severe infective diarrhoea, and thus it is met with more frequently in the summer. In others there is no predisposing disease.

SYMPTOMS.—Like sclero-odema, it begins in the most dependent and peripheral parts, but selects those which are richest in fat, and not those which contain the most subcutaneous loose cellular tissue. Usually the calves are affected first and in a symmetrical manner. The condition soon spreads until the thighs, trunk, and neck, are involved. Last to be attacked are the head and upper extremities. The penis and scrotum (parts in which fat is almost lacking), and the palms and

soles (parts in which the fat is subcutaneous) escape. The skin is not raised, but flat and board-like, and yellowish or marble white in colour. It cannot be picked up or stretched, nor can it be indented by the finger. Echinomata sometimes appear, especially on the legs.

If the whole body is affected, the child lies absolutely rigid, like a statue, his face mask-like, and his limbs slightly flexed.

Serious constitutional disturbance is always present. Usually the temperature is subnormal, being often under 90° F., whilst such extraordinarily low temperatures as 79° F. (Vernon), and even 72° F. (Roger), have been recorded. Respirations are as slow as 18 or 16 per minute, and the pulse-rate falls to 80, 60, or even 30, per minute. The mucous membranes are dry. The urine is very scanty, highly-coloured, and may contain albumen. Convulsions may occur. Feeding is very difficult, and death by coma is to be expected in from two to eight days. Very rarely the course is more protracted and ends in recovery (Rennell). If the disease is complicated by pneumonia or other infection, fever may be present throughout.

MORBID ANATOMY.—The most striking change found after death is an extreme dryness and hardness of the skin and subcutaneous tissues. On incision, little or no fluid escapes. They cut and appear like stearin. Atelectasis is usually present. Sometimes evidences of gastro-enteritis are visible, and occasionally of complications, such as pneumonia, bronchitis, or pleural effusion. Small hemorrhages may be seen in the peritoneum, the pleura, and the meninges, and between the spinal cord and its membranes. The heart and liver may show fatty degeneration.

Sclerema has been studied histologically, especially by Clementowsky, Parrot, Northrup, Ballantyne, and Laithlen. All are agreed that no gross changes exist. The rete is normal or slightly thinned. The fibrous bundles of the corium are arranged more closely than usual, its vessels being empty and constricted; the fatty layer is much in evidence, contains large numbers of fatty acid crystals, and is traversed by more or less broad connective-tissue bundles. Where the fibrous strands are broadest the cells of the enclosed adipose tissue are smaller, and contain unusually obvious nuclei, whilst where they are finer the fat cells are larger. There is nothing characteristic about these changes. They originate merely from the emaciation and marasmus to which the child is reduced.

PATHOGENESIS.—Billard believed that the condition is to be explained by solidification of fat. He thought that this change normally occurred at death, but in sclerema is produced prematurely by loss of heat from any cause. Schindler, Ritter, Saltmann, and others, emphasized the truth that loss of fluid is a common precursor, and ascribed sclerema to drying up of the body. Langer and Knögelmeier have made chemical examinations of the fat in newly-born infants. The latter considers that three factors enter into the causation of this disease: (1) loss of fluid, (2) lowering of temperature, (3) the high solidifying-point of infant's fat. This is due to the small proportion of oleic acid, which amounts to only 43.3 per cent., and does not equal the proportion found in the fat of adults—namely, 65 per cent.—until the child is twelve months old. On the other hand, Thiensch and Siebert state that there is no deficiency in oleic acid.

Other hypotheses are that the disease is bacterial in origin, or arises from vasomotor disturbance, but there is little to support these conjectures.

DIAGNOSIS.—It is distinguished from sclero-odema by the absence of swelling, the board-like induration, fixity and colour of the skin. Unlike sclero-odema, it

cannot be pitted by pressure. Moreover, it is not confined to the first days of life. The temperature is lower, and constitutional disturbance and prognosis worse. It occurs usually in summer, whilst sclero-odema is commoner in winter.

The resemblance between sclerema and that stage of sclero-odema when the subcutaneous exudation has been absorbed is sometimes very close. In these cases the muscles are still temporarily infiltrated, feel hard, and resist pitting. Induration without swelling exists as in sclerema. The previous swelling of the surface and the subsequent softening of the muscles suggest the true diagnosis.

TREATMENT.—Warmth must be provided by the use of an incubator. As long as possible breast feeding should be employed; but this may fail, and spoon or nasal feeding may become necessary. Massage or hot baths are sometimes beneficial. Roussel advocates the subcutaneous injection of 2½ drachms of normal saline three times a day, possibly in conjunction with saline per rectum.

Pseudo-Sclerema.—Under this title a condition is here described which bears some resemblance to both sclero-odema and sclerema. Apparently it occurs more commonly in the British Isles than either of the preceding conditions. The descriptions of sclerema which are to be found in many of the English textbooks do not correspond so closely to sclerema, as it is understood elsewhere, as to the disease about to be considered. Moreover, as pointed out by Lushley, some of the examples of this affection have been demonstrated as true sclerema, and included in the literature under that name. Such are cases described by Argil Moxey, Barrs, Garrod, Blacker, and Birch.

SYMPTOMS.—The condition begins in the first weeks of life, usually before the end of the first month. It does not affect peripheral parts first, but is noticed as circumscribed foci of induration, usually on the buttocks, thighs, or back, but sometimes on the cheeks. These areas do not pit on pressure, nor are they diffuse. They do not gradually merge into the neighbouring healthy tissue, as does sclerema. Often they are very sharply defined, and limited by an irregular bayed margin. The colour of the skin is frequently pale bluish-pink; sometimes it is pallid, sometimes livid.

These infants are not necessarily weakly or marasmic; indeed, there may be little or no constitutional disturbance. The temperature is not subnormal, and the pulse and respiration rates are not slowed.

The cases generally end in recovery. The areas of hardening gradually break up into smaller islets, each of which softens and ultimately disappears. The whole course of the disease is extended over from one to four months. It gradually develops, perhaps not reaching its acme for several weeks, and as gradually declines.

DIAGNOSIS.—It is distinguished from both sclero-odema and sclerema by the position of its onset, by its distribution, by the presence of definite circumscribed patches, by its chronicity, and especially by the absence of severe constitutional disturbance, and the favourable course. From sclero-odema it is also known by the fact that it does not pit on pressure; from sclerema, by the colour of the skin.

As Lushley has pointed out, it resembles more closely scleroderma as the latter occurs in early infancy, and it is open to question whether it should not be included under that title. However, it is not accompanied by a glistening shiny skin nor by alopecia.

TREATMENT.—Warmth, cod-liver-oil internally, and the application of emollients such as Rice ointment or cod-liver-oil to the skin, appear to be beneficial.

Scleroderma is an affection rarely met with in the first weeks of life. Usually it begins as an oedematous swelling, which leads to a coarse thickening and hardening of the skin and connective tissue, and ends in atrophy of those parts. It may be diffuse and affect the whole surface of the body. If this occurs, the patient is hunched, unable to be put in the sitting position, or in severe cases even to roll over. The face is mask-like, the eyes being always open and unable to shut. The mouth is also open, and for this reason breast feeding may have to be replaced by spoon feeding.

Occasionally sclerodactyly is present. The fingers are thin, slender, contracted, and covered by a shiny, parchment-like skin, which is bound down to them closely, and prevents movement except to a very slight degree. This condition usually accompanies a symmetrical scleroderma of the face and extremities rather than the general form.

If the disease occurs as a white, shiny, enamel-like patch, it is known as "morphoea." Sometimes these areas are surrounded by a blue-coloured band. The hair falls out in the affected skin.

In a case recently under the care of the writer, scleroderma and sclerodactyly were associated with hardening of the muscles.

There is no constitutional disturbance, but, naturally, development and growth are retarded. The course is slow, but apparently infants always recover.

PATHOLOGY.—The connective tissue is swollen, glossy, and appears to be swollen with a viscous fluid which has coagulated. There is fibrous tissue overgrowth, but diminution in elastic fibres. The vessels are either narrowed or occluded. Most authorities regard it as a trophoneurosis.

TREATMENT.—Hot packs and emollient applications may be used. Thyroid extract has been successful in some cases. Local injection of thiosinamin ($\frac{1}{4}$ to 1 c.c. with 10 per cent. glycerine) has been recommended recently. Tonic treatment by iron and arsenic is beneficial (see also Chapter XXIII., p. 1161).

CHAPTER II

DISEASES OF NUTRITION

EDMUND CAUTLEY.

1. **ATROPHY.**

2. **RICHTER'S.**

3. **SCARF.**

1. ATROPHY.

SYNONYMS.—*Inanition*; *Athrepsia*; *Malnutrition*; *Infantile atrophy*; *Marasmus*.

Wasting affections of children are acute or chronic. The term *acute inanition* or *acute atrophy* is used for the cases in which emaciation takes place with great rapidity. *Chronic atrophy* includes a large number of different conditions in which the main feature is a progressive loss of flesh or a prolonged period of lack of gain in weight. In its early stages it is spoken of as *malnutrition*, and in late stages and extreme forms it is called *marasmus*. The wasting may be due to disease, or the consequence of starvation, or of a diet which is sufficient in quantity, but unsuitable in quality. Hence it may occur at any age. In infants, generally in the first year of life, it is spoken of as *infantile atrophy*. Loss of flesh, or inability to gain weight, is the only definite clinical feature.

Acute Inanition or Acute Atrophy is almost always due to a deficient supply or an excessive loss of fluid. Thus, it may be the effect of unsuspected starvation in the case of a breast-fed infant when the maternal breasts are large, fatty, and secrete little milk. More often it results from acute vomiting, such as occurs in recurrent vomiting and acidosis, or from the various types of virulent diarrhoea. There is a remarkably rapid loss of weight in these cases. An infant becomes pallid and prostrated. Its extremities are cold, and the skin is dry and cadaveric, sometimes covered with clammy sweat. The fontanelle is sunken, and the edges of the cranial bones overlap. The pupils are small or pin-point. The mental state is one of irritability or fastidiness, and passes into semi-stupor. The temperature is subnormal, the pulse weak and frequent, and the breathing shallow and irregular. The appetite is lost. Vomiting may be absent, or may follow the ingestion of several meals, as the result of overdistension of the stomach from loss of its motor power. The stools are sometimes normal; more often they contain undigested food, and frequently mucus. If no food is taken or passed through the pylorus, the stools consist of epithelial debris, mucus, and bile, and look like meconium. Such stools are seen in severe infantile pyloric obstruction.

In serious cases the aethenia increases. It may be associated with slight cyanosis of the lips and diarrhoea, and prove fatal in a few days. In less severe cases life may be prolonged for three to six weeks, diarrhoea and vomiting being present and the emaciation progressive. The younger the child, the more rapid is the progress and the worse the prognosis. Cases due to starvation are generally

resumable when treated by suitable diet. It is important that the diet should be simple, bland and easily digestible at first, for the mucous membrane and glands are incapable of suddenly dealing with a liberal supply of food, and fatal diarrhoea may be induced. If the illness is due to diarrhoeal disease, the treatment is that appropriate thereto. Rectal, subcutaneous or intraperitoneal injections of normal saline are often needed. The child must be kept warm, carefully fed, and handled as little as possible.

Chronic Atrophy.—One or more of several factors is concerned in the production of loss of flesh, even if we eliminate the presence of actual disease. Possibly there is an inherited defect of tissue, a congenital disability to assimilate food. A family history of tuberculosis or syphilis is of great importance. Apart from the transmission of actual disease, there results a feeble vitality in the infant, due to the inheritance of imperfect tissues, which are incapable of carrying on efficiently the functions of digestion, absorption, and assimilation.

Imperfect development depends on antenatal conditions, diet, and environment. About 80 per cent. of all infants are born healthy. The debility of the remainder is consequent on morbid heredity, notably tuberculosis, syphilis, alcoholism, neurones, and general debility, and to a less extent on factors affecting the mother during pregnancy. Anything which impairs the health of the expectant mother is more or less injurious to the unborn child.

Prematurity is apt to come on inherited disease, and is a common cause of malnutrition. Mortality statistics show an apparent increase in the inherent predisposition to death in early life from prematurity, defective vitality, and congenital defects. In part this is because many stillborn infants formerly escaped registration, and now appear in statistics as prematurely born. But this is not the sole explanation, and it is a curious fact that the mortality from wasting diseases is much greater among male than female infants. A good deal of the increase in this mortality is an indirect effect of improvements in general hygiene, environment, food-supply, and medical science. As a result of these causes there is an existence of a steadily increasing number of physically and mentally feeble creatures, who attain an age at which they are enabled to transmit their defective vitality. Fortunately, the influence of morbid heredity tends to die out by the extinction of the family or through marriage with healthy subjects; and, in addition, there is a constant tendency to a mean in physical and mental development.

Congenital defects may show themselves in the form of malformations of the digestive tract or other structures, which lead to impaired nutrition. Among these may be mentioned hare-lip and cleft palate; stenosis and atresia of the œsophagus, pylorus, small and large intestine, and anus; congenital heart disease; and defects of the urogenital system.

After birth the wasting may depend on inability to suck, in consequence of structural defects of the lips or palate, tonsillar hypertrophy, the nasal obstruction of rhinitis or adenoids, inflammatory affections of the nose or throat, or palatal palsy; or it may be due to small or retracted maternal nipples or an unsuitable teat. More frequently the refusal to suck depends on anorexia or displeasing food. In rare instances there is no obvious explanation, and one is driven to assume an imperfection in co-ordination or in development of the nerve centres for suction and deglutition. Some of these infants may suddenly begin to suckle normally. In a remarkable case reported by Piquet, the reflex area for sucking receded gradually from the tip to the base of the tongue, death resulting from hydro-

cephalus. In a patient in whom I could find no evident cause for the refusal to suck, death was due to tuberculosis. In other instances it depends on temper or on mental defect.

At all ages *varicositatem* has a potent influence on development, and consequently on malnutrition. Overcrowding, defective hygiene, insufficient clothing, exposure to inclement weather, lack of sleep, overfatigue, and general neglect, are deleterious. Education is not an unmixed blessing. Children, who in former days added to the family exchequer, are kept at school. Many of them suffer from mental strain, and in winter especially, from insufficient fresh air, light, and exercise, as well as defective diet. The parents are perhaps unable to provide enough food, and the craze for cheapness has led to the adulteration of food and to the use of tinned, savoury, and unwholesome substitutes for the simpler foods suitable to the growing child. Many a child is brought up on condensed milk, proprietary foods, adulterated bread, kippers, sausages, and pickles—tasty articles of diet which deprave the appetite and are apt to set up gastric and enteric catarrh, malnutrition, and consequent liability to tuberculosis. Tea, alcohol, and tobacco, are also injurious agents in early life; so, too, are many patent medicines and drugs, such as opiates and white bryony. The general result is the existence of numbers of undersized, weakly adults, with digestions already bad, and likely to be still further impaired by tasty and stimulating foods, strong tea, and alcohol. Morally, physically, and mentally, they are ill-developed, and their children are likely to be congenitally feeble and liable to atrophy.

Apart from environment and inherited or congenital defects, development is mainly a matter of suitable diet, and the chief cause of infantile atrophy is the diet. Other factors being equal, breast-fed infants are stronger, healthier, and less prone to disease, than those artificially fed. Hebrew children are taller, heavier, less rachitic, and have sounder teeth, than those of Gentile parentage. The Jewish mother is better fed and cared for during pregnancy, and her child is almost always breast-fed and gets a suitable diet in early life. Malnutrition is rarely due to overfeeding. More often the food is insufficient in quantity or unsuitable in quality. It may be contaminated by bacterial infection, or the state of the alimentary tract may render a suitable diet unsatisfactory for the particular child.

In older children chronic atrophy is a sequence of many of the causes already enumerated. Other conditions and diseases must be excluded—*e.g.*, latent erysipela, tuberculous infection, blood-disorders, diabetes, and chronic interstitial nephritis. A common cause, apt to be overlooked or mistaken for abdominal tuberculosis, is the intestinal dyspepsia often present in early life, and characterized by a large fatulent belly, languor, debility, dark rings under the eyes, attacks of pallor, and wasting.

SYMPTOMS.—To a certain extent the symptoms of chronic atrophy vary according to the age of the child, the cause of the wasting, its rapidity and its duration. The most typical cases are seen in the first year of life. After this age malnutrition is common, but acute inanition and *marasmus* are infrequent, except from acute illness.

In infancy the usual history is that the baby has always been delicate, or that he was apparently healthy at birth and while breast-fed, and began to waste when weaned; that the wasting has continued steadily, in spite of the various methods of feeding tried by the mother, recommended by neighbours, friends, and relatives, or suggested by specious advertisements; and that in defiance of all treatment the

baby has got thinner and thinner, and has had frequent attacks of vomiting and diarrhoea.

In the stage of emaciation the infant is below the weight appropriate to its age and initial weight at birth, or gains weight very slowly for some considerable time. Constructive metabolism is little or not at all greater than destructive metabolism. The deficiency in weight is usually associated with deficient growth in height and delay in the acquirements of the functions of sitting, standing, and walking, as well as slow mental development. Dentition may be delayed, but is often accomplished without trouble or discomfort. The child is more or less anæmic, sharp featured, and wasted, with dark rings under the eyes, deficient elasticity of the skin, cold extremities, and a temperature which readily sinks below normal. He is fretful, irritable, and sleeps badly. The appetite is poor and capricious, indigestion is easily set up, and attacks of diarrhoea are frequent. Sometimes there is catarrhal enteritis or enterocolitis. The fontanelle is depressed. The muscles are flabby, the ligaments weak, and the joints lax and insufficiently supported.

Such is the description of a case of malnutrition, or *chronic atrophy*. There is no dividing line, but I think the term "atrophy" might be strictly limited to those cases in which there is actual wasting, not merely a lack of gain in weight. If there is no record of the previous weights, then the diagnosis must be based on the relative degree of insufficient weight for the age of the child, and the degree of emaciation as shown by the wrinkling and loss of elasticity of the skin and absence of subcutaneous fat.

In the further stage of *marasmus* the condition is much more serious. The name simply means "wasting," and it must be definitely stated that *marasmus* is not a disease. It is merely a late stage in the course of malnutrition and chronic atrophy, arbitrarily defined, and with no line of demarcation. It is generally seen in the first year of life; is not infrequent in the second year; and is occasionally present at all ages as the sequel of prolonged wasting diseases, such as persistent vomiting, diarrhoeal affections, tuberculosis, chronic interstitial nephritis, cirrhosis of the liver, and diabetes.

In a typical *marasmic* infant the facial aspect is that of old age. The skin is wrinkled and thrown into folds on the forehead and between the eyes. It has lost its elasticity, and is devoid of subcutaneous fat. All over the body, especially the arms and thighs, it is dirty in colour, dry, harsh, lax, easily raised from the subjacent tissues, and hangs in folds. The face is pallid, earthy or ashen in colour. The bones stand out sharply, giving the features a pinched look; the temples and eyes are sunken, and the pupils small. The chest and extremities seem absolutely skin and bone, the abdomen is protuberant and tympanitic, and the terminal phalanges are bluish. The tongue is red and dry, and both it and the mouth are often covered with aphthous patches.

The appetite may be either completely lost or increased. The state of the stomach and bowels is variable, much the same as in malnutrition and atrophy. Even water may be vomited. Sometimes the stools appear well digested, but they are then unduly large for the amount of food taken. In other words, only an inconsiderable quantity is absorbed. The circulation is feeble and the blood deficient in hæmoglobin. The temperature is low, and may fall below 95° F. The mental state is one of fretfulness and irritability, or of listlessness and apathy.

As the child gets worse he is liable to develop *rodema* of the dorsum of the feet

and hands, and later on of the eyelids and rest of the body. There is no effusion into serous cavities. Diarrhœa, if present, may become less or entirely cease. Petechial hæmorrhages are apt to appear on the lower part of the abdomen, the whole abdomen, the trunk, the upper part of the thighs, and elsewhere; and the temperature falls to a lower and lower level.

Complications are generally absent in simple malnutrition and in acute inanition. In chronic atrophy and marasmus they may develop at any stage of the wasting process, helping to maintain or increase the emaciation. Aphthous stomatitis is quite common. Herpetic stomatitis is less frequent, but occasionally occurs in successive attacks. Enteritis or enterocolitis is common, and may be temporary, mild, or severe. Redness of the skin appears over the scapula, heels, occiput, and other bony prominences, and may terminate in bedsores unless attended to carefully. Erythema and superficial ulceration of the buttocks result from irritant discharges. Pustular, boil-like spots appear on the scalp, and similar boils or subcutaneous abscesses on the trunk and limbs. The lymph-nodes are apt to enlarge, and reach the size of large peas or small nuts, in the neck, groins, and axilla. Reflex opisthotonos is not uncommon, and the babe may lie continuously with its head thrown back, spine bent, and heels drawn up. Vomiting, enteritis, and bronchitis, are often serious and fatal.

PATHOLOGY.—The appearances in the gut and liver are not such as indicate the causation of the disease. The organs are generally pale. Sometimes there is congestion or œdema of the meninges and brain, hypostatic congestion of the lungs, pulmonary collapse, pleural ecchymoses, and, exceptionally, thrombosis of cerebral sinuses. The gastric and intestinal mucosa is pale, perhaps swollen, and may exhibit patches of congestion, wide areas of erosion, subepithelial hæmorrhages, necrosis of deeper tissues, and secondary fibrin formation. These changes are most marked in the cecal region, because of stasis of food. The kidneys are almost always pale. The cortex is swollen, the glomeruli injected, and the pyramids hyperemic. Microscopical examination shows cloudy swelling and fatty degeneration of the epithelium, especially in the convoluted tubes. In prolonged cases there is fatty and parenchymatous degeneration of the liver. The lymph-nodes, follicles, and Peyer's patches, may be enlarged.

The anatomical changes are secondary to the wasting, and not the cause thereof. Atrophy of the intestinal mucosa is part of the general wasting, and the microscopical appearances are mainly due to gaseous distension and post-mortem changes. The disorder is one of functional impairment of digestion, absorption, and assimilation. In some instances in which a nutritious and sufficient diet is apparently assimilated, and yet the child wastes, the function of assimilation is in abeyance, defective, or lost. With increasing feebleness the circulation becomes weaker, the expansion of the lungs imperfect, and oxidation incomplete. To the malnutrition is added the effect of imperfect metabolism.

On insufficient evidence, the affection has been ascribed to acid intoxication; to auto-intoxication from decomposing food; to deficient activity of nutritive ferments, due to intestinal infection; to a defect in Paneth's cells in Lieberkühn's crypts; and to subacute or chronic infection of the intestinal tract. Recently Langstein and Meyer have found that there is a great loss of mineral constituents in the course of the disease.

It seems unnecessary to invoke any of these hypotheses as the ætiological factor. The simple and reasonable explanation that the emaciation is due to an insuffi-

cient supply of nutritive food in a form suitable for the child is to my mind quite sufficient. An unsatisfactory diet causes malnutrition. As the nutrition of the cells becomes impaired, it is natural that their functional activity decreases. Hence the glands secrete less ferments and less digestive juices, and the cells throughout the body lose their assimilative functions. All other effects are purely secondary.

DIAGNOSIS.—Acute inanition due to insufficient food is easily recognized if the cause is suspected. It may be overlooked in a breast-fed infant. A subnormal temperature generally enables an acute disease to be excluded. I am extremely doubtful of the existence of *inartificial fever*, supposed to occur from insufficient food in the first few days of life. Such cases are probably due to mild sepsis. The rapid wasting of acute inanition distinguishes it from that of marasmus, except when it occurs at the termination of chronic wasting.

The differentiation between malnutrition, chronic atrophy, and marasmus, in the first year of life is merely one of degree. Before diagnosing any of these conditions, it is necessary to exclude wasting due to organic or constitutional disease, such as pyloric stenosis and other malformations of the alimentary tract, gastric and intestinal catarrh, pulmonary diseases, empyema, *necrosis coecæ*, rickets, congenital syphilis, and tuberculosis. Time after time one sees infants in whom tuberculosis is suspected, and no evidence thereof found after death. Less often the autopsy on a "waster" reveals unsuspected and extensive tuberculosis, although during life fever was absent.

Diagnosis depends essentially on regular weighing. Apart from this it is impossible to estimate with any degree of accuracy the amount and rapidity of the emaciation. The cause can be decided by examination of the diet, whether it be human milk or artificial food; examination of the contents of the stomach for mucus, hydrochloric acid, and organic acids; estimation of the motility of the stomach; examination of the feces for undigested food, mucus, worms and their ova, etc.; and of the urine for albumin and indican. In doubtful congenital syphilis, Wassermann's reaction may be of assistance.

In older children investigate the family and past history, the nature of the diet and mode of life. Make a careful examination for pulmonary disease and glandular enlargement in the chest, neck, and abdomen. The suspicion of tuberculosis is very difficult to get rid of. At the time of the second dentition, and often earlier, gastric and intestinal dyspepsia often cause marked wasting, sometimes associated with cough suggestive of pulmonary tuberculosis and abdominal distension, such as occurs in tuberculous peritonitis, or with pharyngitis, enlarged tonsils, and adenoids. At all ages lack of development may depend on naso-pharyngeal obstruction or deficient thyroid secretion. As a child grows in height, he commonly loses in weight, but it must never be forgotten that malnutrition in a growing child may be due to other causes.

PROGNOSIS.—The outlook depends on the degree of wasting, the causation thereof and its duration, and the amount of care devoted to the patient. Acute inanition due to starvation is easily curable; that of chronic diarrhoea depends on the cure of the primary disease. The majority of babies with simple malnutrition, atrophy, or marasmus, dependent on diet and neglect, get well under efficient treatment. Keep them alive, and eventually they develop into normal children.

No cases exercise the skill and patience of the physician and attendants to a

greater extent than some of these wasters. The longer the duration of the wasting, the greater is the care required and the more tedious is the course. The utmost care is needed, and much depends on the faithful attention to instructions, which should invariably be written down fully and clearly. A pig-headed or careless nurse may neutralize the most skilled treatment and quickly destroy the child's chance of recovery. The parents must trust the physician responsible for the treatment, and must realize that progress will be very slow. Experiments in diet are dangerous, and often fatal, especially in hot weather, and bad results are frequently due to changes in diet ordered by competent physicians successively called in consultation. Any physician with experience in the management of these cases will get satisfactory results if the patient is entirely entrusted to his charge and his directions are carried out. A multiplicity of counsellors too often exemplifies the old adage that "too many cooks spoil the broth."

Improvement is indicated by gain in weight, provided there is no oedema, by better appetite and digestion, less fretfulness, and a temperature nearer the normal level.

A downward course is usually gradual. Perhaps towards the end there is a very rapid kind of collapse, analogous to acute inanition. Death is due to asthemia, sometimes in convulsions. It may be preceded by a period of unconsciousness of more or less sudden onset; or it may take place quite suddenly, although there has been no obvious change in the state of the child; or it may be due to cardiac syncope, perhaps the result of flaccid distension of the stomach. Sudden collapse may also be induced by pulmonary atelectasis, broncho-pneumonia, vomiting, diarrhoea, or convulsions.

In every case under treatment the prognosis improves with the age of the child, and varies directly with the environment and the care devoted to it. It is worst in the cases of inherited constitutional debility, and is increased in gravity by hot weather, partly through the debilitating effect of great heat, and partly because of the greater liability to food infection. The mortality is high in hospitals, for only the worst and most prolonged cases are admitted, and in them the power of assimilation sometimes appears completely lost. The smaller the weight in relation to the birth-weight and the present age of the child, the more serious is the prognosis. Nevertheless, it cannot be too strongly impressed on everyone connected with the treatment of the child that no case is hopeless if the atrophy is of dietetic and not of constitutional origin, and that no pains should be spared to restore normal health and nutrition, for these babies ultimately become quite strong and healthy. Strict attention to minutiae is essential to success.

TREATMENT.—Prophylaxis includes all the measures conducive to normal growth and development. For simple infantile malnutrition give a diet appropriate for a child younger in age, and allow shorter intervals between the feeds. A wet-nurse is the best remedy for infants under four months old. If not available, and in other cases, the food must be weak in quality to start with, and must be varied in accordance with the progress. Changes must be made very gradually. A slight excess of food may bring on acute dyspepsia or diarrhoea, undoing in twenty-four hours the good effects of weeks of careful treatment, and may even prove fatal. Marasmic infants digest fat badly, and may develop diarrhoea if there is too much fat in the food. Breast feeding may be continued, partially or entirely, up to eighteen months of age, if the child is small and like a much younger infant. Apart from breast feeding, reliance must be placed on whey, sweet whey powder (a level

teaspoonful to 2 ounces of water), peptonized milk, modified milk, condensed milk, various malted and partly malted foods, and Benger's food. Albumin-water and albumactin can be added if more protein is desired. Cow's milk, if used, must be well diluted at first and given cautiously. The digestive capacity, not the age or weight, is the guide to the appropriate diet.

In mild cases begin with food suitable for a younger infant; in the more severe give the different foods in the order stated above, or make use of modifications and mixtures of the various foods suggested. Allow water freely in small quantities at a time. Examine the stools daily, since the necessity for dietetic alterations depends chiefly on their characters.

Suitable additional foods are the yolk of egg, bone marrow, raw-meat juice, and cod-liver-oil, provided the child has reached an age at which he can digest fat. A good mixture consists of the yolk of one new-laid egg; glycerine, oz. i.; ol. morrhue rel. ol. olive, oz. i.; and creosote, min. v.-x.; in doses up to dr. i. four times daily. The fructole medullæ et glycerophosph. (S. & M.) is a suitable preparation.

General measures, suitable at all ages, but modified according to age and environment, include a liberal supply of fresh air; general friction or massage with olive-oil or cacao-butter; sponging with cold water, about 65° to 75° F., after the morning bath; regular bathing, sleep, and meals. A marasmic infant must be rubbed with oil, kept warm, wrapped in cotton-wool, and surrounded with hot bottles or kept in an incubator. In large institutions ventilation must be free, and each infant allowed 800 cubic feet air space. Alcohol, gavage, and saline injections, per rectum or sub cutis, are sometimes needed.

Malnutrition in older children is treated by simple nutritious diet, regular meals, regular habits, plenty of rest and sleep, cold sponging, open-air life, limited studies, and no excitement. Feced feeding is inadvisable. Set the alimentary tract in a satisfactory state, and then give iron, arsenic, cod-liver-oil, or bone marrow and glycerophosphates. To improve the appetite, give a mixture of the tinctures of *sax. virgica*, *calumba*, aniseed, and orange. In small doses it is very beneficial for infants.

2. RICKETS.

RICKETS was mentioned in Grant's "Bills of Mortality" in 1604 as a cause of death, but it was not fully described until Francis Glisson published in 1650 a monograph entitled "*De Rachitide sive Morbo Puerili qui vulgo The Rickets dicitur.*" According to Glisson, it first showed itself in the West of England, in Dorset and Somerset, about thirty years previously. Probably it has been in existence from an unknown period, although not mentioned by the ancient writers Hippocrates, Galen, and Celsus. In point of time its recognition occurred about a century before the artificial feeding of infants was mentioned in medical books. Its incidence corresponded roughly with the decline of maternal nursing; and the gradual spread of other methods of rearing infants produced numbers of cases, and thus attracted attention. Its increasing prevalence is due to the increasing disinclination and inability of women to nurse their offspring, and to the aggregation of the population in industrial centres. The decrease in its severity is due to improved hygiene, superior methods of artificial feeding, the better education of mothers, and the more general provision of adequate medical treatment.

Gilson gave the disease the name of *rachitis* (*rákhis*, spine), on account of the deformity of the spine commonly present, and because of its similarity to the popular name *rickets*, either a corruption of the Norman-French word *ripette*, for deformities, or of the Anglo-Saxon *weorðen*, to twist.

DEFINITION.—Rickets is a chronic general disorder of nutrition, occurring during infancy from faulty diet and hygienic deficiencies. It involves the whole body, but its most obvious effects are recorded in the skeletal system, producing deformities which may affect the patient throughout life, and, if a female, prove a source of disaster during confinement. The osteogenetic tissues undergo abnormal proliferation, and lose the power of making normal bone. The general metabolic disturbance involves the mucosa, and is a grave source of alimentary and respiratory affections; weakens the muscles and ligaments, and assists in the production of various disabilities and deformities; and affects the nervous system, giving rise to various nervous complications. The disease is in itself not fatal, but is a potent factor in determining the outcome of an illness such as broncho-pneumonia, which in another than a pigeon-breasted child might not be even serious.

VARIETIES.—Cases vary in character and severity. The most acute exhibit profuse sweating, visceral disturbance, and perhaps bony tenderness. Bony tenderness is due to associated scurvy, and there is no such disease as acute rickets. Generally the bone symptoms and resulting deformities predominate, but I think that severe instances of this type are becoming less common. In a third variety the catarrhal symptoms are chiefly in evidence. *Acrostatic rickets* is a rare type, in which there is a remarkable laxity of the muscles and ligaments. In the most common variety there are moderate skeletal changes, associated with a tendency to alimentary and respiratory catarrh and mild general symptoms.

EMBIOLOGY.—Although called the *English disease*, it exists everywhere. It is common in the temperate zone; infrequent in Iceland, Greenland, Norway, and Denmark; rare in the tropics, India, China, Japan, and the southern parts of Italy and Spain; and almost unknown near the equator. The increasing prevalence of the disease, in countries in which it was formerly rare, is associated with the decrease in maternal nursing and the introduction of proprietary foods.

It is rare at high altitudes. It is more frequent in urban than in country districts, though by no means uncommon in the latter. Favourable climatic surroundings may be insufficient to counteract the ill effects of bad feeding and lack of ventilation in cottage homes. If slight bending or *craniotabes* is regarded as proof of the disease, it will be found in half the infantile population of large towns. The worst cases are seen in the spring, as a sequel of the climatic conditions of an English winter and an indoor life. Yet they do not occur among the Eskimos, although the winter is prolonged and the child kept still more indoors. The lack of fresh air is counterbalanced by prolonged breast feeding and a liberal supply of fat.

Racial peculiarities may be counteracted by the habits of civilization. Negroes are rarely, and Italians infrequently, affected in their own countries, but in New York they are specially liable to the disease. No class is exempt. The disease is almost as prevalent among the rich as among the poor, though it is rarely as severe, for it receives earlier treatment.

Heredity may be disregarded. Possibly there is a slight proclivity to the disease in some families. More probably the incidence of the disease in several

children depends on the methods of feeding and management. Ill-health in the mother is a cause of deficiency in the infant, and deficiency from any cause is a factor predisposing to rickets. A deficiency of protein and fat in the mother's diet during pregnancy and lactation causes infantile debility and an insufficient milk-supply. Frequent pregnancies are injurious, but their bad effects are partly counter-balanced by the mother's improved knowledge of the proper methods of rearing infants.

Age and Sex.—Both sexes are equally liable. The usual age of onset is six to twelve months. The disease may begin earlier, but it is not often obvious before six months of age, a fact suggestive of a post-natal cause. By the eighteenth month it is distinctly marked. It rarely begins at a later date, though it may occur at any time during the first dentition, and perchance even in later life.

Intra-uterine Rickets, sometimes called *fetal* or *congenital*, is open to doubt, though such cases have been described. The fetal form is supposed to develop and recover during intra-uterine life. Marfan (1906) recognizes (1) a latent form, only diagnosable by the microscope; (2) an extreme type in still-born children, with many deformities; (3) a rare type in which the deformities are characteristic, and disappear in two years; and (4) a common variety in which there is craniotabes at birth, and deformities develop later, with generally a past history of syphilis or other serious morbidity in the mother. Charrin and Le Play (1905) regarded as rachitic the craniotabes, rosary and enlarged epiphyses of a child born at eight months of a mother, aged forty-seven, who had suffered from privation. They based their opinion on macroscopic and microscopic examination and on chemical analysis.

It must be pointed out that an open fontanelle, soft bones, costo-chondral swellings, and a protuberant belly, are common to all new-born infants, and are insufficient proof of rickets. Multiple fractures *in utero*, craniotabes, and slight bending of the ribs, are not rachitic, and may justly be regarded as of the same nature as experimental osteoporosis. The epiphyses are not enlarged. Some of the recorded cases appear to be of this nature. In all, or almost all, instances the histological changes in the bones and cartilages are not characteristic of rickets.

The existence of intra-uterine rickets should be regarded at present as "not proven." It must not be confounded with syphilitic osteochondritis or achondroplasia. Craniotabes is present in developmental cerebral defects and in anosteoplasia (skido-cranial dysostosis). Fractures *in utero* are generally due to perimetrical aplasia (osteogenesis imperfecta).

Diet.—In prolonged lactation, or if the mother's milk is deficient in quality, usually in fat, the breast-fed child may develop the disease. In artificially-reared infants it results from a diet poor in fat; more readily if there is also a deficiency in protein, and still more so if there is an excess of carbohydrates. It is unusual to find an infant brought up on condensed milk, alone or in combination with a proprietary food, who is not definitely rachitic. Such a diet contains an excess of carbohydrates, and is deficient in fat and protein. Sometimes the food-supply contains the requisite percentages of the proximate principles of diet, but in a form unsuitable to the assimilative powers of the child. The importance of diet must not be overrated. Some infants thrive on most unusual foods. An infant eight months old looked the picture of health, and showed no signs of rickets, although he was said to have been brought up entirely on oatmeal gruel made with water. And the breast-fed infant may develop the disease, very rarely in a severe form, while the bottle-fed child remains healthy.

The effect of diet is shown by experiments on animals. Puppies, fed by Guérin for four or five months on a meat diet, became rachitic, while others of the same litter, suckled, showed no sign of the disease. Blaud-Sutton found that young lions at the Zoological Gardens became very rachitic if weaned early and fed on raw meat only. The addition of milk and cod-liver-oil to the diet cured them in three months. Two young monkeys fed on vegetarian diet became rachitic. Other feeding experiments on young animals have induced profound marasmus and death, without causing rickets. Apparently nutrition is so impaired that the proliferation of the osteogenetic tissues, characteristic of rickets, cannot take place. Generally, a deficiency of protein causes weakness and impaired nutrition, and excess of carbohydrates sets up gastro-intestinal disturbance and toxæmia. The effects of diet are further considered in discussing the pathology of the disease.

Exercise.—Emmellay (1908) has shown, by experiments that confinement and lack of exercise have an important bearing on the production of rickets; so much so that he ascribed the disease to lack of exercise. Oxidation is diminished, and carbonic acid excretion is imperfect. Animals and birds kept in cages developed rickets, although they got sun and fresh air. He could induce the disease in puppies by confinement alone, but not by means of any variety of diet if free exercise were permitted. The prevalence of rickets in towns and in the winter months, and its rarity in hot countries, can be advanced as evidence in favour of the effects of lack of exercise. In Japan the disease does not occur in spite of prolonged lactation. Maternal nursing and an open-air life in a mild climate are most valuable prophylactic agents.

Associated Diseases.—Syphilis and tuberculosis have no etiological significance, except in so far as they are a source of general debility, interfere with digestion and assimilation, and reduce the resisting power of the infant. Catarrhal affections of the respiratory and alimentary tracts, and thoracic deformity, render rachitic infants more liable to tuberculous infection. Associated digestive disorders are partly a sequence, partly a cause, of rickets. They indicate that the diet is unsuitable. If alimentary disturbance is severe or prolonged, the secondary malnutrition or marasmus masks the rachitic process. Marked signs of the disease are lacking in the marasmic, for assimilation and growth must take place in order to produce the characteristic physical signs. Wasting disorders are antagonistic to rickets, because the child has not sufficient anabolic vitality for the production of the characteristic proliferative changes in the cartilages and bones.

Symptoms.—The chief features are the softening and deformity of the skeletal system. The early symptoms are constitutional—viz., fretfulness, restlessness, and disturbed sleep, throwing off the bedclothes, disinclination to move about, head-sweating, constipation, irritability of the nervous system, and liability to respiratory and alimentary catarrh. Bowing of the ribs, craniotabes, epiphyseal enlargement, and deformities, develop gradually.

The complexion is almost always pale in severe cases, but the blood shows no characteristic changes. Hemoglobin may be reduced to 60 per cent. The anemia is due to the same cause as the rickets or to adventitious factors. It is not directly due to rickets, for ordinarily the number of red cells and the percentage of hæmoglobin are increased. Nucleated red cells, polychromatophilia, and myelocytes, are rare. The white cells show no definite change.

Some children are rosy and well-nourished; others are pale, fat, and flabby, with little resisting power, and liable to acute affections of the mucous membranes

This is the type known as *fat rickets* as opposed to *thin rickets*, wasted cases with considerable malnutrition. *Ptyosis* is only present if there is some complication.

Digestive System.—The appetite may be unimpaired throughout. It is generally good, sometimes excessive, and there is marked appreciation of salt. Vomiting is uncommon. Constipation is frequent in early and in severe cases. It is due to lack of muscular tone in the intestinal and abdominal walls, and sometimes to chronic intestinal catarrh. It may alternate with diarrhoea, if the hard, dry stools set up a chronic colitis and the excretion of large quantities of mucus. The stools are often deficient in pigment, pale and patty-like, if the diet has not been attended to; occasionally green, slimy, and offensive. The alimentary catarrh increases the liability to diarrhoea, which is often acute.

The urine presents no marked features. It is faintly acid, and perhaps deficient in calcium. It has been said to contain a great excess of lactic acid (Heitzmann) and of phosphates. And from this it has been argued that exogenous phosphates are excreted instead of being built up into bone. In numerous cases there is no excess of these constituents, and other causes must be sought for to account for their presence.

Nervous System.—There are no nervous lesions, though many nervous manifestations. Defective nutrition of the nervous tissues accounts for several symptoms. Sweating is profuse during sleep. The sweat is acid, stands out conspicuously on the forehead, and may be so profuse as to soak the pillow. Increased general sweating and exanthema are occasionally present. Similar sweating may occur in debility from other causes. Restlessness during sleep is a common symptom. The infant may wake up and cry every quarter of an hour. During sleep he is apt to roll his head from side to side, rub the hair off the occiput, burrow in the pillows, toss about and throw off the clothes. Such symptoms are not peculiar to rickets. The susceptibility to reflex irritation is exaggerated. There is increased liability to muscular spasm in the form of laryngospasm, tetany, and general convulsions. Facial irritability may be the only sign, and must be regarded as a danger-signal. These convulsive affections are most common at three to nine months of age, and are often secondary to stomacic or intestinal disturbance, either through reflex irritation or the action of toxins. The degree of rickets is no measure of the liability to convulsions or other nervous phenomena. Possibly both are connected with disordered calcium and magnesium metabolism, and with alterations in the functions of the parathyroids and hemo-lymphatic organs.

PHYSICAL SIGNS.—The appearance of the skull is suggestive of hydrocephalus, but it is square rather than globular. The *head* looks too large, actually or in proportion to the trunk. It is irregularly square, with flattened crown and broad, high forehead. Occasionally it is dolichocephalic, with raised and prematurely ossified sutures, a type more common in the degenerate than the rachitic infant. In mild cases the edges of the cranial bones show periosteal thickening. In more severe ones cranial bosses or hyperostoses are formed in the frontal and parietal regions. At times they are so large and prominent as to stand up above the level of the median and transverse grooves, due to the sagittal and parietal sutures, forming the *hot cross-bun* type of skull, or *like carri*. They are composed of soft, bluish-red, vascular masses of spongy bone, as much as $\frac{1}{2}$ inch thick, about the centres of ossification. After death they can be indented by the finger or cut with a knife, blood and serum being easily squeezed out. Seen through the thin scalp, they have a pale bluish tinge. While forming they are a possible source of

local discomfort and irritability, and one sees cases of head banging, rolling, boring, etc. In time they are absorbed, or are organized into light, porous bone, or form diffuse laminae of dense ivory-like bone. The upper part of the occipital bone is sometimes so flattened by pressure as to appear nearly vertical on side-view. Marked asymmetry of the cranium is caused by flattening of the parieto-occipital region on one side, and prominence of the frontal eminence on the other.

The anterior fontanelle is large, and may remain open until the end of the first dentition, instead of being closed by the eighteenth month. Even at two years it may measure 2 inches across, and up to five years may be represented by a depression closed by cartilage. The other fontanelles and sutures remain open unduly long, and ossification of adjacent bone is delayed. The lambdoid suture may not close until the fifteenth instead of at the end of the third month; the



FIG. 4.—ASYMMETRY OF THE SKULL IN RICKETS.



FIG. 5.—ASYMMETRY OF THE SKULL IN RICKETS.

coronal may remain open until the end of the second year instead of closing at the end of the fourth month; and the sagittal, normally closed at the end of the first year, may remain open until the third year.

Craniotabes.—Ehrlicher in 1843 first described *craniotabes*, softened areas in the membrane bones of the skull, as "rachitis of the skull." Of this there are two varieties: (1) The free margins of the flat bones, especially the parietal and occipital bones, are soft and late in ossifying, and remain membranous for a long time. This membranous condition is most frequent along the margins of the lambdoid suture. In severe cases it is found along the margins of each cranial suture and round each fontanelle. In consequence of surrounding ossification, isolated soft spots may be formed. The membranous part of the occipital bone may be quite unossified. This variety of *craniotabes* is one of marked flexibility rather than true softening. It is found in infants from the third to the twelfth month of life, rarely earlier and never later. In younger infants it is generally dependent on serious congenital cerebral defect; the cranium may be membranous even at birth. In older ones it may be due to hydrocephalus or cerebral tumour. It is most common in infants brought up on condensed milk and in those seen in the earlier months

of the year. The delayed ossification, or possibly partial reabsorption of lime salts, from newly-formed bone, is either due to deficiency of lime salts in the food or their presence in an unsuitable form. Lack of fresh air and exercise are predisposing factors by interfering with metabolism. This type of rickets may exist without other evidence of rickets, or merely with slight beading of the ribs. It is not a positive proof of rickets, but it shows that the diet is unsuitable and defective in lime salts, the sort of diet which might be expected to give rise to rickets. In support of a rachitic aetiology we may mention the value of anti-rachitic treatment, and the association of this type of softening with laryngospasm. (2) The second variety is uncommon. It occurs in infants under six months of age, and consists of soft areas, round or oval, $\frac{1}{2}$ to 1 inch in diameter, chiefly situated in the posterior half of the parietal and the membranous part of the occipital bones. On pressure the spots yield with a parchment-like crackling. It is probably of syphilitic origin, though it may be associated with rickets, and is due to absorption of previously-formed bone.

The hair is thin and worn off the scalp, sometimes off the sides, by friction and sweating. The superficial veins of the scalp are prominent and easily visible through the thin skin. They may form deep grooves in the bones, distinguishable with difficulty from irregular sutures.

The face looks small by contrast with the large head and prominent forehead. The antero-posterior diameter of the upper jaw is lengthened, giving it a beak-like aspect; that of the lower jaw is shortened through flattening of the anterior portion, the incisor teeth being in a straight line, and the outline of the jaw forming a sharp angle at the site of the canine teeth. These malformations, together with imperfect alveolar development, prevent proper adjustment of the crowns of the teeth, and the incisors may turn inward.

Irregularities in the teeth and dentition vary with the time at which the disease originates. The lower central incisors are often cut at the usual date, while the later teeth are delayed, cut irregularly or "cut on the cross," or follow each other in rapid succession. By itself delayed dentition is not proof of rickets. Many children a year old are edentulous, but not rachitic. The teeth seem prone to discoloration and decay, and often show horizontal ridges and depressions in the enamel—changes which may occur independently of rickets. The milk-teeth should escape, if these changes are due to rickets and if rickets is of post-natal origin, for calcification is advanced at birth; yet they frequently decay very early. The permanent teeth calcify during the first year of life, and are more likely to be affected. Dentition is more apt to cause illness than in the normal child, because of the increased nervous instability and the liability to catarrh of the mucous membranes.

The Chest.—The first and most constant sign is enlargement of the cartilages at their junction with the ribs, producing the beads, or nodules, which form the rachitic rosary. These nodules are first felt, and are subsequently most obvious, at the fifth and sixth costo-chondral articulations. They vary in size, and are not always visible to the eye, though easily palpable. They are rarely felt before the third month, occasionally at one month, and are even more obvious on the pleural surface. Slight projections in the new-born and on the pleural surface of still-born infants must not be regarded as rachitic. In untreated cases the nodules enlarge up to the end of the second year of life, and then slowly disappear. Except in recrudescence or late rickets there is little trace of them after the fifth year, and

bone at all in adults. Posterior heads are due to greenstick fracture of the ribs at or near the angles. They are angular rather than nodular projections on the external surface, single or unilateral, variable in position, and asymmetrical. *Sparrows* bending may be seen on the pleural surface as the result of partial dislocation of the ribs backward at the costo-chondral junction.

The chest appears small in its upper part by contrast with the large head and protuberant belly. Its shape is altered by the action of atmospheric pressure on the yielding chest wall at the site of least resistance—that is, just external to the heads. Hence arises a lateral flattening or broad shallow concavity on each side, extending from the second or third rib to the hypochondrium. This tends to press forward the sternum and produce slight pigeon-breast. Pressure of the arms and the dorsal decubitus also modify the shape of the yielding boxes. In its typical form pigeon-breast is not present in uncomplicated rickets, and is seldom seen before six months of age. It is due to respiratory obstruction. It is an exaggeration of the ordinary rachitic thorax, which is fiddle-shaped in transverse section, through increase in the antero-posterior diameter and decrease in the transverse diameter by the sinking in of the lateral furrows and convexity of the sternum and cartilages. Sometimes the sternum is depressed between the projecting cartilages. A further effect of atmospheric pressure on the weakened thorax is the *Harrison's sulcus* or rachitic girdle, a transverse groove at the level of the upper limits of the liver, stomach, and spleen. These organs prevent the lower part of the chest wall falling in as much as the upper. The liver being large and resistant, the groove is much more marked on the right than on the left side. It is exaggerated by inspiration. The unyielding abdominal viscera also cause *over-rising of the lower ribs* and widening of the epigastric angle. The ribs on the left side are a little the more convex because of the underlying heart. The area of cardiac dullness is often increased and the apex displaced outward. The left ventricle is more exposed, and after death may exhibit a white patch due to attrition against a head.

Breathing is increased in frequency, and is chiefly diaphragmatic, if the thorax is much affected. Lung expansion is inefficient. Partial collapse of the lung with emphysema of the anterior portions is found under the lateral grooves. Bronchitis, broncho-pneumonia, and patches of collapse, are often present.

The spine is normal in mild cases. In severe ones it projects in a gradual kyphotic curve in the dorso-lumbar region, from the middle of the back to the sacrum when the child sits up. The curve is apt to be mistaken for commencing dorsal caries, but, being due to laxity of the ligaments, it almost invariably disappears when the child is suspended or is laid on the belly with the legs extended. Permanent deformity may result from contractures, or from bony changes in erect and prolonged cases. The bodies of the vertebrae are unduly soft and vascular. Between them and the discs there is a bluish-white proliferating zone of cartilage. An anterior curve may be produced in the cervical region by the child sitting cross-legged with his hands on the floor in front to support himself, the head falling back between the shoulders. Lordosis is rare. It may be associated with pelvic deformity. Lateral curvature, commonly dorsal with convexity to the right, in children under three years of age, is usually rachitic, and may be associated with some rotation of the vertebrae.

The pelvis varies in shape with the age of the child at the onset, and the posture assumed. It is apt to be deformed, more triangular than oval, and is occasionally very irregular or "crumpled." The promontory of the sacrum is driven forward,

and the antero-posterior diameter of the pelvis diminished. The pubic arch is widened, and the acetabula become placed more anteriorly, leading to a waddling gait. The iliac crests are thickened. These changes are due to the weight of the body and abdominal viscera, and may prove subsequently a grave difficulty in labour.

Upper Extremity.—The clavicle may be thickened at the sternal end; prominent about the middle from greenstick fracture; or may project upward and forward at the junction of the inner with the middle third of the bone, as the result of muscular traction and the weight of the arm. The infrapneumatic fossa tends to undue convexity. In an infant who supports himself on his hands, or crawls on hands and knees, the humerus develops a forward and outward curve about the attachment of the deltoid. Its epiphyses, especially the lower one, are enlarged. Much epiphyseal change takes place in the radius and ulna, notably at the lower ends, and gives rise to the appearance called "double-jointedness." In crawlers these bones develop an antero-external curve, and are very liable to greenstick fracture. Pronation of the hand is due to a spiral bend in the radius. The hands are not characteristic, though an attempt has been made to describe a rachitic type. The fingers are somewhat hyperextended, and may seem beaded or spindle-shaped, the joints being smaller than the middle of the phalanges on account of periosteal thickening. Neuman states that this thickening can be demonstrated by X rays.

Lower Extremity.—Deformity is more common because of the weight of the body in standing and walking. The femur curves forward and outward. If the child is unable to stand, and sits much on the nurse's knees, the bone may curve directly forward in consequence of the weight of the leg. A cross-legged attitude in sitting causes rotation of the bone outward, and the toes are then much turned out in walking. Coxa vara is due to the weight of the trunk.

Knock-knee is more common in females. It is due to an absolute or relative overgrowth of the internal condyles of the femora, or to weakness of the lateral ligaments. Bow-legs in slight cases are apparent only, being due to epiphyseal enlargement. In children who can stand there is some outward curvature, but the main curve is an antero-posterior one, usually situated, and perhaps extreme, at the junction of the middle and lower thirds of the bones. In rare instances the curve is the result of subperiosteal fracture. Bowing may be unilateral, and associated with knock-knee on the other side, or unequal on the two sides. Enlargement of the lower tibial epiphysis may be the only rachitic sign in the lower limb. The bones of the foot are not appreciably affected, even in severe cases. Obviously, slight deformities of the limbs are due to epiphyseal overgrowth, while more severe and permanent ones result from bending of the bones or from subperiosteal fractures. These fractures result from trivial causes. They are usually of the greenstick variety, and situated at the point of greatest curvature of the bone. They are most common in the radius, ulna, clavicle, and ribs, in the order given. They may occur during sleep, perhaps without pain, and are often overlooked. Much callus is formed.

Ligaments and Muscles.—Laxity and elongation of ligaments is a common, and perhaps severe, symptom. In association with muscular hypotonicity, it produces that extraordinary mobility of joints in which, for instance, the toes can be put in the mouth or the feet at the back of the head, though weakening of the capsule of the hip-joints, a condition known as *acetabular rachitis*. Spinal curvature, knock-knee, overextension of the knee (call-knee, genu recurvatum), weak

ankles, and flat-foot, are due to this cause. The muscles are small, flabby, atonic, and badly developed. It is impossible to estimate accurately the relative parts taken by the weakness of the ligaments and that of the muscles in the production of these deformities. Probably muscular weakness is the primary factor, while the secondary ligamentous failure is more apt to end in permanent trouble. The motor functions generally are impaired, delayed, or lost. The child is late in learning to sit, stand, and walk. This is advantageous in that it prevents deformity from the weight of the trunk. Sometimes the muscles are so weak and ill-developed that the child is supposed to be paralyzed. Such weakness may be very marked, although the skeletal changes are slight. The electrical reactions are normal or exaggerated. The total result is an arrest of growth and development, especially of the jaws, pelvis, and lower limbs, and the child is undersized.

Abdomen.—The abdominal and intestinal muscles are weak, the contracted chest pushes down the abdominal viscera, and the pelvis is flattened. Excess of carbohydrate food, disordered digestion, and intestinal catarrh, give rise to fatulent distension. These factors produce the *pot-belly* or *frog-belly*, in which the abdomen is uniformly enlarged, tense, and tympanitic. The stomach and colon are much distended by fermentation of their contents. The recti are often separated (diastasis of the recti), and umbilical hernia may be present.

I am doubtful of the existence of a true rachitic enlargement of the liver or spleen. Granted that such enlargement is often present, there are generally other causes which would account for it. In a huge number of cases there is no reliable evidence of true hyperplasia. Apparent enlargement of the liver, normally large in the infant and projecting into the abdominal cavity because of the more horizontal position of the ribs, is due to rachitic deformity of the chest and laxity of the ligamentous supports, causing downward displacement and greater exposure of its surface. A portal cirrhosis, interlobular in type, has sometimes been found, and is probably due to toxemia from intestinal catarrh. Hogben (1888) described a hypertrophic cirrhosis of biliary type; but jaundice does not occur. Similarly, the spleen is often to be felt on account of downward displacement. True hyperplasia may result from anemia, toxemia, and other causes.

Adolescent Rickets, sometimes called *late* or *recrudescence* rickets, is rare. From thirty to forty cases are on record, but some of them will not bear strict investigation. Two-thirds occur in females. The common age of onset is twelve to eighteen years; less often six to eleven years. Ossification is not yet complete. At puberty rapid growth takes place, and may be disordered. A typical case in a girl, eleven years old, came under my care in 1896. She was wasted, anemic, extremely feeble, and had irregular fever, up to 102° to 103° F., for sixteen days. The skeletal changes were characteristic, and under treatment there was great diminution in the size of the heads and epiphyses. Probably this was a case of the recrudescence type. Cases may be divided into three groups: (1) Recrudescence or relapse, the disease never having been completely in abeyance; (2) true recurrence; (3) primary adolescent rickets, the patient having been unaffected in childhood.

The skull is normal, for its ossification is completed. Stiffness and pain in the joints and leg muscles are common initial symptoms, and the gait may suggest hip disease. Enlargement of the epiphyses and bending of the ribs develop. The chief deformities are flat-foot, genu valgum and varum, tibial and femoral curves, *costa vara*, and *scoliois*. Unilateral cases must be diagnosed with special caution. *Costa vara* may be due to acetabular separation of the head of the epiphysis. *Scoliosis* as

puberty cannot be accepted as an isolated rachitic phenomenon. One of Clinton's cases showed premature sexual development. Some recorded cases are probably instances of osteomalacia.

HISTOLOGY.—The epiphyses are enlarged, and the bones become soft and flexible. The anatomical changes are due to increased and irregular cartilaginous and subperiosteal growth, irregularity or failure of ossification, and irregular and often excessive medullary absorption.

Normally a long bone is at first represented by a rod of cartilage. The cells in the middle, at the centre of ossification, are piled up in columns. Calcareous salts are deposited in the matrix, and enclose groups of cells in spaces, the primary areolæ. Simultaneously a layer of true membrane bone is deposited on the outer surface from the osteogenic layer of the periosteum. In the next stage apertures are formed in this new bone by the erosive action of the subperiosteal vascular osteogenic tissue, which burrows into the calcified matrix and forms large spaces, the secondary areolæ. These spaces are filled with embryonic marrow, and lined with osteoblasts, the walls being composed of calcified cartilage. Thus, at this stage the temporary cartilage is replaced by vascular osteogenic tissue.

The calcification of the cartilage extends towards the growing ends of the bones. It is followed and supplanted by the osteogenic tissue, which deposits layers of bone on the walls of the secondary areolæ. The medullary cavity is formed by progressive absorption. Throughout these stages bone continues to be deposited from the osteogenic layer of the periosteum on the outer surface, while the layers on the inner surface are being absorbed, and the medullary cavity increases in size. Calcification is only a temporary, intermediate process. The calcified tissue is absorbed, and is not converted directly into new bone. Growth in length is due to proliferation and ossification of the cartilage at the epiphyseal line. Growth in thickness is due to proliferation and ossification of the deep osteogenic layer of the periosteum.

Microscopical examination of the growing end shows several layers: (1) Hyaline cartilage; (2) a layer of proliferating cells and matrix, without any orderly arrangement; (3) a layer of cells in regular columns; (4) a zone of calcification; (5) a zone of ossification.



FIG. 5.—LARY RICKETS IN A CHILD ABOUT SEVEN YEARS.

Rachitic changes are chiefly found in the proliferating and columnar layers. The older the child, the smaller is the combined depth of these two layers. In the foetus the proliferating layer is the thicker of the two, and is said to be much enlarged in fetal rickets. In extra-uterine life the columnar layer is the larger, and is chiefly involved, perhaps attaining to eight times its normal depth. By compression between more slowly growing parts it bulges laterally, thus forming the beaded ribs and enlarged epiphyses. Another notable feature is the great vascularity of the columnar zone. Normal articular cartilage is free from blood-vessels, and even in the actively-growing rib cartilages they are few and small. In rickets the microscope reveals excessive vascularity at the epiphyseal line, abnormal proliferation and irregularity of the cartilage cells, patches of calcification in cartilaginous regions, and patches of cartilage in places which should only show calcification. The new bone seems imperfectly formed, and in places appears to have been formed by the direct ossification of cartilage, without the intervention of osteoblasts (*cf. enchondromata*). The medullary spaces are irregular and badly developed. The medulla is abnormally vascular, and contains red jelly-like material, like granulation tissue. Calcification is deficient at the growing zone, and much osteoid tissue is formed.

The cartilage at the epiphyseal line is thickened, bluish-white, softer than normal, and almost gelatinous. On the one side it blends with the cartilage of the epiphysis, and on the other it has an irregular dentate border and contains scattered calcified areas. In severe cases the whole epiphysis is involved, and the ossifying centre becomes softer, larger, and unduly vascular.

The periosteum strips off easily. It contains large blood-vessels, and may be so thickened as to encroach on the medullary cavity. In the osteogenetic layer of the cranial membrane bones similar changes take place to those in the long bones—viz., increased thickening and vascularity, excessive cell proliferation, imperfect calcification of the new tissue, and the formation of "spongoid" bone, instead of hard porous bone. Spongoid bone is thick and soft, devoid of fine salts, and not unlike decalcified bone. The deepest layers are firmly ossified.

In spongy bones there is much thickening, erosion of bony trabeculae, and the formation of large, irregular spaces filled with cells, blood-vessels, and connective tissue. This gives the ends of long bones a very porous aspect. Their shafts are abnormally flexible, because of deficient formation of periosteal bone and increased medullary absorption. They are shortened and unduly curved, and their angles are rounded off. The curves are exaggerations of normal curves, produced by the weight of the trunk and limbs. Abnormal curves result from unnatural positions and abnormal muscular action. Normal bone contains 37 parts of organic and 63 of inorganic matter. In the worst rachitic bone the proportions are 75 organic to 25 inorganic matter. Gastmann (1911) found an excess of magnesium as well as a deficiency of calcium. The bones are so soft as to be cut with a knife. Fractures result from trivial causes. They are usually of the greenstick variety.

Termination.—Eventually the active proliferation in the cartilage and beneath the periosteum is replaced by ossification. The bone becomes less vascular, normal bone is formed, and spongy masses condense and contract. Porous, spongoid bone may become abnormally dense and compact, hard like ivory. Epiphyseal swellings decrease, and may finally disappear. Beading becomes imperceptible. Cranial bones shrink, and may leave no trace. Slight bony curves disappear, and

severe ones grow less. The active process is over by the end of the first dentition. Premature union of the shafts and epiphyses may cause arrest of growth.

PATHOGENESIS.—Either imperfect nutrition or chronic inflammation may be invoked as the causative factor of the rachitic process, which may be summed up shortly as consisting of increased vascularity of the medulla and growing portions of the bones, with secondary excessive cell proliferation and irregularities of calcification and ossification. When the vascularity subsides, normal bone is formed. The process is very much like that of chronic inflammation. Bones affected with chronic inflammation are deficient in lime salts. And it can be shown that an experimentally-produced hyperæmia causes proliferation of cartilage cells, absorption of newly-formed bone, and deficiency of lime salts.

The causation of the increased vascularity is difficult of explanation. Undoubtedly there is a perversion of tissue metabolism. It has been ascribed to deficient alkalinity of the blood, retention of carbonic acid, deficiency of lime salts, excess of lactic acid, toxæmia, and defective processes in the thyroid or adrenal glands. Not one of these hypotheses is wholly satisfactory.

The Lime Hypothesis.—According to this hypothesis the deficiency of lime salts in the bones is due to a deficiency of lime and phosphoric acid in the diet, defective absorption, or increased katabolism. Roloff and Voit produced rickets in pigs and young dogs by a diet containing very little lime. Baginsky and Chassat (1842) produced it in young animals by exclusion of lime from the diet. Undoubtedly, exclusion or limitation of lime in the diet causes bony changes, but these are of the nature of osteoporosis in which slight preparation is made for ossification and osteoid tissue is calcified in spite of the deficiency of lime. It differs from rickets in that it can be cured by the administration of lime salts, and the soft tissues are also deficient in lime. Moreover, in rickets there is extensive preparation for ossification and much osteoid tissue formed, but it remains uncalcified although there is plenty of lime in the diet. Uncalcified cartilage is in excess. It contains a high percentage of lime, and marked proliferation of cells is impossible without a supply of this salt.

Cow's milk contains much more lime than human milk. Farinacea are rich in lime and phosphoric acid, and they are foods on which rickets often develops. Possibly, in the case of infants fed on condensed or sterilized milk, the effect of prolonged heat on the organic compounds of lime renders them more difficult of absorption and assimilation. According to Vierolet, absorption is quite as good as in health. The amount of lime in the urine is the same as in healthy children. These salts are mainly excreted in the feces, so urinary excretion is not much guide to the degree of assimilation. An excess of fat in the diet causes soapy stools and the withdrawal of alkalis and earthy salts. But it is not a diet rich in fat which gives rise to rickets. Phosphoric acid is set free when calcium bases unite with fatty acids; so it is equally improbable that the disease is due to deficiency of this acid.

Lactic Acid Hypothesis.—It has been asserted that an excess of lactic and other organic acids gets into the blood, and, reaching the bones, dissolves out the lime. These acids can be produced by an excess of carbohydrate food and deficient oxidation. Possibly deficient metabolism leads to the formation of by-products, which affect cartilage, bone, and general body metabolism. Heitzmann regards lactic acid as an osteoplastic irritant, and claims to have caused rickets by giving it to animals persistently by mouth and subcutaneously. In flesh-eating animals

rickets was succeeded by osteomalacia; in herbivorous ones osteomalacia occurred alone. These experiments are of little value, for the diet was deficient in lime salts, and they have not been confirmed by other observers. There is no proof that lactic acid can cause rickets. It has not been found in rachitic bone. Neither lactic acid nor calcium lactate is present in excess in the urine. If the disease is due to lactic acid, lime should appear in the urine as calcium lactate, and not as calcium phosphate.

Twin Hypothesis.—The bone changes are such as might result from a chronic irritative process, possibly due to a toxin of intestinal origin. On the other hand, there is frequently no evidence of intestinal derangement. Wegner (1881) claims to have set up rickets by small doses of phosphorus, through its action as an irritant.

Hypothyroidism has been advanced as an explanation on various grounds. Thyroid extract affects the elimination of calcium and the consolidation of fractures. According to Wegner, the bone changes in thyroidectomized rabbits correspond to those of the first stage of rickets. Phosphorus is supposed to be beneficial in treatment, and stimulates the thyroid. Further, an enzyme is present in milk and is derived from the thyroid. It is destroyed by boiling, and rickets develops in infants fed on cooked milk.

Extirpation of the *adrenal glands* in animals produces a condition like rickets or osteomalacia. The internal secretion profoundly affects metabolism. Excess of the extract causes diabetes in animals.

No hypothesis is fully confirmed. The one reliable fact is the dependence of the disease on improper diet, apparently on deficiency of fat, which gives rise to increased vascularity. Both the vascularity and the bone changes are due to the circulation in the blood of some irritant, which leads to the absorption of lime and other changes. Absorption is seen in the production of craniotabes by the softening of previously ossified bone. Sometimes craniotabes appears due to unequal ossification, uncalcified areas being surrounded by calcified ones. If the blood is deficient in lime, either because of a deficiency of this salt in the food or from mal-assimilation, it may re-absorb lime from bone in order to maintain its equilibrium. Up to the present there is no proof that there is such a deficiency of lime in the blood.

Diagnosis.—Epiphyseal enlargement and beading of the ribs are generally diagnostic, but may occur in achondroplasia. A large head, widely open fontanelle, cranial bosses, craniotabes, irregular or retarded dentition, restlessness, head sweating, throwing off the clothes at night, and backward physical development, may be due to other causes, yet in conjunction are almost certain proof of rickets. A hydrocephalic head is globular in shape. In rickets the enlargement is frontal and occipital, not general; in rare instances it may result from macrocephalus. Cranial bosses and craniotabes occur in syphilis. An open fontanelle is found in cretinism and cleido-cranial dysostosis; delayed dentition in cretinism; delayed walking in mental backwardness; muscular weakness in paralysis and amyotonia congenita; and curvature of the spine in caries. If the bony and epiphyseal changes are slight and the muscular weakness great, it is difficult to be certain that they are both due to rickets. Even if the signs of rickets are marked, the extreme weakness may suggest an additional affection. Normal reflexes and electrical reactions differentiate muscular weakness from that of infantile palsy. In amyotonia they are weak or absent. In cerebral palsy the reflexes are exaggerated and associated with rigidity.

The large belly may simulate that of tuberculous abdominal disease. The pain and tenderness of scurvy must not be ascribed to rickets, for the two diseases are often existent in the same child. Hip disease and congenital dislocation of the hip must be diagnosed from *coxa vara*. Enlargement of the condyle of the femur or of the wrist may simulate tuberculous osteitis. Syphilitic epiphysitis is generally metatarsal and in the first six months of life. Rachitic changes are not often obvious until the second six months. Mentally deficient children are often rachitic, but backwardness is not due to rickets, except in so far as mental development may be retarded by malnutrition or physical disabilities, through inability to join in childish games. Many nervous and catarrhal affections have their origin in this disease. Nevertheless, the association of two diseases is no proof of their mutual interdependence, and the rachitic element may lead to error in diagnosis. As a rule there is no difficulty in the recognition of rickets, but it is important not to ascribe to this disease illness of other causation merely associated therewith.

Prognosis.—The disease is very chronic, and lasts for months or up to the end of the first dentition. Its course is modified by treatment. Periods of latency and exacerbation depend on diet and hygiene. Usually, active symptoms subside when a mixed diet is given—that is, at about the end of the first year. Relapses are rare.

Different parts of the skeletal system are progressively involved, recovery taking place in one while another is becoming affected. In the first six months of life craniotabes and heading may be the only physical signs. Later on the thorax becomes deformed, and then kyphosis and curvatures of the bones develop. The signs of improvement are diminished sweating and restlessness, disappearance of craniotabes, increasing muscular power, and improved general nutrition. Deformities slowly and steadily improve, except in very bad cases, in which they persist to a variable degree throughout life, in the shape of pigeon-breast, Harrison's sulcus, eversion of the costal arch, kyphosis, pelvic deformity, knock-knee, bow-legs, and flat-foot.

Rickets is never fatal *per se*, but it reduces the resisting power, and is an important cause of increased mortality from other diseases, especially catarrhal affections of the alimentary and respiratory systems. Laryngitis, bronchitis, and bronchopneumonia, are much more dangerous, because of the thoracic deformity and the weakness of the thoracic muscles. Death may result from laryngospasm, convulsions, tetany, or gastro-enteric disorders. The thoracic deformity retards growth by interfering with efficient lung expansion and oxidation. Apart from this, the disease has no permanent influence on the general health. Impaired epiphyseal growth may affect growth in height, and the malnutrition may delay mental development. Rachitic children often talk late and learn new words slowly. Some of them are unduly precocious through being left much in the society of adults.

Treatment.—Prophylaxis consists in the care of the mother during pregnancy and lactation; and suitable diet for the child, together with fresh air, sunlight, a daily bath, and exercise or massage. Active treatment includes hygienic, hydrotherapeutic, climatic, and dietetic measures, drugs taking a secondary place. Special measures are needed for the prevention and cure of deformities. Mild cases recover without any active treatment.

Hygiene.—(1.) the child live in well-ventilated, sunny rooms, and be out in

the open air as much as possible. He must be warmly clad and the feet kept warm, a hot bottle being placed in the perambulator in cold weather. Country air is superior to that of towns. There is no special virtue in the air of the seaside or high altitudes, except as a stimulus to appetite and metabolism. Low-lying, damp, and marshy places, and houses surrounded by trees, are unsuitable. Preference should be given to residence in a warm, dry climate, and to houses built on gravel or sandy soil, especially if there is any respiratory catarrh.

The clothing must be light and loose. Tight garments, interfering with respiratory movements, are injurious. A moderately firm abdominal binder is permissible, if there is much flatulence, but induce compression forces the diaphragm upward, and induces partial collapse of the lower lobes of the lungs. The night clothes must be pyjamas and socks, or a soft woollen or flannel nightgown tied below the feet, because of the tendency to throw off the bedclothes. The horsehair pillow must be low and firm, and have a circular central orifice if there is much sweating. The mattress should be smooth, hard, and made of horsehair; and must be aired thoroughly during the day. Body linen should be changed frequently, because of the sweating.

[The value of exercise has been mentioned. Oxidation and excretion are diminished by confinement. Encourage free movement of the limbs during the bath and at other times, and do not hamper them by heavy swaddling clothes. Exercise must be limited in severe cases, if likely to produce or increase deformity, and replaced by systematic massage.

Hydrotherapy.—Give a morning bath of sea-water or salt and water (rock salt, 1 ounce; water, 2 gallons). If the skin is irritable, add sodium carbonate (1 drachm) and starch (1 ounce). For infants under six months old give the bath for ten to fifteen minutes at 75° F.; for older children give it for a few minutes at 70° to 80° F., followed by dry rubbing with the hand or a flesh glove. Or the child may be doused with cold water while sitting in a warm bath, and then massaged. If he is blue and chilly after the bath or douch, the treatment must be omitted. Sponge the body every evening with warm water, and rub olive-oil in thoroughly.

Diet.—A liberal amount of fat and protein are essential. If the child is breast-fed, analyze the milk and ascertain the defect, supplementing it by other food or the milk of some other animal. Breast milk is the most suitable for infants under nine months of age. If not available or contra-indicated, give a modified cow's milk. Use the top half or top third of fresh milk after it has stood in the cold for two to four hours, so as to insure a sufficiency of fat in the mixture after dilution. This is preferable to adding either gravity or centrifugal cream. Avoid excess of carbohydrates in the diet. Condensed milk and proprietary carbohydrate feeds are unsuitable because of the deficiency in fat and protein and excess of carbohydrates. Do not overburden the digestion at first; or give much fat, because of the presence of, or liability to, intestinal catarrh.

For an artificially-reared baby begin with a mixture of equal parts of top milk, barley-water and lime-water, giving 3 to 6 ounces every three hours at three to twelve months of age. Gradually change to top milk and barley-water equal parts, or whole milk diluted with one-third water. At nine to twelve months of age the child may have 20 to 30 ounces of whole milk; cream dr. 2 to 4, or butter dr. $\frac{1}{2}$, or *el. morrhue*, t.i.d.; porridge and milk, or rice and milk, twice daily; and custard pudding once daily.

At twelve to eighteen months allow milk, oz. 30 to 60; cream, oz. $\frac{1}{2}$, t.d.s.; the yolk of an egg lightly boiled, milk puddings, bread and butter, bread and bacon fat, and some cereal food for breakfast. Less milk, diluted with one-third water, must be given if there is constipation or impaired appetite. Margarine, beef dripping, and brains can be given. Mashed potato should be given after the twelfth month; and pounded-up fish, chicken, and red meat, can be added at fifteen to twenty-one months of age. Fresh fruit juice and the yolk of egg should always be prescribed. Salt can be given freely. There is probably enough phosphoric acid in the tissues to serve on any spare sodium. Under such a diet the fatty fat due to excess of carbohydrates is gradually got rid of, and the child becomes firmer and stronger.

Drugs.—It is difficult to estimate the value of drugs for the disease subsides at twelve to twenty-four months or earlier under proper diet and management. Lime salts are valueless. Lime-water is only of use as a diluent and antacid. Oil morrhuae is the only drug of any real value, and cannot be replaced efficiently by other oils, perhaps because it is more readily absorbed, or because it contains substances of peculiar value in rickets. It must be given in small doses as soon as the state of the alimentary canal is normal. Otherwise it is apt to set up diarrhoea. Give it for months, except on Sundays and in hot weather or if the stomach is out of order. It can be given pure or in emulsions with maltine, syr. fer. phosph. co., syr. calcis lactophosph., lime-water, etc.; or in mixtures such as—(1) Oil mor., oz. 2; glycerin, dr. 2; pulv. assae, dr. 2; maltine, dr. 4; syr. calc. hypophosph., dr. 4; aq. cinnamon, ad oz. 4. (2) Oil mor., oz. 1 to 2; calcium and sodium hypophosph., ss gr. 16; ol. camell. min. 2; glycerin, oz. 1; miscil. tragacanth., ad oz. 4.

Although hypophosphites, phosphates, glycerophosphates, and lactophosphates, are often given, there is no certain proof of their value. Hypophosphites are said to be excreted unaltered in the urine. Phosphorus has been recommended by Kassowitz as "the iron of rickety children," in drachm doses of mixtures such as—(1) Gr. i. in ol. amygdal. dulcis oz. 21; (2) gr. i. in ol. morrhuae oz. 20 to 30. On the other hand, it has been pointed out that phosphorus is an irritant, and sets up changes in bone suggestive of rickets. I believe the effect is due to the oil with which it is given. If phosphorus is desired, it is best given in the form of organic compounds present in yolk of egg, hard roe of fishes, and calves' brains; phytin, a vegetable preparation; peptylin, a synthetic compound of phosphorus and albumin; and fersan, an albuminous compound of phosphorus and iron.

Special Measures.—Digestive disorders are curable by suitable diet and the treatment of any cause present. Half a grain of hyd. c. creta can be given every second or third night; and a mixture of sod. sulphocarb., gr. 3 to 5; tr. nucis vomicae, min. $\frac{1}{2}$ to 1; tr. rhei, min. 5 to 10; ol. ricini, min. 10 to 15; aq. calces, ad dr. 1, t.d.s. For anaemia and general flabbiness give iron or arsenic—e.g., vin. ferri, min. 20 to 30; ferri sulph. exsicc., gr. 2 to 4; tr. fer. perchlor., min. 5 to 15; or a mixture of fer. et amm. cit., sod. bicarb. and tr. nuc. vomicae, between meals. For profuse sweating sponge with tepid water, and give a small dose of tr. belladon. or liq. atropinae at bedtime; and quinine tannate, gr. 1 to 2, with glycerine and water, t.d.s. I have never seen any decided benefit from adrenalin. If there is a convulsive tendency, add sod. bromide, gr. 2 to 4, to each dose of the cod-liver-oil emulsion, or give a dose at night.

Encourage aeration of the blood by efficient ventilation and the removal of any cause of respiratory obstruction, such as adenoids and enlarged tonsils. Breathe

ing exercises are often injurious, for the chest wall sinks in during inspiration, and deformity may be exaggerated.

Of Deformities.—Efficient treatment prevents serious results. Grave deformities are a reproach to the medical attendant. The tendency to spinal curvature is counteracted by not allowing the child to sit up, but it is absurd to keep a mild case constantly recumbent. The amount of exercise must depend on the severity of the disease and degree of deformity. All exercise and massage must be followed by rest. Otherwise the tired muscles still further fail to support the joints, and the ligaments yield. Sitting must be prohibited in girls with active rickets, because of the pelvic deformity to which it gives rise. Moderate exercise and massage help to prevent and cure bow-legs, knock-knee, and flat-foot. Long external splints of unequal length will help to keep an active child off his legs, but he is apt then to crawl about unaided and develop curves of the upper limbs. The midday sleep and recumbency in a perambulator or spinal carriage in the open air are also of use in limiting activity. For bowing of the tibia, a plaster splint can be applied from a little above the knee to the ankle, and fastened by three straps, round the ankle, below the knee, and round the middle of the calf. The last one is the most important. It is fastened round a leather pad on the outer side of the calf, and is tightened daily. External splints must be worn at night if the limbs are curled up in bad positions. An external splint is useful for knock-knee; in severe cases a Thomas's splint may be needed. Osteotomy, for the cure of rachitic deformity, should be postponed until the seventh year, for many severe cases recover with practically normal limbs. The natural tendency of growing bone is to resume its normal shape. The general treatment of deformities must be directed to prophylaxis. Massage and exercise are preventive and curative, and cannot be replaced by instrumental appliances. The latter should be looked on as chiefly of temporary value and a means of enabling a child to take more exercise than would be possible otherwise.

2. SCURVY.

Scurvy in children is due to a deficiency of meat and fresh vegetables. It is like the disease in adults, and is rarely seen in this country. Infantile scurvy was long overlooked, although Glisson, in his "Treatise on Rickets" (1699), recognized and described it as distinct from rickets, and having no essential connection therewith. In 1873 Ferguson published the case of an infant aged fifteen months. Cases were described by Sir Thomas Smith in 1874 as "haemorrhagic periostitis"; by Gee in 1881 as "periosteal cachexia"; by Cheadle in 1878 as "scurvy supervening on rickets," and in 1882 as "osteal or periosteal cachexia and scurvy." It has also been called "acute rickets," "scurvy rickets," and "haemorrhagic rickets," but these names are inaccurate, convey an erroneous idea of its pathology, and should be discarded. Cheadle recognized the affection as true scurvy, yet, as the result of a full description by Sir Thomas Barlow in 1883, showing that the scurvy was the central factor, and the rickets variable and unimportant, it has been known by the name of *Barlow's disease*.

Scurvy may be defined as a constitutional disease dependent on some prolonged error in diet, and characterized chiefly by pain and tenderness, anaemia, dusky gums, and hæmorrhages. Subperiosteal hæmorrhages are not common in the scurvy of adults. In infants the actively-growing periosteum is very vascular and liable

to hæmorrhage. The disease occurs chiefly in infants fed for many months on cooked milk or proprietary foods. It is easily curable, but is very chronic if untreated, and may prove fatal.

Ætiology.—Scurvy is often unrecognized because of its slight severity and rapid subsidence under change of diet. Its prevalence in infants coincides with the rapid extension of artificial feeding and the production of proprietary foods in the latter half of the last century. It is becoming less prevalent, for its recognition has led to the use of simple preventive measures. It occurs in any climate, in town and country, and at all times of the year. An open-air life may delay its onset. It is rather more common among private than hospital patients, because the former are more apt to be kept for prolonged periods on the unsuitable diet. An individual susceptibility has been noted. One of twins may be alone affected, although both have been reared in the same manner. Males are a little more susceptible than females. The disease is rare under six months and after fifteen months of age; 75 per cent. of the patients are six to twelve months old. Fat, anæmic babies are as much affected as the ill-nourished.

Dietetic Factors.—In 553 collected cases the diet consisted of:—Proprietary foods, 250; sterilized milk, 122; pasteurized milk, 40; boiled milk, 14; humanized milk, 6; uncooked cow's milk, 5; condensed milk, 72; dried milk foods and water only, 21; breast milk and additional food, 2; breast milk, 21. Cases in the breast-fed and in the first few weeks of life must be accepted with caution as genuine scurvy. Obviously, the diet most likely to cause the disease is the one farthest removed in character from human milk and from fresh, as opposed to cooked, food. Some proprietary foods induce it, although given with fresh milk. Babies fed on a partially cooked food, such as pasteurized milk or a partly sterilized and modified milk mixture, only become scorbutic after many months.

Symptoms.—The onset is gradual, with fretfulness, impaired appetite, anæmia, malaise, and loss of weight. The first sign to attract attention may be immobility of the legs or general tenderness, the child crying when handled, or even approached, for fear of being moved. These symptoms may have existed for a week or two previously, and yet have subsided. In such a case the actual cause has not been recognized, and the temporary improvement has been due to some change in diet. Occasionally epistaxis or hæmaturia is the first and only symptom.

Provided that the teeth are cut or almost through, the gums may exhibit swelling, hyperæmia, and slight purplish discoloration. In the mildest cases they are unaffected. In severe cases they are swollen, bulbous, ecchymotic, dusky to purple in colour, and may ulcerate and bleed freely (*hæmorrhagic gingivitis*). Sometimes the swelling forms a fungating mass large enough to hide the teeth, sloughs develop, and the breath is very offensive. Hæmorrhagic discoloration of the hard palate may be present.

Tenderness and pain induce complete immobility or pseudo-paralysis of the limbs. The legs are extended and everted, sometimes flexed and rigid. These symptoms depend on local pathological changes. The most common site is the lower third of the thigh, occasionally the clavicle, scapula, and ribs, and rarely the upper limb. The swelling, not invariably obvious on inspection, is due to subperiosteal effusion of blood in the first instance. This may cause separation of the epiphysis from the diaphysis, and soft crepitus. Occasionally the long bones fracture. The overlying skin is smooth, tense, shiny, perhaps bluish, not hot, and

sometimes pits on pressure. Hemorrhages may occur over the flat cranial bones, forming large bluish swellings like sarcomata. The thorax is often tender. The sternum may sink in from fracture or from separation at the costo-chondral juncture, producing a peculiar flattening of the front of the chest.

Anæmia is generally well marked, and is independent of hemorrhage. The blood is deficient in red cells and in percentage of hæmoglobin. Bleeding often takes place from the nose, not infrequently from the kidneys, and occasionally from the ear, into the eyelids, behind the eyeball, from mucous surfaces, into the muscles, under the skin, and into various serous cavities, the cranial cavity, the joints, and, indeed, any organ of the body. Intrarenal hemorrhage may give rise to albumin, blood, and casts, in the urine, simulating acute nephritis. Albuminuria may occur alone. True nephritis is a rare complication. Pyelitis is sometimes present.

Fever is due to complications, or to the general malaise and disturbed sleep induced by pain. Muscular pains are severe and cause loss of rest. Fainting attacks are not uncommon. Diarrhœa is of serious import, and broncho-pneumonia apt to prove fatal. The state of the gums may interfere with feeding.

MORBID ANATOMY.—Blood is extravasated into the actively growing periosteum, between it and the diaphysis, leaving the bone bare and impairing its nutrition. Necrosis is rare. The periosteum may be completely separated, the epiphyses detached, and the shaft free in a cavity full of blood. New bone forms in the detached periosteum, for its osteogenetic power is not destroyed. At the junction of the cartilage and the shaft the lymphoid cells in the marrow are replaced by a reticular tissue poor in cells, and often the seat of hemorrhage. The changes in the marrow and the destruction of osteoblasts lead to inefficient formation of new bone. Consequently the normal processes of bone absorption cause thinning of the bone in these regions, rarefaction, partial dislocation at the epiphyseal line, and partial or complete fracture from very slight violence. Such fracture gives rise to little displacement, for the periosteum is untear.

PATHOGENESIS.—The disease is the result of the deficiency of some property in fresh food, which is destroyed by heat, or of an inadequate supply of fresh food. The effects of this deficiency are exaggerated by an unsuitable diet containing an excess of starch.

The heating of milk reduces its antiscorbutic properties in direct proportion to the height of the temperature and the duration of exposure. The heat destroys the enzymes, changes the proteins in some manner, and converts soluble salts of lime into insoluble salts—e.g., insoluble calcium phosphate—as well as forming a crystalline and less soluble calcium citrate from the more soluble amorphous salt. Hence condensed milks and proprietary foods containing milk are very deficient in antiscorbutic power.

According to the citric acid hypothesis, scurvy depends on a deficiency of, or change in, the citrates in the diet. Citric acid is present as calcium citrate (C. E. Colette) in cow's milk up to 0.1 to 0.15 per cent. It is destroyed by sterilization. On the other hand, the addition of citrates to the diet, or of alkaline phosphates to keep the citrates in solution, has no apparent remedial action. Nor is the disease due to the lack of other vegetable salts. Potash salts are of no value in its treatment, and cooking does not affect the anti-scorbutic value of potatoes and greens.

Not does infantile scurvy support the hypothesis that the disease is due to *tailed food* or *protein*. There is no evidence of *protein*-poisoning; and cases are occasionally seen in infants fed on human milk and on diets devoid of animal food and *micro-organisms*.

Sir Almaroë Wright ascribes it to an acid intoxication, very similar to that produced experimentally in herbivores by the ingestion of a surplus amount of mineral acids over bases. He states that meat and cereals contain a large excess of mineral acids, especially phosphoric acid, while vegetables and fruit contain an excess of bases. A diet of meat and cereals, excluding fruits and green vegetables, will produce scurvy. He divides foods, according to their ash content, into—(1) Acid or scorbutic—viz., eggs, fresh and salt meat, and many cereals; (2) neutral or non-scorbutic—viz., sugar, vegetable oils, and animal fats; (3) alkaline or anti-scorbutic—viz., milk, blood, lime-juice, orange-juice, potatoes, green vegetables, etc. In adult cases he found that the alkalinity of the blood was decreased. He advises the administration of the salts of the oximalde organic acids, such as sodium lactate, to restore the normal alkalinity of the blood.

In contravention of these views, it must be pointed out that the reduction in the alkalinity of the blood, if present, is due to the anaemia. Joffe maintains that blood is an acid medium, for it contains phosphates of calcium and magnesium in solution, whereas they would be precipitated in an alkaline or faintly acid medium. The acidity is due to acid phosphates, but the acid reaction to litmus is masked by the excess of bicarbonates, which are acid salts. Further, scurvy does not occur in *anaemia*; and lime-juice raises the acidity of the urine, and yet cures the disease. According to Hutchinson, Wright's division of foodstuffs is based on inaccurate chemistry, and only eggs, and meat deprived of all blood, leave an acid ash. Experiments by Axel Holst of Christians (1908) quite upset the hypothesis. Scurvy was produced in guinea-pigs by a diet of bread and cereals; by a diet of compressed dried potato, but not if cabbage-juice was added; by a diet of dried carrot, even if soaked in a solution of bicarbonate of soda; and by a diet of dried barley and water, but not if the barley was allowed to sprout first. Yet in the sprouted and unsprouted barley the same amount of acids and salts was present. Nor did the addition of sodium citrate or calcium carbonate to the unmineralized diets prevent the disease. It is therefore a justifiable conclusion that scurvy is not due to acid intoxication or to reduced alkalinity of the blood.

DIAGNOSIS.—Mild cases in edentulous infants may be unsuspected and overlooked, especially if the baby is fat and its environment satisfactory. Obesity obscures a moderate subperiosteal swelling, particularly if it is bilateral and symmetrical. Anaemia, tenderness, and pain, are the cardinal signs, and often present before the gums are affected. Hematuria or frethulent and spongy gums may be the only symptoms.

Examine every infant with pain or tenderness in the limbs for other evidence of scurvy, and ascertain the nature of the diet. Rheumatism can be excluded, for it is almost unknown in infancy. The disease may be mistaken for rickets, since it is often coincident, but uncomplicated rickets is not painful. The pseudo-paralysis of the lower limbs, if present in a rachitic infant with marked kyphosis, may be mistaken for the *paralysis* paraplegia of spinal caries. In the absence of localized swelling and discoloured gums, tenderness and pain are sufficient to exclude infantile palsy. A swelling limited to one limb or epiphysis must be differentiated from acute periostitis or epiphysitis, and from *osteoma*. Syphilitic

epiphysitis occurs in wasted infants under six months old, and is generally multiple. Multiple osteomyelitis is very rare, and, like localized osteomyelitis, is associated with high fever.

Anæmia is almost invariable. In only one case of marked tenderness and spongy gums have I found it absent. Epistaxis may be erroneously regarded as accidental; hæmaturia ascribed to sarcoma; subcutaneous hæmorrhage mistaken for purpura; and the condition of the gums ascribed to acute leucæmia. Ulcerative stomatitis is not limited to the gums nor associated with other signs of scurvy. Orbital hæmorrhage, usually of the upper lid, is most suggestive. The diagnosis is often confirmed by the rapid recovery of the patient on an anti-scurvitic diet.

Prognosis.—Mild cases show definite improvement in a day or two, and get rapidly well if properly treated. In bad attacks the prognosis must be guarded, because of the liability to severe hæmorrhage, syncope from profound anæmia, and death from diarrhoea and exhaustion. Treatment may fail in advanced cases of anæmia, with extreme cachexia, and enteritis or broncho-pneumonia. Convalescence is tedious. The absorption of effused blood and thickening is slow; and the separated periosteum may form a bony sheath.

Treatment.—Prophylaxis consists in proper diet. Avoid the use of milk which has undergone laboratory treatment, physical or chemical alterations, or violent mechanical procedures, such as centrifugalization and homogenization. Give uncooked milk and the juice of oranges or grapes, sweetened, or an infusion of fresh carrots. Cream and meat-juice may be added, if necessary for nutrition, but may interfere with digestion or set up diarrhoea. Potato cream can be given to infants over six months old. It is made by adding two teaspoonfuls of the outer portions of a baked floury potato to an ounce of milk. From one to two teaspoonfuls is given three to four times daily, alone or in the first part of the milk feed. Vegetable soup, carrot and potato purées, are suitable for older children. Unboiled milk is not essential in mild cases. It may be just brought to a boil and mixed with fresh barley-water, and the juice of fruit or vegetables added or given separately.

The child must be handled, moved, and dressed, as little as possible. Bandage an affected arm to the side. Place tender legs between sandbags. Rarely the pain of rapid and extreme periosteal swelling may be relieved by a small incision and packing. Paint tender gums with an antiseptic, astringent lotion of tr. myrrh and Listerine. Sodium lactate and citrate are harmless, and can be given internally. Vinum ferri is useful for the anæmia. Give iron, arsenic, cod-liver-oil, malt, and hypophosphites in convalescence.

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CHAPTER III

SOME FORMS OF OEDEMA IN CHILDHOOD

G. F. STILL.

FETAL DROPSY.
OEDEMA NEONATORUM.
CONGENITAL OEDEMA.
OEDEMA IN WANTING CONDITIONS.
PERSISTENT OEDEMA, NOT CONGENITAL.
TOXIC OEDEMA.

OEDEMA IN TETANY.
OEDEMA IN CONGENITAL SYPHILIS.
OEDEMA WITH ANEMIA.
ARCTO-NECROTIC OEDEMA.
OEDEMA WITH PURPURA.

THERE are forms of oedema which are peculiar to infancy, and there are others which, although they may persist into adult life, first come under the medical man's notice in infancy or childhood. The oedema which is due to heart disease or to nephritis is considered elsewhere; the present chapter is concerned only with that which occurs apart both from cardiac and from renal disease.

Fetal Dropsy.—The earliest period at which oedema may show itself is during intra-uterine life, when the foetus may be the subject of general dropsy. This condition is not inconsistent with live birth, but as a rule, if born alive, the infant has survived but a few hours, or at most a few days. The most important observations on this curious affection have been collected by J. W. Ballantyne in his work on antenatal pathology. They are briefly summarized in the following account:

The mother is usually a multipara approaching the menopause. Sometimes two or three successive pregnancies result in a similarly affected foetus. The mother herself has seldom shown dropsy or albuminuria. Syphilis also is but rarely present in the mother, whose health, indeed, during pregnancy has not been greatly affected, although in many instances there has been some vague ailing. The oedema of the foetus is general, but one part of the body may be more affected than another; there is usually more or less ascites, and sometimes also fluid in the pleura. Various congenital abnormalities have been found associated with the oedema, such as malformation of the heart, the vessels, the uterus, or the bones, but none of them appear with any constancy, so that they can hardly be supposed to bear any causal relation to the oedema. The kidneys also show no constant variation from the normal, and there seems no reason to believe that the oedema is of renal origin. This statement, however, requires qualification, for some have supposed that renal disease in the mother may be the cause of the oedema in the foetus; but, as already mentioned, there is no evidence of maternal nephritis in many of these cases; its aetiology is, indeed, obscure.

Ballantyne points out that the placenta is usually large, soft, and oedematous, and he considers that, amongst various causes which may account for fetal dropsy,

may be conditions increasing the blood-pressure in the placenta, and causing thereby structural changes in it, which result in backward pressure and transudation of serum in the foetal body. Another group of causes may be structural anomalies in the foetus itself, whether in its blood or in its organs, in which case the oedema may sometimes be of cardiac or renal origin. Such, however, are probably exceptional.

Oedema Neonatorum.—In extra-uterine life the earliest appearance of oedema is that known as "oedema neonatorum." This condition is met with usually in feeble infants, and is often associated with a subnormal temperature, a weak pulse, and poor respiratory movements, resulting in more or less atelectasis. It is noticed usually within twenty-four hours after birth, and is of very serious significance inasmuch as the infant affected with it usually dies within a few days. This, however, is not always the case, and as post-mortem examination—in some cases, at any rate—shows nothing more than pulmonary atelectasis, recovery seems to be possible, if only the infant can be stimulated to stronger respiratory efforts.

TREATMENT.—With the object in view of increasing the respiratory movements, the infant may be placed in a hot bath, and its head and shoulders splashed with cold water; gentle flicking with a towel dipped in cold water may also be used to excite crying. The infant should be kept warm before a fire, and its strength maintained by beginning food earlier than one otherwise would, and it may be advisable to add four or five drops of brandy to some of the feeds.

Congenital Oedema.—Many cases have been recorded of persistent oedema dating actually from birth, and presumably of intra-uterine origin, though different in pathology from those already described as oedema of the foetus. One group of such cases is that described by Milroy, who drew attention to the occurrence of congenital oedema in several generations of the same family. Since then several instances of familial affection have been recorded, and in nearly all of these it has been congenital. It is not, however, essentially a congenital disease, for in some members of these families it has first appeared in childhood or about adolescence. This familial affection, now known as Milroy's disease, occurs with about equal frequency in males and females. It affects only the legs, which are swollen with a leaeny, hard oedema. There is seldom any pain with the oedema, which persists throughout life, though in some cases recorded by Hope and French there were repeated attacks of pain and swelling in the legs, with heat and redness of the skin, and some rise of temperature.

The oedema seems to interfere little, if at all, with the general health of the patient, and is even consistent with energetic exercise, although for the prevention of discomfort it may be necessary to bandage the legs firmly. Milroy recorded the occurrence of this affection in twenty-two out of ninety-seven persons in six generations of one family. Recently Hope and French described thirteen cases in five generations, and Rolleston three cases in two generations of one family.

The duration of life seems to be affected little, if at all. Many of the recorded cases had reached adult years; some even their three-score years and ten, or more.

It has been supposed that this form of oedema is allied to such vasomotor neuroses as Raynaud's disease and factitious urticaria. Hope and French point out the resemblance of the acute exacerbations in their cases to attacks of angio-neurotic oedema, to which some observers have thought that Milroy's disease is allied.

There are cases which resemble closely the familial affection mentioned above, but differ in being *sporadic*. Sutherland has recorded one such case in an infant, aged seven months, who showed oedema of both feet, extending up to the ankles. The swelling had been present from birth, and was associated with occasional attacks of urticaria or lichen urticatus—a fact suggestive of a vasomotor origin.

The writer had under his care an infant, aged five months, in whom both lower limbs had been oedematous from birth. In this case there was *some* cutaneous *scars* occupying the inner side of the left thigh. This association suggests some abnormality in the venous or lymphatic channels rather than any vasomotor disturbance.

There are other cases of congenital oedema in which the distribution of the swelling is altogether irregular. These are less rare than the familial affection. Usually one limb only is affected; sometimes, however, the arm and leg of one side are both swollen, or the distribution may be quite irregular, involving, for instance, the arm on one side and the leg on the other.

The writer had under his care a female child, in whom there was swelling of the right side of the face and of the right forearm and hand from birth. It was still present when the child was last seen—at the age of about nine months. In this case the swelling on the one side was permanent, but there was at times some swelling also of the left upper limb. The oedema in these sporadic, as in the familial cases, is of a *bruecy*, hard kind, but allows of distinct pitting on firm pressure.

The growth of the affected limbs is sometimes distinctly in excess of that in the sound ones, so that the swollen arm may be not only larger in circumference, but also larger than the other. This tendency to excessive growth in the oedematous limbs is, of course, a serious factor in prognosis where one leg is affected, as a difference of half an inch in the length of the legs will entail some limping in gait.

It is difficult to suppose that all these congenital cases are of the same character. In some, no doubt, there is a congenital abnormality of vasomotor function; in others it would seem that there is some anomaly of the lymphatics; in others, again, it may be that the veins are in some way abnormal, or the existence of abnormalities, both in the veins and in the lymphatic paths, may underlie the oedema. In this connection it is noteworthy that, as has been discovered in recent years, the true origin of the lymphatics is as outgrowths from the veins, not as dilated spaces in connective tissue.

TREATMENT.—The only treatment which appears to have been of any value, and that only by relieving discomfort, is the application of firm pressure, whether by firm bandaging or by the use of an elastic stocking in the cases where the lower limbs are affected. Where there is reason to suppose that abnormalities of the lymphatics may play some part in causing the oedema, it is conceivable that the operation of lymphangioplasty devised by W. S. Handley may be of value. Benefit has been recorded from electrical treatment, and there is no doubt that temporary diminution of the oedema can be obtained by skilled massage.

Oedema in Wasting Conditions.—Perhaps the commonest cause of oedema in infancy is wasting, whether this be due to prolonged failure of digestion or to chronic diarrhoea. In older children, also, extreme wasting, especially with abdominal disease—for instance, *tuberculosis mesenterica* and the so-called “*caducal disease*”—may be associated with dropsy. In such cases it is very common for oedema to appear

first on the dorsum of the hands and feet, and thence to extend up the legs and forearm. Often therewith the swollen limb is cold, and shows a blue mottling, and on pressure there may be deep pitting. The urine in these cases is usually free from albumin, or shows only a very slight trace, which is not uncommon with diarrhoeal affections in infancy. The causation of oedema with these wasting conditions is by no means clear. It has been suggested that, as in other forms of oedema, retention of chloides in the system may play some part in its production. As clinical evidence showing that the salts of the blood plasma do exercise a part in the occurrence of oedema, the fact may be mentioned that sodium citrate, given in

excessive doses to an infant already wasted, will sometimes produce a well-marked general dropsy. Inasmuch, however, as it occurs almost always when the child has fallen into a condition of much wasting and exhaustion, it seems likely that some altered state of the blood, and perhaps of the walls of the bloodvessels, together with feebleness of circulation, may be sufficient cause for the oedema.

Certainly the occurrence of this phenomenon must always be taken as an indication of extreme gravity. It would not be correct to say that it always passages a fatal ending, but in the majority of cases it is followed by such a termination. There are, however, sufficient exceptions to this rule to make it important in every case to use all possible means to avert this ending.

TREATMENT.—The treatment is that demanded by the particular wasting disease which has caused the oedema, but in addition this symptom usually indicates the urgent necessity for free stimulation. Whatever may be its ultimate pathology, it is associated almost always with conditions of exhaustion and feeble circulation, and alcohol, antivenic, and the maintenance of the body heat by hot-water bottles or otherwise, form an important part of the treatment. In view of the fact that sodium citrate, which diminishes the coagulation of the blood, sometimes produces general oedema, it seems reasonable to suppose that calcium lactate or calcium chloride, which have the opposite effect upon the blood, may have some value in preventing and diminishing oedema in these cases.

Persistent Oedema, beginning in Early Life, but not Congenital, is a condition which has many times been recorded, and is for the most part entirely obscure in origin.

In some instances the oedema has appeared in infancy. Croder Griffith, and Newcomet, record the case of a boy who was quite free from oedema until he had reached at the age of three months. At this time, or shortly afterwards, swelling of the face and of the left leg appeared. That on the face varied in degree, but both were still persisting at the age of four years.

The accompanying photograph shows a child in whom oedema of the left leg first appeared at the age of six years without any apparent cause. Hughes, who recorded this case, and has kindly permitted the use of this photograph



FIG. 7. — PERSISTENT OEDEMA, BEGINNING IN EARLY LIFE, BUT NOT CONGENITAL.

Swelling of the left leg since the age of six years. (Hughes' case, *Lancet*, 1912).

here, tells us that after the lapse of fourteen months the oedema persists unaltered.

Langmead records one in which oedema began in the left leg at the age of twelve years, and then spread to the right leg, and subsequently to the hands, and was still present at the age of eighteen years.

Many of these anomalous cases of more or less localized and persistent oedema have been recorded. The urine is found to be free from albumin, and no abnormal blood condition is present. Their pathology remains extremely obscure. The persistency of the condition seems opposed to any angio-neurotic origin, although, as already mentioned in connection with congenital oedema, there are cases in which the occurrence of recurring urticaria, or even of more definitely angio-neurotic attacks in patients thus affected, at least suggests the possibility of some relation between these conditions.

No treatment has been found to have any effect upon these cases of persistent oedema.

Toxic Oedema.—At all periods of childhood cases of oedema are seen in which the explanation of the oedema appears to be some auto-intoxication.

In some at least, perhaps in the majority, the source of the toxin is gastro-intestinal. In infancy an attack of diarrhoea is sometimes complicated with general dropsy, quite apart from any wasting or exhaustion. In later childhood foul stools, perhaps loose and undigested, are occasionally associated with a general oedema, which gives to the child all the appearance of an acute nephritis, although the urine is found to be entirely free from albumin.

In a boy of eleven years, who came under the writer's care for dropsy without albuminuria, the stools were extremely offensive and unhealthy, although there was no diarrhoea. In this case the urine was found to be considerably diminished in amount. Diminution of urine has been noted by various observers in these cases of non-albuminuric oedema, apparently dependent upon some toxæmia. Goodhart refers to this diminution of the urine as a marked feature in some of these cases, and also to the association of effusion in the serous cavities with the dropsy, instancing the case of a boy, aged four years, in whom general anasarca was associated with some ascites and some fluid in one pleura.

In infancy a general oedema is sometimes associated with the excretion of urine of extremely low specific gravity. At this age a specific gravity of about 1000 is normal, but in the cases now under consideration the urine may show a specific gravity of 901, although the quantity is not apparently increased. It is almost or entirely colourless, and urea is sometimes barely appreciable by the ordinary tests. The child with such general dropsy has the appearance of acute Bright's disease, but the urine is entirely free from albumin. Apart from some pallor there are no other symptoms, and with a return of the urine to the normal state the oedema may entirely disappear. No doubt such a condition implies a temporary depression of the metabolic activity, but the reason of this is unknown.

Perhaps under the same heading of "Toxic" should be included the cases in which general oedema without albuminuria occurs after scarlet fever. Many such cases have been recorded, and their origin is obscure. Henech and others have insisted upon the possibility of acute nephritis without albumin in the urine, and consider that some of these cases following scarlet fever may be instances of this exceptional occurrence.

Edema in Tetany.—This association, a by no means infrequent one, may be mentioned here as possibly belonging to the toxic variety. Usually the swelling is limited to the hands and feet, but may extend some way up the limbs. It has been suggested that the edema is usually due to venous obstruction, the veins being compressed by the tonic contraction of the muscles affected by the tetany. There are, however, cases in which the face also is edematous, and it is clear that this cannot be due to any such cause; and as there is much in the clinical history of tetany to connect it with intestinal toxæmia, it seems probable that the edema may also be due to this cause, at least in some cases.

It must be mentioned that tetany is often associated with a slight albuminuria, whether edema is present or not. There is no reason, however, to suppose that the edema in such cases is due to any renal condition.

Edema in Congenital Syphilis.—It is difficult to say what is the explanation of the edema which is occasionally seen in infants with congenital syphilis, but the association may be mentioned here as possibly depending upon a toxic condition. In some such cases it is true that the infant is marasmic, and the dropsy should be classed with that due to wasting; but there are others in which the child is not wasted, the urine is normal, and the only explanation which suggests itself for the dropsy is the presence of more or less marked anemia.

Treatment.—The treatment of edema of toxic origin must necessarily depend upon the source of the toxæmia—and this is often a matter of conjecture. When the offensive or unhealthy character of the stools makes it probable that this may be the source of the condition, intestinal antiseptics, such as salol, creosote, or *β*-naphthol, should be given, and irrigation of the bowel with a weak solution of boric acid or of resorcin may prove valuable.

The diet also may require special modification, particularly in the direction of excluding foods which are specially liable to produce fermentation.

Where tetany is present, the administration of bromide, to relax the spasm of the muscles, seems to favour the disappearance of the edema—a fact which supports the idea of a purely obstructive origin.

In the syphilitic cases the treatment will naturally be the administration of mercury in the form of grey powder or otherwise.

Edema with Anæmia.—In infancy and childhood, as at other periods of life, a profound degree of anemia from any cause may lead to more or less dropsy. Thus, in infancy splenic anemia is sometimes complicated, not only by edema of the limbs, but by dropsical effusion into the pleural and peritoneal cavities; and in later childhood such conditions as lymphadenoma and acute lymphatic leukemia in the advanced stage may be associated with more or less general edema.

With improvement of the blood condition, the dropsy may disappear entirely, but the occurrence of edema in such cases must always be taken as of grave significance.

Angio-neurotic Edema (Acute Circumscribed Edema).—Amongst the many varieties of anomalous edema in childhood, that supposed to depend upon vaso-motor disturbance, the so-called "angio-neurotic edema," is one of the most curious.

In certain children particular foods are apt to induce a sudden localized swelling—for instance, of one limb, or of some part of the face. In one child oatmeal porridge always produced swelling of one half of the face within a few minutes

after it was taken. In another, strawberries produced sufficient swelling of the face almost to close both eyes. T. Fisher has described the case of a girl, aged nine years, in whom a warm bath repeatedly produced sudden swelling of the eyelids, which lasted from twelve to twenty-four hours. Sometimes no obvious cause is to be found for the oedema which may recur many times in various parts of the body in the same child; the photograph shows oedema of one half of the face which appeared suddenly in a child of five years and eight months who had suffered with a previous attack of sudden oedema at two and a half years, on both occasions without apparent cause.

Obviously such an affection bears a very close relation to urticaria, which is equally bizarre in its causation, but they are related also to another group of cases in which a nervous origin is not so clear—namely, certain varieties of purpura. To these reference will be made more fully below; here it is only necessary to point out that in some cases of simple purpura transient oedema occurs, quite localized and quite irregular in distribution, exactly resembling, in fact, angio-neurotic oedema.

In Henoch's purpura, also, a similar feeting oedema is sometimes seen. This latter form of purpura at least seems most reasonably attributed to a toxic origin, whether bacterial or otherwise, and on this account the possibility arises that angio-neurotic oedema also may be rather toxic than nervous in origin. It has even been suggested that urticaria and angio-neurotic oedema may be instances of anaphylaxis.

There are also some rarer cases which seem most naturally explicable by this hypothesis of toxic origin—children in whom irregular pyrexia, which lasts many weeks or months, is associated with the occurrence of sudden outbursts of acute circumscribed oedema.

In one such case—a girl, aged five and a half years, in whom the fever lasted for at least a year, sometimes reaching 105°—there were sometimes attacks of shivering, and at other times oedema would appear in an eyelid, or in a hand or foot.

It is difficult, however, to reconcile this view with some of the clinical facts; for instance, the case mentioned above, in which oedema followed repeatedly whenever a warm bath was given. Moreover, there seems to be a special liability to this angio-neurotic oedema in persons of nervous temperament. The fact also that heredity constitutes one factor in this form of oedema seems opposed to any view which would give to it the significance of a protective reaction.

Quincke, in 1882, and several observers since, have recorded instances in which



FIG. 8.—ANGIO-NEUROTIC OEDEMA IN A BOY AGED FIVE YEARS AND EIGHT MONTHS.

Sudden and temporary swelling of the left side of the face without apparent cause.

several members of a family have shown this affection. One of the most notable was that recorded by Osler, where twenty-two out of thirty-nine persons in five generations suffered from angio-neurotic oedema.

The swelling usually gives rise to no discomfort, except perhaps a feeling of tension or stiffness in the affected part. It pits on pressure, and is not tender to touch. Sometimes there is a purplish discoloration, so that the patch may look almost purpuric. It is not associated with any constitutional disturbance, except in so far as the disease—for instance, Henoch's purpura—with which it may be associated causes any disturbance.

The onset of the oedema is usually sudden. Its duration may be hours or days.

Cases have occurred in infancy, but these are rare. It is more common in children from four years upwards, and it is said to be more frequent in boys than in girls. Its only risk consists in the liability to its occurrence in the larynx. Some have thought also that it may effect the lung, producing acute pulmonary oedema. Certainly, in the large majority of cases, this affection is quite an innocuous one in childhood.

TREATMENT consists in the avoidance of the particular food, or other cause, known to produce the attacks. Arsenic seems to be of value sometimes in preventing recurrences, and during the attack calcium lactate is worthy of trial.

Oedema with Purpura.—As already mentioned, there is a close association between the transient attacks of circumscribed oedema and purpura. With so-called simple purpura, a sudden swelling will sometimes appear in one eyelid, or in one limb, or elsewhere, without trace of hæmorrhage in the swollen part; while in other parts of the body typical patches of purpura are appearing. For instance, a girl, aged seven years, was under the writer's care, with large spots of purpura, which kept appearing for several weeks on various parts of the body. During this period the child at one time developed a sudden swelling in the left upper eyelid. This lasted only a day or two, but some days later an extensive oedema of the chest wall appeared in the right axilla. This also proved quite transient, and at another time one hand became slightly swollen for a day or two, and subsequently one foot. None of these patches were purpuric, nor were they tender, and the child's temperature was generally normal.

Similar oedema is frequent with Henoch's purpura, and some have even thought that the severe intestinal symptoms of colic and passage of blood, which are the distinguishing features of this form of purpura, may be due to a swollen condition of the mucous membrane corresponding to that in the cutaneous tissue.

It is tempting to regard the oedema which is associated with purpura as toxic in origin, especially as it is clear that purpura itself is often due to poisons, whether endogenous or exogenous. It must be remembered, however, that the same cause, whatever it may be, which renders the vessel walls unduly pervious to serum, may conceivably, if it be carried a small stage farther, allow of the passage of blood-cells; so that purpura may simply be a further stage of one and the same affection.

On this view an angio-neurotic oedema might be associated with actual hæmorrhage, although the underlying cause was a purely nervous disturbance.

TREATMENT.—The oedema in these cases calls for no special treatment, beyond that which is suitable for the purpura; for this arsenic is sometimes distinctly of value, and in the abdominal variety described by Henoch polyvalent anti-streptococcic serum has been found useful (*cf.* Chapter IX., p. 541).

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CHAPTER IV

DISEASES OF THE ALIMENTARY SYSTEM

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DISEASES OF THE MOUTH :

Stomatitis.

Ulcers, Pseudomembranous.

Perforans.

ANOMALIES OF THE TONGUE AND PALATE.

Deformities of the Jaws.

Affections of the Gums.

Eruption in Infancy :

Yeasting.

Colic.

Simple Diarrhoea.

Cyclical Vomiting.

Intoxication in Later Childhood.

Constipation in Infancy.

Constipation in Later Childhood.

Congestive Dilatation of the Colon.

Infective Diarrhoea.

Colic Disease.

Colitis :

Acute Colitis.

Chronic Colitis.

Muco-Membranous Colitis.

Intestinal Obstruction.

Appendicitis.

DISEASES OF THE STOMACH :

Congenital Pyloric Stenosis.

Dilatation.

Hæmorrhage.

Ulcer.

Tumours.

DISEASES OF THE RECTUM AND ANUS.

Intestinal Parasites.

DISEASES OF THE MOUTH.

Catarrhal Stomatitis.—The catarrhal form of stomatitis may either occur alone or along with one of the other varieties. It is commonest in the first two years of life.

ETIOLOGY.—The inflammation may result from trauma, from irritation by too hot liquids, or from infection, the latter being probably the cause of the stomatitis met with occasionally in the course of one of the specific fevers. Eruption of the milk-teeth seems prone to act as an exciting cause.

The **SYMPTOMS** are mainly local, and consist in increased flow of saliva, along with redness and swelling of the lining membrane of the mouth; the gums are somewhat swollen, and the tongue thickly coated. The interior of the mouth feels hot, and is apparently tender, so that the infant often refuses food. Constitutional symptoms are slight or absent, and the submaxillary lymph-glands are usually not affected.

The disease runs a short course, and no special treatment is required, but all foods should be given cold or nearly so. Locally, a lotion of 10 grains of borax to the ounce of water may be used as a wash.

Maculo-Fibrinous Stomatitis.—This form of stomatitis is sometimes also spoken of as "aphthous," but the latter term is best avoided, as it is apt to lead to confusion. As the description "maculo-fibrinous" indicates, the condition consists in spots of fibrinous exudation into the superficial layers of the mucous membrane. It is not uncommon after the first year. The chief cause of the disease is want of

cleanliness in the feeding-utensils and surroundings of the child, and the condition is therefore most commonly met with in hospital practice. Like the catarrhal form, it often occurs during the eruption of a tooth. No definite micro-organism has yet been identified in association with it.

The SYMPTOMS are much the same as those of catarrhal stomatitis, but there is usually more constitutional disturbance, and slight fever is commonly present. The adjacent lymph-glands are frequently somewhat enlarged.

Inspection of the mouth shows the presence of numerous yellowish-white spots situated on the lips, margins of the tongue, lining membrane of the cheeks, and palate. The spots vary in size from that of a pin-head to a split pea, but they often run together, forming larger patches. Pathologically, they consist of a fibrinous exudate into the deeper layers of the epithelium, from which the surface layer is soon shed off, leaving a superficial crust with a yellowish-white base. The latter is finally thrown off, and the spot heals without leaving a scar. The diagnosis involves no difficulty, and the disease runs a short course.

TREATMENT is the same as in the catarrhal form, but chloride of potash should be used locally (10 grains to the ounce), and may also be given in solution in the proportion of 2 grains every two hours. In obstinate cases the spots may be touched with a 2 per cent. solution of silver nitrate, or, as Holt recommends, finely-powdered burnt alum may be applied with a camel-hair brush.

Ulcerative Stomatitis.—The ulcerative form of stomatitis is only met with after teeth are present in the gums, and is specially apt to occur when caries is present. Any enfeeblement of the general health acts as a predisposing cause. The exciting agent has been shown to be a spindle-shaped bacillus (*Bacillus fusiformis*), an anaerobic organism with definite morphological and staining peculiarities. Along with it there is usually found a corkscrew spirochaeta, which, however, is met with only on the surface of the lesions, and is believed to be of parasitic nature. Numerous pyogenic organisms are always to be found as well. It is interesting to note that the *B. fusiformis* also occurs in cases of Vincent's angina and noma, and there is therefore reason to believe that these affections, as well as ulcerative stomatitis, are merely different manifestations of the same process. (See also Chapter VI., p. 267, and Chapter XII., p. 661.)

SYMPTOMS.—The inflammation begins as a marginal gingivitis round the necks of the teeth, leading to swelling of the gum, which becomes of a livid colour. Ulceration then appears round the teeth, the ulcers having a sloughy base, from which a fetid discharge takes place which imparts a strong odour to the breath. Ulcers may also form on the lips, tongue, and lining of the cheeks, especially where these are in contact with ulcerated surfaces on the gums, and in the severer cases a superficial necrosis of the jaw may even occur.

The general symptoms are more marked than in the forms of stomatitis already described, and consist in fever, malaise, and more or less depression. The tongue is usually thickly coated, and there is a marked disinclination for food.

DIAGNOSIS.—The condition has to be diagnosed from mercurial stomatitis and from scurvy, but the history and other symptoms will usually lead to a correct conclusion. The disease lasts for a few days, although it is sometimes more prolonged when the general health is poor, but complications are, fortunately, rare.

TREATMENT.—Local treatment consists in the use of a lotion of permanganate of potash (1 in 1,000), or peroxide of hydrogen (1 in 4), applied frequently as a wash. Any loose teeth should be removed, and if the ulcers prove indolent they may be treated with silver nitrate (2 per cent.), or burnt alum may be applied as in the muculo-fibrous form. Internally, potassium chlorate is of great value, and may be given in 2-grain doses every two hours for the first day or two. The strength should be maintained by abundant feeding, fresh fruit and vegetables being given liberally. During convalescence an iron tonic is indicated.

Gangrenous Stomatitis (*Scorpi, Cancrum Oris*).—This form of stomatitis is, fortunately, but rarely met with at the present day, except in children living in institutions under bad hygienic conditions. It usually occurs before the age of five.

Ætiology.—The chief predisposing cause is debility induced by one of the acute specific fevers, especially measles, but it sometimes arises spontaneously in poorly-nourished and weakly children. That the coming on of the gangrenous process is an infective agent is generally admitted, but it is not yet agreed whether it is always a specific micro-organism. Some cases appear to be due, like ulcerative stomatitis, to the *B. fusiformis*; in others an anaerobic bacillus in dense spiral threads (*cladotrix*) has been described; whilst in yet other cases streptococci have been found. It seems probable, on the whole, that, given the predisposing conditions, the disease may be produced by several different organisms.

Symptoms.—The cheek is the part of the mouth which is the site of the disease, and it is rare for more than one side to be involved. The first sign is the appearance of a heavy swelling on the outer surface of the cheek; the skin over which looks glossy and of a waxy pallor. Inspection of the inner aspect shows a bleb, which rapidly gives place to a dark sloughing ulcer, from which a fetid discharge is poured out. Meanwhile, the constitutional symptoms are often surprisingly slight, and the child does not appear in pain, and may take its food well.

As the ulceration spreads, a necrotic area appears on the outer aspect of the swelling, which soon breaks down, so that the whole thickness of the cheek may be tunnelled through. The destructive process now spreads rapidly in all directions, the gums being invaded and destroyed, the teeth loosened, and the surface of the jaw attacked. An indescribably foul odour is given off from the affected area. The constitutional symptoms become severe, fever and great depression set in, delirium may be present, and death usually ensues either from diarrhoea or a septic pneumonia.

The **Diagnosis** is easy, as the disease can be mistaken for nothing else, and the prognosis is always grave, not more than 15 per cent. of the cases recovering. The whole duration of the process is usually less than ten days.

Treatment.—It is now generally agreed that the best chance of effecting a cure consists in early and free excision of the necrotic area by cutting wide of it through the surrounding sound tissues, and afterwards cauterizing the edges of the wound. Otherwise, the treatment is the same as for ulcerative stomatitis. Strict isolation of the patient is desirable, especially in institutions.

Thrush.—Thrush is a parasitic form of stomatitis very commonly met with in infancy, especially during the first two months of life.

Ætiology.—The disease is due to the growth of a vegetable parasite, but it has not yet been finally settled to what botanical class this belongs, although the

question has given rise to much discussion. Some (Robin) regard it as an *Oidium albicans*; some adopt the view of Grunwitz that it is a saccharomycos; whilst others consider it, with Flourens, to be more closely allied to *Monilia candida*. The fungus consists of long filaments composed of unequal segments, each usually possessing a retractile nucleus at both ends, and mixed up with the filaments are a number of oval spores. The growth of the fungus is favoured by an acid reaction in the mouth, and by anything which lowers the vitality of the child. Infection usually takes place from dirty feeding-bottles or "comforters," and is facilitated by anything which produces an abrasion of the mucous membrane, such, for instance, as too vigorous cleansing of the mouth. The disease is certainly to some extent contagious.

SYMPTOMS.—The symptoms are the same as those of catarrhal stomatitis, but on inspection of the mouth the white patches produced by the growth of the fungus are easily seen. They appear first upon the tip of the tongue and the inside of the lips, but may spread so as to involve the whole lining membrane of the mouth, and sometimes coalesce over an extensive area. In conditions of pronounced cachexia the parasite may even spread down the oesophagus, where it can be readily recognised after death. The mucous membrane between the patches of growth exhibits a condition of intense catarrh.

The disease is commonly associated with gastro-intestinal disorders, and especially with diarrhoea, in the production of which it may play a part. An intertrigo is not uncommonly seen upon the buttocks, which is often ascribed by the mother to the thrush "passing through the child."

DIAGNOSIS.—The diagnosis is not difficult, but the deposit may closely simulate milk-curd, from which, however, it can be distinguished by the fact that it is adherent to the mucous membrane, and when detached leaves a slightly abraded surface. In case of doubt a microscopic examination of a portion of the growth macerated with liquor potassæ will at once reveal the nature of the disease.

PROGNOSIS.—The prognosis is, as a rule, quite favourable, but in cases in which the strength of the infant is much reduced the presence of the disease may be a bar to recovery by interfering with the taking of food.

TREATMENT.—The prevention of thrush consists in taking care to insure cleanliness of the nipple, feeding-bottles, and comforter. When the disease is established, the bowels should be opened with a dose of castor-oil, and indigestion corrected by a mixture of rhubarb and soda. Locally, mild antiseptic lotions should be applied after every feed by means of a soft camel-hair brush or a cotton-wool swab. Glycerine of borax, or a solution of salicylic acid (1 in 25%), or permanganate of potash (1 in 1,000), are suitable applications.

Gonorrhoeal Stomatitis.—Gonorrhoeal stomatitis is sometimes met with in young infants as the result of direct infection. The symptoms are those of an intense catarrhal stomatitis, along with the formation of yellowish-white patches on the tongue and hard palate, and it can be diagnosed by a bacteriological examination of the buccal secretion. General infection of the blood has sometimes supervened upon the disease, but the prognosis is generally favourable. Treatment consists in the local use of nitrate of silver solution (1 per cent.).

Streptococcal and diphtheritic stomatitis have also been described.

Ulcers Pterygoidea (Bodley's Aphthæ).—These consist in a bilateral superficial ulceration, the ulcers being situated over the base of the pterygoidea. The ulcers

are usually about the size of a pea, superficial in character, and surrounded by an area of inflamed mucous membrane. They are met with exclusively in the newly-born, or at all events during the first few weeks of life, and are apparently painful, so that they render sucking difficult. In exceptional cases the ulcers become joined at their margins, so that a horseshoe-shaped area is produced.

Their mode of origin has given rise to some discussion, but they are probably the result of injury in washing the mouth, the mucous membrane being rather tense and vulnerable at the site affected. As a rule they heal rapidly, but if they prove indolent they may be touched with a 2 per cent. solution of silver nitrate.

Perleche (German "*Fauler Ecken*").—This condition, for which there is no English term available, consists in a form of ulceration which affects the angles of the mouth. It is met with chiefly in children of school age in the poorer classes, especially in the winter months, and is probably contagious, but it is doubtful whether it is due to a specific organism. The disease begins with a roughening of the skin and mucous membrane of the angles of the mouth, and is usually symmetrical. As the result of stretching of the parts by the opening of the mouth, cracks and fissures form, from which a serous exudation is poured out. A sensation of burning and discomfort results, which causes the child constantly to lick the affected area, whence the French name for the disease. The condition usually lasts for two or three weeks, but heals spontaneously without producing scars.

The Diagnosis is not difficult. Herpes may simulate it, but is usually not symmetrical, and shows the presence of vesicles. Congenital syphilitic plaques are distinguished by the history and the presence of other signs of specific disease.

Treatment consists in the local application of a weak mercurial ointment, or of a dusting-powder of orthoform. Deep fissures may be touched with silver nitrate.

ANOMALIES OF THE TONGUE AND FRENUM.

Congenital Defects.—The anterior portion of the tongue may be congenitally absent. The whole tongue is never completely undeveloped, since the arrest of growth only occurs in that part derived from the tuberculum impar. Irregular development of this portion may produce the split or bifid tongue. The defect may easily be closed by making raw the adjacent surfaces and then suturing.

Hypertrophy of the tongue due to overgrowth of the muscles is generally seen in cretins and congenital idiots. Partial excision is of value if obstruction to respiration is caused by the enlargement. Excessive length of tongue has been recorded, and is generally associated with a lax frenum. As a consequence of the abnormal mobility, "tongue swallowing" may ensue with fatal results. The tongue either curls upon itself so that the tip occludes the pharynx, or the base falls backwards, obstructing the upper opening of the glottis. An acquired mobility may ensue as the result of injudicious operating upon the frenum for so-called "tongue-tie," when tongue-swallowing is likely to occur. Two such cases, which ended fatally, were under my care at the Children's Hospital, Great Ormond Street. Attempts at fixation and control by posture are of little avail, and, if the patient does not die of suffocation, septic pneumonia generally sets in.

"Tongue-tie" is a myth of heavy antiquity. Butlin records three genuine cases. The frenum with the mucous plicæ on either side are the natural anterior anchorage of the tongue. Their length is variable, and they may be short from

delayed development. Subsequent growth invariably allows all normal functions of the tongue to be perfectly performed. The reason that the mother so frequently adduces for her suspicion of "tongue-tie" is that the child cannot suck properly, and tosses its head away from the breast when feeding. This is almost always due to nasal obstruction either from adenoids or syphilitic rhinitis, or from the breast closing the nostrils during suckling on account of the flatness of the nipples. Since the mouth is full at the same time and nasal respiration is impossible, the infant naturally gets out of breath. Appropriate treatment for any of these causes soon allays all anxiety about the condition of the tongue. In addition to tongue-swallowing, fatal hemorrhage and serious septic ulceration have followed improper operative procedures upon the frenum.

Tumours.—*Dermoids* of the tongue may develop in its substance between the geniohyoid muscles, and are said to have their origin in a persistent thyroglossal duct. Others on section have the appearance of true thyroid gland-tissue. In a boy, three years old, obstructive dyspnoea had been produced by a large median cyst which was growing from the site of the foramen caecum. On excision, it was found to be continuous with the upper end of the thyroglossal duct. After removal and stitching the site of the wound, the patient had no further trouble.

Angiomas, lymphangiomas, and tumours with these two elements mixed are very common. When localized, they may be easily excised. If diffuse, they can be destroyed gradually by puncture with the actual cautery. In a child, aged three years, in whom the whole of the anterior two-thirds of the tongue was a diffuse mass of naevoid and lymphangiomatous tissue, repeated cauterization was necessary for a year before the whole mass was destroyed. The tongue is now fully mobile, and articulation and deglutition normal.

Erythema Migrans, or Geographical Tongue, is a type of superficial chronic glossitis very common in children. The small red tender areas spread, and by contrast with the surrounding surface of the unaffected portion of the tongue have greyish or whitish circinate margins. The cause is unknown and the condition unimportant. Spontaneous cure as a rule results. It occurs most frequently in debilitated children.

Black Tongue, in which the free ends of elongated filiform papillae are discoloured black by the action of bacteria, is not infrequently present in children. The lesion is not serious, and no treatment seems to be effective.

Ramula is a name applied to swellings arising in any gland whose duct opens beneath the tongue on the floor of the mouth, due to blocking of the duct. Mucous and salivary glands may be alike affected. Excision is the best cure for them, but when the cause of obstruction is an impacted calculus, simple removal of that is often sufficient.

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DEFECTS IN JAW.

These defects are as a rule congenital, affecting both jaws as the result of deficient development.

In the *upper jaw* failure of the bony elements of the two sides to unite results in one of the many varieties of cleft palate. Various displacements of the premaxillary element may be associated with this condition. In extreme cases failure of the nasal processes of the superior maxilla to develop results in a cleft extending on the surface from the base of the skull to the floor of the mouth. Premature synostosis of the palatal plates of the superior maxilla may lead to vaulting of the hard palate. This condition encroaches upon the nasal respiratory passages,

leads to overcrowding of the teeth, and is frequently associated with a defect of speech resulting in "nasal" intonation.

In *prognathous development* of the upper jaw proper apposition of the teeth of the two jaws is prevented, and prolonged treatment at the hands of a skilled dentist will result in great amelioration of this disability. In the *lower jaw* defective development affects as a rule the horizontal process only. Complete absence of this portion on one side has been recorded. The whole of the lower jaw may be undersized in proportion to the rest of the bones of the face, with the result that the lower teeth are overlapped by the upper ones when the mouth is closed. Considerable improvement may be effected by the use of restraining apparatus and appropriate exercises.

Eruptions occur rarely, more frequently in the lower jaw than in the upper, and should be removed if producing any pressure effects.

Osteosarcoma, a group of tumours formed within the substance of the jaw from excessive proliferation of one or more of the tooth elements of the second dentition, are but

rarely found in children. In exceptional instances they have been noted as present at six months of age, although advice with regard to them has not been obtained until many years later. More have been recorded between the ages of seven and fifteen years, but as a rule adolescence has been reached before they have attained such a size that removal has been urgently sought. In the fibrous variety it has been suggested that rickets is a predisposing cause.

Osteitis is a name given to a variety of tumour springing from the margin of the gum. The structure of the tumour cannot be determined until a section of a portion of it has been examined under the microscope. Simple inflammatory masses, fibromata, angiomata and sarcomata—both myeloid, round, and spindle-celled—make up the group. Removal is indicated in all cases. Fortunately, the



FIG. 9.—PHOTOGRAPH OF GIRL, ABOUT NINE YEARS, WITH SWELLING OF THE LEFT SIDE OF THE LOWER JAW, DUE TO THE PRESENCE OF A MIXED-CELLLED SARCOMA.

sarcomata appear to be of low malignancy, and after free removal do not show any great tendency to recur.

Malignant Tumours of the jaws are extremely rare. As a rule they are either myelomata or mixed-celled sarcomata. In the accompanying picture (Fig. 9) the swelling of the lower half of the left side of the face was produced by a large bony tumour of the body of the left side of the mandible. Examination of sections after removal showed that its structure was in parts that of a myeloma and in parts that of a spindle-celled sarcoma. The patient was aged nine years.

Inflammatory Diseases of the jaw start as a rule from some carious focus in a tooth. The upper jaw and the body of the lower jaw are consequently the most frequent sites of the trouble. The lesion may be localized, forming the so-called "alveolar abscess," or may be so diffuse as to involve a very large area of the jaw, resulting in suppurating periostitis or osteomyelitis. Drainage by incision is followed by most satisfactory results. If, however, the ascending ramus of the lower jaw is involved, free drainage even through the mouth is almost impossible, and formation of sequestra is a frequent consequence.

Chronic Ostitis and Periostitis of a simple nature may be the result of neglected dental caries. More rarely it is due to syphilis, tuberculosis, and actinomycosis. Surgical treatment is indicated in most cases, unless a speedy disappearance results from proper care of the teeth or the use of antisyphilitic remedies. A Wassermann reaction should never be omitted as an aid to diagnosis.

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AFFECTIONS OF THE OESOPHAGUS.

Congenital Malformations.—The congenital malformation of the oesophagus most commonly met with is that in which the upper half of the tube terminates as a cul-de-sac, and the lower half opens into the trachea on the posterior surface a little above the bifurcation. This condition is incompatible with life, and terminates fatally as a rule at about the seventh day from birth, although survival until the twelfth day has been recorded in one case. Plastic surgery fails on account of the difficulty of the procedure involved, in addition to the fact that the tissues at the site of operation are inevitably septic.

Atresia of the Oesophagus, similar to the congenital atresia affecting all other parts of the alimentary tract has been recorded. It affects as a rule the mediastinal portion of the oesophagus, and is in consequence inaccessible to direct surgical intervention.

Pharyngeal Diverticula arising from the site of the junction of the oesophagus and pharynx are said not to occur in children, although Bland-Sutton records one case in a boy aged fifteen years. He regards them as being of congenital origin, due to the persistence of the pharyngeal segment of branchial clefts. Keith, on the other hand, maintains that they are acquired, and associates their formation with the activity of the sphincter muscle of the upper end of the oesophagus. Hence their absence from children. Their presence can be recognized

by difficulty in swallowing; regurgitation of food, especially large quantities of decomposing material at some interval after a meal; and the presence of an elastic tumour in the neck, whose size varies with the ingestion of food and with its regurgitation. The only treatment is to dissect out the sac and close the opening.

Occlusion of the Oesophagus as a congenital abnormality may occur as the result of pressure by the right subclavian artery when this arises as an independent trunk from the extreme left of the arch of the aorta. The artery then passes behind the oesophagus to reach the right side of the neck. A case of this nature ending fatally has been recorded by Batten and Kellock.

Oesophageal Pouches of an acquired nature are almost always situated in the mediastinal portion of the oesophagus, and arise from its anterior wall immediately behind the bifurcation of the trachea. They are frequently attached to the posterior surface of the trachea or to one of the bronchi by a fibro-muscular cord—Ridibert's band. Sometimes an enlarged and inflamed lymphatic gland effects the union between the alimentary and respiratory tracts at this level. Constant "traction" during respiration, coughing, and deglutition, on one of these connecting bands, is held to be responsible for the formation of the pouches. Their presence may be suspected on account of dysphagia and regurgitation of food. The passage of a bougie under an anæsthetic will generally reveal the presence of an obstruction in the upper part of the mediastinum. Whilst the point of one bougie is engaged in the opening of the pouch, a smaller one can generally be passed by the side of it, down the remainder of the oesophagus as far as the cardiac orifice. This proves that the main lumen of the oesophagus is not structurally occluded by isthmus changes. Examination with the oesophagoscope with von Brünig's attachment will generally reveal the opening of the pouch. A skiagram taken after the administration of bismuth is of value in estimating the size and exact location of the sac. In a case in a boy, aged five years, under the care of Garrod and the writer, such a pouch from the mediastinal oesophagus was readily demonstrated. His chief trouble was dysphagia, which has been much improved by passing a small bougie into the sac, and then passing large ones down the remainder of the oesophagus. The only alternative to this procedure is to attempt to remove the sac by an operation through the mediastinum. The risks of septic mediastinitis following upon this are so great that it should only be adopted when all other methods of relieving the dysphagia have failed.

Acquired Strictures of the oesophagus in children are exceedingly rare. The common causes of their formation in adults—*i.e.*, the swallowing of corrosive fluids and boiling water—generally end in the immediate death of the child. In the rare event of survival, with subsequent stricture-formation, progressive dilatation by bougies must be attempted.

Foreign Bodies.—The narrowest portions of the oesophagus are at its commencement and at the level where it is crossed by the left bronchus. These are the sites of election for the impaction of foreign bodies. They may, however, become lodged and traverse the whole length of the alimentary tract. Coins, pins, brooches, nails, buttons, collar-studs, marbles, fish-bones, and bottle-stoppers, are the common objects swallowed by children. Their position may often be located by a skiagram, and removal should only be undertaken after they have been viewed by means of the oesophagoscope. Extraction by "coin-catchers" and "bag forceps"

should be abandoned except in cases of urgency where respiratory embarrassment from pressure has ensued. Much damage has been done in this way by forcible blind removal. In cases in which treatment is sought for only at a long interval after the impaction of the foreign body, septic ulceration of the mucosa of the oesophagus is invariably present. Adjacent structures may be involved, and, from perforation of the oesophagus in its mediastinal portion, fatal septic mediastinitis ensues. Perforation of the aorta from a halfpenny impacted in the mediastinal portion of the oesophagus has been recorded by Turner. With the introduction of the oesophagoscope with von Brünig's modifications, the removal of a foreign body from the lumen of the oesophagus by an external incision is seldom necessary.

Functional Dysphagia, unassociated with any detectable structural lesion, occurs in children, and presents very similar features to the disease in adults. The difficulty in swallowing is intermittent—quite lengthy periods may intervene in which swallowing is performed without any difficulty at all; it is much aggravated by observation and indignant sympathy; it is often accompanied by a good deal of spitting and spluttering; it is profoundly modified by suggestion, and for a time may make a complete disappearance; whilst, finally, under an anæsthetic, the passage of bougies and the view of the oesophagus obtained by the oesophagoscope show that no organic obstruction is present. Treatment by suggestion and discipline at the hands of a sympathetic nurse who is a stranger to the child effect a cure in most cases. Home influences should as a rule be removed, and the child placed in the suitable environment of a nursing home.

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INDIGESTION IN INFANCY.

The chief symptoms of indigestion in infancy are vomiting, colic, and diarrhoea. It will be convenient to consider each of these and its treatment separately.

Vomiting.—ÆTIOLOGY.—Indigestion which results in vomiting during the period of infancy is usually due either to excess of fat or of curd in the feeds; the former occurs both in breast and bottle fed babies with considerable frequency, but curd indigestion is commonest in those fed artificially. When excess of fat is the cause, the vomited material is yellowish in colour and of a rancid odour. Excess of curd leads to the vomiting of dense clots with a sour smell. In either case, if the irritation in the stomach be sufficiently intense, some degree of gastritis is set up, as shown by the presence of mucus in the vomits. The general symptoms consist in restlessness, pain, thirst, and fretfulness; there may be slight fever as well, especially if severe gastritis be present. The tongue is usually coated, and there is a sour smell in the breath. Colic and diarrhoea are frequently present in addition.

DIAGNOSIS.—The vomiting of indigestion has to be distinguished—

1. From the almost effortless regurgitation of *surplus food* when the stomach is overfilled by too large feeds. This is a very common occurrence both in breast

and bottle fed babies, but is not accompanied by any disturbances of the general health.

2. From the vomiting of *pyloric stenosis* (q.v.).

3. From *symptomatic vomiting*, such as occurs at the beginning of pneumonia or meningitis. At the outset of such a case indigestion is very apt to be simulated, but the appearance of other symptoms and signs soon makes the diagnosis clear.

4. In babies about the period of weaning a form of vomiting is sometimes met with which is apparently *voluntary* in character. In such a case, when solid food is given, the child may either "hawk" it up from the back of the throat, or may actually swallow it, but almost immediately reject it again. This is apparently not due to indigestion, but is either produced by an exaggerated irritability of the pharynx and stomach, or is purely voluntary and a sign of resentment against solid food. It is characteristic of such cases that the food will often be taken much better from a stranger than from the mother or the usual nurse, and with a little perseverance and firmness the vomiting soon ceases.

TREATMENT.—In all acute cases the stomach should be washed out at once with a weak solution of bicarbonate of soda. If for any reason this is not feasible, the child may be allowed to drink a quantity of tepid water, which is usually speedily rejected, and brings with it any acids which may be causing irritation. When the stomach has been thoroughly emptied, milk should be withheld for twenty-four hours, and plain water given instead. In chronic cases the method of feeding must be inquired into in detail. Care must be taken that the bottle is thoroughly clean and that the food is not given too quickly. It may be necessary also to reduce the amount given at each feed, or to lessen the proportion of fat or casein, or to render the latter more digestible by citrating or peptonizing the milk (see Prolegomena, IV.). The local application of heat to the epigastrium is useful in all cases, but drugs are not of much help. In acute cases, however, calomel is useful, $\frac{1}{2}$ grain being given every hour for six doses. In chronic cases small doses of carbonate of bismuth are helpful, especially if there be evidence of acidity.

Colic.—Ætiology.—Colic results from irritation of the bowel by the presence of gas, acid, or organic acids. Frequently all three causes co-operate. The usual source of the disorder is improper feeding, and especially the use of too strong mixtures. Want of cleanliness in the preparation and administration of the food is also a common cause. Not infrequently, however, one meets with cases, especially in very young infants, where no fault can be found with the food, but the child suffers continually from colic. In these cases it seems fair to assume that the trouble is due to an exaggerated sensibility of the nervous apparatus of the bowel, or to some lack of co-ordination in its working, which passes off in time as the intestine settles down to its work.

Symptoms.—The chief symptom of colic is pain, which is manifested by screaming. The abdomen is hard and the legs are drawn up during the paroxysms. The child is usually ravenous for his food, as each meal gives temporary ease, but owing to the great restlessness and want of sleep, the natural gain in weight is apt to be interfered with. The extremities are often cold, and during an attack the child may look blue or white round the mouth, and there may be slight twitching of the hands, feet, and face. In severe cases and in excitable infants an actual convulsion may result.

DIAGNOSIS.—The screaming of colic must be distinguished (1) from that due to hunger, (2) from that caused by pain of other origin. The screaming of hunger is more continuous and less paroxysmal than that of colic, and the practiced ear has usually no difficulty in distinguishing the two. The other forms of pain which may simulate the colic are (1) that which results from phimosis, in which case the screaming is associated always with attempts to pass water; (2) the pain of renal colic, caused by the passage of uric acid gravel (the grains of this can be detected on the napkins); (3) the pain of *enteric* or *teething*: in this case the child will be noticed to put his hands to the head or mouth when the pain is felt.

In all doubtful cases the cessation of the pain when flatus is passed is conclusive of colic as the cause.

TREATMENT.—**L. Ipecacuate.**—During a paroxysm heat should be applied to the abdomen in the form of hot fomentations or hot flannels, or in severe cases the child may be put in a warm bath. An injection of hot water into the bowel should also be given, and a few drops of brandy or of spirits *etheris nitrosi* administered by the mouth.

2. **Prophylactic.**—The feeding should be revised with a view to making the food more digestible and to obviating the formation of curds (see Prolegomena, IV.). Care should be taken that the abdomen is kept warm by a good binder, and that the feet and legs are warmly covered. Regular massage of the abdomen with warm oil is also helpful. A cammimative mixture consisting of 2 or 3 grains of bicarbonate of soda with a drop or two of tincture of ginger in 1 drachm of an aromatic water (peppermint, dill-water, etc.), sweetened with a little glycerine, should be given regularly between feeds in order to neutralize acids and aid in the expulsion of flatus. If the bowels be sluggish, a few minims of magnesium solution may be added to the mixture with advantage. If, on the other hand, the bowels tend to be loose, one may add a small dose of tincture of opium. In the case of a breast-fed baby it is often useful to let the child suck a little from both breasts at each feed, and to give an ounce of water containing a couple of grains of citrate of soda from a bottle in the middle of the feeding. In extreme cases, and if the weight is not going up, it may be necessary to wean.

Diarrhoea.—The diarrhoea of indigestion may be spoken of as simple or catarrhal diarrhoea, as distinguished from the infective or epidemic type of diarrhoea described in another section. It may be set up by an excess of any of the food constituents—casein, fat, or sugar—or by decomposition of the food (e.g., sour milk), and its occurrence is facilitated, and in some cases caused, by chill. What part micro-organisms play in its production it is difficult to say, but it can readily be understood that the presence of catarrh of the intestine, however caused, favours the growth of these, and may prepare the way for the infective form of the disease.

Symptoms.—The chief symptom is an increase in the number of the motions. The character of these varies, and depends to some extent upon the cause. Thus, when casein is in excess in the feeds, the motions are "curdy," containing larger or smaller masses of milk-clot. In fat diarrhoea the motions are loose and slimy, and contain small granular masses composed of fatty acids and soaps. At first sight these closely resemble the finer milk-curds, but can be distinguished from them by the fact that they are soluble in a mixture of equal parts of alcohol and ether. Excess of sugar is less common as a cause of diarrhoea, and the stools in such a case are usually pale, frothy, and highly acid. Whatever the cause of the

CYCLIC VOMITING.

The condition termed "cyclic vomiting" (also known as periodic or recurrent vomiting) was first described by Glee, and consists of attacks of severe vomiting accompanied by prostration, which recur at more or less periodic intervals.

ETIOLOGY.—The disease is decidedly commoner in the female sex. The attacks usually begin about the third to the fifth year (although they may date as far back as the first year), and are especially frequent between the ages of five and eight. They tend to cease spontaneously towards the period of puberty. The children affected are usually of a decidedly "neurotic" type, and tend to suffer habitually from constipation and indigestion. A family history of gas, migraine, or "bilious attacks," is not infrequent. Fatigue, chill, and excitement, may all serve as exciting causes of an attack in a child who is predisposed.

SYMPTOMS.—For some days or hours preceding an attack, certain prodromal symptoms are noticed; these consist in languor, irritability, and fretfulness. Complaint is often made of headache, the bowels are more than usually constipated, and the motions tend to be pale and offensive. In some cases the attack is ushered in by slight sore throat or a "cold in the head." As the process develops, vomiting sets in, and soon becomes urgent. The vomited matter consists at first of food, but this is soon followed by bile, and finally, in severe attacks, by grumous blood-stained material. As the vomiting goes on, the temperature usually rises, and may attain a high level. The child is at first restless and irritable, but soon becomes apathetic and drowsy, and lies with sunken eyes and a retracted abdomen, in a state of extreme prostration. Intense thirst is complained of; but even water may be vomited. Constipation is extreme, and may equal that of intestinal obstruction. Meanwhile the odour of acetone becomes noticeable in the breath, and the urine shows a marked keto-acetic acid reaction.

The attack usually lasts for two or three days, although a duration of a fortnight is not unknown, and then gradually passes off, leaving the child much reduced in weight and strength.

PATHOLOGY AND PATHOGENESIS.—In the few cases that have come to autopsy, the only constant change found has been marked fatty degeneration of the liver. Langmead lays stress upon the enormous size of the fat globules in the liver-cells, which are comparable only to those found in acute yellow atrophy and phosphorus-poisoning. The cells in the neighbourhood of the portal canals are specially affected. Slight degenerative and necrotic changes have also been found in the mucous membrane of the stomach and intestines. The exact nature of the morbid process at work in the production of the disease, and its relation to these changes, is still obscure. It is generally believed, however, that one has to do with a failure of the "antitoxic" function of the liver, induced, possibly, by an intestinal auto-intoxication. Mellanby, who has investigated a typical case very carefully from the chemical point of view, found that the patient constantly excreted a certain amount of creatin in the urine, although a normal child excretes none at all, and that during an attack the creatin excretion reached a maximum. He is inclined to attribute this to abnormal intestinal decomposition. On the other hand, the acidosis which is often such a prominent feature in cyclic vomiting, and to which many have attributed the production of the symptoms, is now

believed to be merely a by-product in the pathological chemical process at work, and to be the result simply of the acute starvation which the vomiting induces, although, when once developed, it may be responsible for the production of part of the clinical picture in an attack. It seems probable, also, that there is in these patients a permanent "hepatic inadequacy," which comes to a head, as it were, in the attack, and which may consist in or be manifested by a failure of the liver properly to deal with fat. Hence it is that on the one hand a diet which contains an excess of fat-forming food may throw too great a strain on such a liver; while, on the other hand, constipation may conduce to the process by leading to the production of excessive toxin-formation in the intestine, and the results of treatment tend to show that both these factors play a part in the genesis of the disease.

DIAGNOSIS.—In a typical case the diagnosis is easy, especially if one can get a clear history of previous attacks of a similar character. A first attack, however, may present some difficulty. *Intestinal obstruction* may be closely simulated; but abdominal pain is here an early and marked feature, whereas in cyclic vomiting it usually only comes on as the result of the retching. Early and pronounced acidosis is in favour of cyclic vomiting, and so also is a retracted rather than a distended condition of the abdomen. The mental condition is usually clearer in intestinal obstruction, and one can generally discover some local signs in the abdomen. Notwithstanding these points of difference, the diagnosis may be difficult; but great care should be taken to come to a correct conclusion, for operation on a case of cyclic vomiting is very likely to prove fatal from post-anæsthetic poisoning.

Meningitis may be simulated by an attack of cyclic vomiting, especially as the prodromal symptoms are very similar in both; but headache is usually much more, and vomiting much less, severe in meningitis. The occurrence of a spasm is in favour of meningitis, but the writer has known it be present at the outset of an attack of cyclic vomiting also. As the case goes on, the absence of all physical signs of cerebral disease enables one to exclude meningitis, and in a doubtful case lumbar puncture may be resorted to.

Appendicitis, especially when induced in by much vomiting, may have to be thought of in the diagnosis; but abdominal pain, as in *intestinal obstruction*, is usually a trustworthy guide to the nature of the case. The history is a case of recurring slight attacks of appendicitis may closely simulate that of cyclic vomiting, but here again abdominal pain will usually be a more prominent feature in the anæsthesia than it is in cases of cyclic vomiting. Local signs of disease of the appendix would, of course, clinch the diagnosis. Between ordinary so-called bilious attacks and cyclic vomiting the difference is probably one of degree rather than of kind, but in "bilious attacks" there is usually a closer relation between the onset of an attack and dietetic errors; there is not the same regular tendency to recurrence.

PROGNOSIS.—The attacks, though often very severe and alarming, are, fortunately, rarely fatal, although death has occurred in a few instances. The tendency to the disorder passes off spontaneously towards the age of puberty, but its place is then apt to be taken by some other periodic affection, such as migraine.

TREATMENT.—So soon as the prodromal symptoms appear, a smart purge should be given, followed, if need be, by a large enema. When vomiting begins,

water should be given by the mouth as long as it can be retained, and afterwards by the rectum. If collapse be marked, normal saline should be given subcutaneously or into a vein. Morphia is the only drug which controls the vomiting to any extent; it may be given hypodermically in a dose of $\frac{1}{4}$ grain to a child of five. If symptoms of acidosis be pronounced, bicarbonate of soda should be given freely by the mouth (dissolved in soda-water), per rectum, or intravenously, and glucose may be added to the enemata.

Between attacks great care should be taken to insure a regular action of the bowels. Any aperient is useful, but the mercurials appear to be of special value, and a combination of rhubarb and grey powder, in doses proportionate to the age of the child and the severity of the constipation, will usually meet the case. There is also some reason to believe that the regular administration of an alkali, such as bicarbonate of soda, is of some value in warding off attacks; it may be given with bitters before meals for long periods.

The diet should be carefully revised, the amount of milk taken being kept within strict limits (not more than a pint daily), and the starchy foods and sweets reduced (see Indigestion in Later Childhood, *infra*). Fatigue, chill, and excitement, should be guarded against.

It is necessary to add a word of warning as to the danger of administering anaesthetics to children who are the subject of cyclic vomiting owing to their special liability to suffer from delayed "chloroform-poisoning." Operation in such subjects should only be undertaken when urgently required, and, for choice, soon after an attack of vomiting is over. Special care should be taken in the preparation of the patient to guard against the superposition of acidosis by the administration of alkalies and glucose for some hours before the anaesthetic is administered. Ether should be used in preference to chloroform.

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INDIGESTION IN LATER CHILDHOOD.

1. **Acute Indigestion and Acute Gastritis.**—Acute indigestion in children after the period of infancy may result from overloading of the stomach, or from the taking of some irritating, indigestible, or decomposed article of food. Chill also may cause it. No sharp line can be drawn between simple acute indigestion and acute gastritis. All depends upon the intensity of the irritation. If the latter be sufficiently severe, some degree of catarrh is always set up.

SYMPTOMS.—The chief symptoms are epigastric pain and vomiting. At first the contents of the stomach are brought up, afterwards bile. If catarrh has been excited, mucus will be present in the vomit, and in extreme cases, where there is much retching, there may even be a little blood. The child complains much of thirst, and there is more or less prostration. The tongue is thickly coated and the breath foul. The temperature is usually elevated, and if there be much gastritis, may reach a high level, and even be attended by rigors. Occasionally convulsions

occur. The symptoms usually subside in two or three days, but in severe cases may be prolonged for a week. They often pass off with diarrhea.

DIAGNOSIS.—The diagnosis has to be made from vomiting due to other causes, such as that which occurs at the outset of the acute infections (symptomatic vomiting), from cyclic vomiting, and from the vomiting of acetonuria. Two conditions, however, must be specially borne in mind—viz., appendicitis, and acute intestinal obstruction. The distinguishing features of these are described elsewhere.

TREATMENT.—All food should be withheld for twenty-four hours. Thirst may be relieved by teaspoonsful of hot water, or by giving fragments of ice to suck. A linseed and mustard poultice may be applied to the epigastrium. Washing out of the stomach is hardly practicable at this age, but if there be any doubt as to all the contents having been brought up, one or two tablespoonfuls of tepid water may be given. These will probably be rejected and bring away any remains of food along with mucus.

Drugs are of little assistance, but $\frac{1}{2}$ grain of calomel may be given every hour for six doses with advantage. It should be placed on the back of the tongue and washed down with a little water. Afterwards bismuth is the most useful drug. If the vomiting be prolonged and exhausting, it may be necessary to give a small dose of morphia hypodermically ($\frac{1}{4}$ grain for a child of three years).

2. Chronic Indigestion.—Chronic indigestion is extremely common after the period of infancy. It often begins between the third and fifth years, but is perhaps most frequently met with about the time of the cutting of the permanent teeth. It is probable that the symptoms are due to a chronic catarrhal condition of the whole alimentary tract, but this is merely an inference; for, so far, at least, as the writer knows, no observations have been made on the state of the gastric secretion in these cases. In favour of the catarrhal nature of the affection is the fact that considerable quantities of mucus are often passed from the bowel, and it is on this account that Estace Smith applied the term "*mucous disease*" to this form of indigestion, and the disorder is often referred to by that title in this country.

ÆTIOLGY.—The disease occurs in all classes, and is perhaps just as frequent amongst children living in the best surroundings as amongst those who are less fortunate. It is frequently met with in the subjects of the neurasthenia of childhood, and there can be little doubt that many children are constitutionally predisposed to it from some inherent debility of the digestive organs.

SYMPTOMS.—The symptoms of chronic indigestion in childhood are often vague and indefinite, and not always such as to suggest an affection of the alimentary system. Perhaps the commonest complaint for which the child is brought is "wasting"—"he is getting so thin," as the mother says. In addition, she may complain that the child is irritable, fretful, and peevish, or that he is languid and dull, uninterested in toys and games, and always wanting to "lie about." He is often depressed, and sheds tears on slight provocation. "Nervousness" is also a common symptom. The appetite is usually impaired, but occasionally voracious, although, in spite of large meals, flesh is not gained; at other times it is capricious, and the patient is very fussy about what he eats, or even has a craving for quite abnormal things, eating bits of coal or pieces of lime picked off the wall (pica, or dirt-eating). Abdominal discomfort of some sort is usually present. Sometimes

it takes the form of epigastric pain, or, more often, is referred to the lower part of the abdomen, coming on in short, sharp attacks of colic soon after the taking of food. As a rule the bowels are constipated, the stools being frequently pale in colour and coated with mucus. They may show the presence of undigested food particles, and thread-worms are often present in them. At other times the motions are loose and offensive, and there is often a tendency for the bowels to act immediately after a meal, the action being preceded by colicky pain (hentero diarrhoea).

Nervous symptoms are frequent, the commonest being general nervousness and restlessness with disturbed sleep, amounting sometimes to actual insomnia, as there may be night terrors. Headache is often complained of, especially on waking, and there is not uncommonly nocturnal enuresis. Sudden attacks of pallor, sometimes accompanied by faintness, are often noticed. These are apparently the result of defective vaso-motor control, but are apt to be ascribed to weakness of the heart or to minor epilepsy.

Cough of a short, hacking character is a frequent symptom—the so-called "stomach cough." It is due to an irritable condition of the pharynx, or to the presence of enlarged tonsils or adenoids, and is unattended by any signs in the lungs.

On physical examination the child will be found to be pale, though not usually anæmic, with dark rings or puffiness under the eyes, and an appearance of listlessness and fatigue. The skin is sometimes dry and harsh, the subcutaneous fat scanty, and the muscles small and flabby. The tongue is usually coated with a thin and shiny, fawn-coloured fur, through which the fungiform papillæ stick up as red points. The breath may be offensive. The pharynx is red and irritable, the lymphoid follicles swollen, and the tonsils usually enlarged. Adenoids are often found. The abdomen is sometimes distended by flatulence, and a splash may perhaps be elicited over the stomach, but as a rule there is little to be made out of abdominal examination.

The urine is often turbid from the presence of pale urates, and may contain a trace of albumin (functional albuminuria).

DIAGNOSIS.—The combination of wasting and a hacking cough in these cases is very apt to suggest a diagnosis of tuberculosis. It should be remembered, however, that pulmonary phthisis is extremely rare in childhood, and the absence of physical signs in the lungs and the rapid improvement under suitable treatment should soon enable one to eliminate any such possibility. The other common mistake is to regard these children as anæmic, and to treat them vigorously with iron to the still further detriment of the digestion. This error can be avoided by inspecting the mucous membrane of the lips and gums, and the conjunctivæ, when it will usually be found that the pallor of the face is not due to any real want of blood. Finally, there is a great danger of treating symptoms (e.g., night terrors, cough, etc.) in these cases, whilst overlooking or neglecting the underlying indigestion which is the real cause. If the great frequency of this form of digestive disorder and its protean manifestations are borne in mind, this omission will be avoided.

TREATMENT.—1. *Hygienic* treatment is of the first importance. The child's whole life should be inquired into and regulated with a view to correcting any errors in his management. Over-fatigue, both mental and physical, must be avoided, and the limits of work, play, and rest, must be laid down. Mental strain and excitement are extremely injurious, and it may be necessary to curtail lessons and to insure

that more time is spent in the open air. Half an hour's rest in the recumbent position after the midday meal is often very helpful. An adequate amount of sleep must also be secured. The details of bathing and clothing, too, may require attention. Cold baths are usually injurious, and the limbs should be warmly covered and an abdominal binder worn in the winter months. Climate treatment is of the greatest help, and a change of air to the seaside, or a leaving mountainous place, will often do more for the patient than all other treatment put together.

2. *Dietetic*.—The chief principle of diet must be the avoidance of an excess of carbohydrates, for there can be no doubt that these are the ingredients of the food which do most harm. The following should therefore be avoided: Bread (especially wholemeal), potatoes (unless in very small quantity and mashed), porridge, starchy puddings, cakes, sugar and jams, vegetables (except spinach, cauliflower, and vegetable marrow), and raw fruits. Milk should be taken sparingly, especially when the stools are pale, and only if diluted. The diet should consist of meat (preferably mutton), chicken, fish, fat bacon, rusk or crisp toast, a little mashed potato, and the vegetables not forbidden above, custard, jellies, stewed fruit (preferably sieved), baked apples, and a little plain cake occasionally (e.g., sponge-fingers). Water should be drunk at the midday meal, and a little weak cocoon and milk at breakfast and tea.

Meals should consist of breakfast, dinner, and tea, with a rusk and a little milk between breakfast and dinner, and the same or a little clear soup at bedtime.

Great care should be taken that the food is properly chewed, and to this end it may be necessary to have the teeth attended to by the dentist. "Baking" the food should be carefully guarded against, and plenty of time allowed for meals. Liquids should only be taken at the end of the meal.

3. *Medicinal Treatment*.—The most useful drugs are alkalies and bitters (given before meals), and aperients. A mixture of soda, tart. vomica, and gentian is a standard prescription—e.g.:

Sod. bicarb.	grs. ʒ.
Tinct. tart. vom.	{	
Spiritus chloroformi	{	ss. ℥ss.
Inf. gent. ex.	ad ℥i.

To be given shortly before each meal for a child of five.

If the tongue be coated, 5 or 10 drops of compound tincture of rhubarb may be added to each dose of the above with advantage; or if nervousness be a prominent symptom, 5 grains of bromide may be substituted for the tart. vomica. Of aperients there is none better than a combination of rhubarb (pulv. rhob. 5 grains) and gly. powder (1 grain) given every night, but aloes, cascara, and senna, are also useful. So long as the stools are pale, however, mercury should be given in some form.

In the exceptional cases in which there is a tendency to hepatic diarrhoea, a couple of drops of tincture of opium may be added to each dose of the bitter mixture.

When the tongue is clean and the appetite restored, 1 drachm of malt extract may be given after each meal; and if there be any true anaemia, iron may be administered cautiously, preferably along with an aperient.

The treatment outlined above will usually be successful, but in cases in which nutrition is much impaired it may be necessary to have recourse to a modified "rest cure," just as in cases of neurasthenia.

Accessory means to the restoration of health are the correction of any error of refraction which may be causing eye-strain, and the excision of enlarged tonsils and the removal of adenoids, if these be present. The improvement in the digestion and general health which follows this operation is often surprising.

CONSTIPATION IN INFANCY.

Chronic constipation is met with both in breast and bottle fed babies, but much more often in the former than in the latter. One can distinguish between true constipation, in which several days may elapse without any action of the bowels at all, and a mere costiveness, in which a motion occurs daily, but is too small, hard, and dry.

ÆTIOLOGY.—The cause of constipation in infancy is sometimes obscure, but amongst the factors producing it may be reckoned—(1) Malformations (e.g., atresia of the rectum or anus, congenital dilatation of the colon, etc.); (2) impairment of tone in the intestinal wall or abdominal muscles, such as occurs in rickets; (3) feebleness of the rectal reflex, or a failure of the bowel to respond to it: this is not uncommon in very young babies, and is very conspicuous in mentally defective infants, particularly cretins; (4) deficiency of the digestive juices, especially, perhaps, of the intestinal juice, or bile: this seems to be associated with costiveness and dryness of the stools; (5) faults of diet. In breast-fed babies these take the form either of poverty of the milk as a whole or of a deficiency of fat in it. Human milk is so thoroughly absorbed that, if it be scanty or poor, there may be no residue left to stimulate peristalsis (physiological constipation). The constipation which is so characteristic of congenital pyloric stenosis is akin to this being due to failure of the milk to reach the bowel in sufficient quantity. In bottle-fed babies an excess of casein in the feeds is a commoner cause of constipation, and produces large hard stools which are passed with difficulty and much straining.

It is usual to assign faults in the mother's diet or the existence of constipation in herself as possible causes of constipation in a breast-fed baby, but it is extremely doubtful whether such an assumption is justified.

SYMPTOMS.—The symptoms produced by constipation in infancy are very slight. Occasionally, indeed, and especially in those fed on the breast, there may be no symptoms at all, and the child may remain in quite good health even although there is only an action at long intervals. More often there result such symptoms as colic, screaming, fretfulness, and failure to gain weight, whilst the constant straining may give rise to prolapse of the rectum, and fissure, hæmorrhoids, or even piles.

TREATMENT.—The treatment of constipation in early life will depend upon its cause. Malformations, of course, can only be dealt with surgically. Much can be done by a good nurse to establish a regular habit by accustoming the infant to expect an action of the bowels at a given time every day. If failure of voluntary effort is the cause, the use of the domestic "cone of oiled paper," or the insertion of a soap suppository, or the injection of glycerine and water (ʒi. to ʒi.) into the rectum, is usually sufficient to prompt an action. If there be deficiency of tone in the abdominal muscles or bowel, massage along the line of the colon carried out for five or ten minutes night and morning is often helpful. Some slight lubricant may be used to prevent chafing of the skin during the process.

muscles, or of secretion of the bile and intestinal juice. Impaired innervation of the intestine is, no doubt, also a factor. It is extremely difficult, however, to say which of these causes is chiefly at work in any given case, and certainly in many instances several of them are acting in co-operation.

SYMPTOMS.—The consequences of constipation in the child, as in the infant, are often surprisingly slight, but as a rule some impairment of the general health results. There is often physical languor and mental dullness, headache is frequently complained of, and the appetite is defective. The complexion is apt to be pasty, the tongue furred, and the breath offensive. The other symptoms are those which have already been described under the heading of Chronic Indigestion, but special mention should be made of the tendency these children have to suffer from "bilious attacks" and from bouts of fever of obscure causation. The urine often shows the presence of an excess of indican.

TREATMENT.—The first point in treatment is to take pains to insure a regular habit. With this object the child should be accustomed to go to stool regularly at the same time daily, preferably after breakfast, and care should be taken that plenty of time is allowed for the discharge of this necessary function. It is the duty also of the mother or nurse to see that the relief obtained is a sufficient one, for a too scanty daily evacuation constitutes a state of constipation just as truly as a failure of the bowels to act every day. This is a point which is very apt to be overlooked, and one is often assured that the bowels are "regular" simply because the child goes daily to the closet.

Diet is not of so much help in the treatment of chronic constipation as is generally believed, for it is only the slighter cases that can be favourably influenced by this means. The adoption of a "coarse" diet, indeed, containing much wholemeal bread, vegetables, and raw fruit, may do more harm than good by overloading the bowel, besides setting up flatulent distension. It is often of advantage, however, to give some stewed fruit or a baked apple at breakfast, and to see that plenty of water is taken throughout the day. In districts where the water is hard or chalky, still Salutaris or Malvern Water should alone be used for drinking. The amount of milk taken may need to be restricted, and eggs are best avoided as being a very constipating food. Honey and treacle are laxative articles of diet to many children if they can be taken without harm to the digestion.

Drops.—In all the severer cases one is obliged to have recourse to the use of drugs. These should be given in accordance with the same principles as were laid down for their use in infancy—namely, that they must be given regularly, and that the more tonic aperients should be selected for choice, cascara, aloes, and senna, being the most suitable.

Cascara may be given in the form of the aromatic elixir, or mixed with malt extract, whilst older children can usually take it in tablet form. Of the preparations of aloes, the compound decoction (5*ss*. or more three daily) is one of the best, or a small tablet or pill of aloin may be employed if the child can swallow it. Senna may be used in the form of the syrup or along with sulphur in the well-known "compound liquorice powder." It is also contained in the "syrup of figs" and in "Tamar Indian" lozenges. An infusion of the pods is another mild and efficient form in which to employ it. Any of these drugs alone, or two or more of them in combination, may be used, but stress must again be laid on the importance of giving them regularly. The dose required can only be discovered by experiment.

and in some cases very large quantities are required; but by persevering in the use of these for weeks or months, if necessary, one can usually succeed in gradually training the bowel into habits of spontaneous action.

Enemata in the case of older children are best reserved for emergency use, as their habitual employment, besides being troublesome, is apt to result in over-distension and partial paralysis of the colon.

CONGENITAL DILATATION OF THE COLON.

"*Idiopathic Dilatation of the Colon*," apparently of congenital origin, or at least unassociated with an obvious obstruction that could be productive of such a condition, was first described by Billard in 1820. Very little attention was paid to the disease until Hirschsprung's detailed monograph at the Berlin Congress for Children's Diseases in 1886. Since then some hundreds of cases have been reported. Its cause remains unknown, and most of the writings on the subject

merely reiterate the well-known features of the disease that were described in full by the earlier observers. A complete review by Finney in 1908 of the writings of 296 authors supplies full details of all aspects of the disease, offers some hypotheses of its origin for consideration, and makes reference to most of the published cases. Recent papers by Judd and Hey Groves traverse the same ground.

The disease may occur in early infancy and at all periods up to the age of seventy years. Males are more frequently affected than females, and there does not appear to be any hereditary tendency. It rarely runs a rapidly fatal course, but the ultimate issue of the disease is unfavourable in young subjects. Periods of remission and exacerbation may be present, whilst there is a raised susceptibility to



FIG. 59.—CONGENITAL DILATATION OF THE COLON OF A CHILD AGED ONE YEAR AND NINE MONTHS.

Note the enormous enlargement of the transverse and descending colon and part of the iliac colon.

intercurrent affections. Its gravity may be estimated by Löwenstein's figures, who showed that in fifty-nine cases subjected to medical treatment only the death-rate was 56 per cent., and in forty-four cases treated surgically the death-rate was 48 per cent.

PATHOLOGY.—The whole of the colon or any isolated portion may be affected. The pelvic and iliac colons are the commonest sites, and the limits of the enlargement are bounded abruptly with no gradual transition to normal intestine. A true hypertrophy is invariably associated with the dilatation, and involves all coats of the gut, but is most marked in the muscular and submucous coats. The mesocolon may be thickened, and various secondary changes in the glands and mucous lining of the intestine have been recorded. The enormous size of the large intestine is well shown in the photograph of a post-mortem view of a child, aged

two years, with the viscera in position immediately after opening the abdominal cavity.

SYMPTOMS.—Constipation of an obstinate character, distension of the abdomen, and visible peristalsis along the tract of the large intestine, unassociated with vomiting, marked pain, or a prolonged disturbance of the general health in the early stages, are the characteristic clinical features met with. The disease thus differs from cases of intestinal obstruction in an obvious manner, since such a degree of constipation and distension could not occur from an acquired obstruction, without the life of the patient being in imminent danger, whilst pain and vomiting would be marked features of the case. The constipation may be complete for so long as a month, and is then relieved by the passage of many copious decomposing motions during the course of a day or two. These attacks of constipation and diarrhoea may alternate over many years. Hyper-resistance of the large intestine is generally present during the period of distension. Dreadful cases may be recognized by the help of sigmoidoscopic examination, and of X-ray pictures, taken after the injection of an oil and bismuth emulsion.

TREATMENT.—Purgatives and diet have little effect upon the course of the illness. Abdominal massage and simple enemata are the most valuable forms of medical treatment. Provided that a daily evacuation of the bowels can be obtained by these means, whilst the general nutrition of the patient remains unimpaired, this line of treatment should be persevered with. From the beginning of the illness, however, such treatment may be ineffectual, and surgical measures must be adopted.

In most cases, moreover, medical treatment tends to fail in the end, and it is unreasonable to persist until the emaciation of the patient has become so marked as to prohibit surgical interference. Any signs of impaired general nutrition under medical treatment should be regarded as an indication for the prompt adoption of operative procedure. Excision of the affected portion of the gut is the end to be aimed at, but preliminary measures, such as entero-anastomosis, cecostomy, and appendicostomy, may be considered advisable when the general condition of the patient is bad, and the amount of intestine involved is extensive.



FIG. 11.—PHOTOGRAPH OF A BOY, FOUR FIVE YEARS, WITH CONGENITAL DILATATION OF THE COLON, SHOWING THE COILS OF THE DISTENDED BOWEL.

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INFECTIVE DIARRHŒA.

SYNOPSIS.—Summer diarrhœa; Epidemic diarrhœa; Acute gastro-intestinal infection; Zymotic or Epidemic enteritis; Cholera infantum.

The form of diarrhœa which is here spoken of as "infective" to distinguish it from the simple diarrhœa of indigestion is one of the chief scourges of infant life. It prevails epidemically in the third quarter of the year, although sporadic cases may be met with at any time, and in unusually hot seasons it may become very widespread, especially in large towns. All children below the age of five are liable, but the incidence is much more severe before the age of two, the chief fatality being amongst children in the second and third trimesters. Infants below the age of three months seem to be comparatively immune. Boys appear to be more susceptible than girls.

Ætiology.—It is generally admitted that the exciting cause of the disease is infection by micro-organisms, and the bacteriology will be dealt with later, but the predisposing causes must now be considered. By far the most important of these are certain meteorological factors, and especially a sustained high temperature. It was pointed out long ago by Rolland that the summer rise of diarrhœa mortality does not begin until the mean temperature recorded by the four-foot earth thermometer has reached about 56° F., no matter what the previous atmospheric temperature has been. Later observers consider that an average mean—or, according to some, minimum—air temperature of 60° F. is necessary to start an epidemic, and that cases only begin to occur when such a temperature has lasted about a week. The influence of a continued high temperature is probably exercised in several ways. It depresses the vitality of young children, favours the production of dust and the breeding of flies, leads to decomposition of food, and in all probability also, and perhaps most important of all, provides the conditions necessary for the growth of the micro-organisms which cause the disease.

Next in importance to a high mean temperature as a factor in producing the disease must be put unhygienic surroundings. Dirt, overcrowding, and all the other characteristics of urban life in poor neighbourhoods, undoubtedly play a very large part. To these must be added a contaminated milk-supply and want of cleanliness in feeding utensils.

The infection is probably conveyed by dust, and there is good reason to believe that flies act as carriers.

The chief predisposing factor in the child is the existence of any digestive derangement. Bottle-fed infants are very much more often affected than those fed on the breast, although the latter are by no means immune. The important influence of age has already been referred to.

BACTERIOLOGY.—In spite of much research on the subject, the bacteriology of infective diarrhœa is still far from clear. It seems tolerably certain, however, that all cases are not due to the same organism, and amongst those which have been isolated from the stools in different epidemics are the *Proteus vulgaris*, the *Bacillus coli*, a streptococcus, and the *B. enteritidis*. In America the dysentery bacillus (Flexner type) has frequently been found, and seems to be the cause of cases of the dysenteric type, but it appears to be less commonly met with in this country. Jellie has isolated a streptococcus, mostly in the form of diplococci,

which was present in large numbers in the stools, and sometimes in the blood in typical cases, attended by marked prostration. On the other hand, Morgan, in an investigation covering several epidemics in this country, has proved the association with the disease of a bacillus (Morgan's bacillus) having definite cultural peculiarities, which he believes to be causally related to the affection.

The subject is a difficult and complicated one, and it seems probable that, as indicated above, several types of organisms may give rise to the clinical symptoms to which the name "infective diarrhoea" is applied, provided the conditions in the bowel are favourable.

PATHOLOGY.—The changes found after death are often surprisingly slight, considering the severity of the disease, and seem to vary in different epidemics. As a rule the mucous membrane of the stomach and intestines is slimy or velvety in appearance, and often somewhat oedematous, showing the characters of a mucous catarrh. There may be areas of congestion, and here and there small petechial hemorrhages, and the stomach may contain a little altered blood. The lymphoid tissue of the alimentary canal, and indeed throughout the body generally, is often atrophied. In some cases there is slight pitting of the lymphoid follicles of the colon.

The liver is usually fatty or shows parenchymatous degeneration, and the kidneys often exhibit the latter change in a high degree. In a few cases one finds multiple abscesses in the renal cortex, with descending pus streaks in the medulla. Uratic infarcts are common. The lungs usually show some oedema and congestion of the bases, and in the more protracted cases small patches of broncho-pneumonia. Purulent parotitis has been observed in a few instances. The brain shows no characteristic changes, but external and internal hydrocephalus are sometimes observed.

SYMPTOMS.—The disease may set in insidiously with a gradually increasing diarrhoea, or the invasion may be abrupt, the earliest symptoms being a rise of temperature and prostration.

Diarrhoea is not necessarily a marked feature of the disease, and the worst cases are often those with fewest stools. In a few cases there is actually constipation. The character of the motions varies greatly, but usually they are greenish, slimy, and offensive, becoming dark and watery in the later stages. If the colon be much affected, visible mucus and blood may be present. The discharges are usually attended by colicky pain and the expulsion of much flatus. Vomiting is usually present at the outset, but is absent in a certain proportion of cases. More rarely it is a prominent feature, and persists even after the diarrhoea has subsided. The vomited matter consists of the stomach contents mixed with mucus and bile.

The temperature is usually more or less elevated at some period at least of the disease, but the height, duration, and course of the pyrexia, vary greatly. Hyperpyrexia often sets in at the close in fatal cases. Prostration is an early and pronounced symptom, and is shown by depression of the fontanelle, pallor, pinched features, inelastic skin, and coldness of the extremities. As a rule the child passes into a state of stupor, but sometimes restlessness, twitching, and convulsions, are seen.

The urine is scanty, and often shows a trace of albumin and the presence of a few hyaline casts.

The duration of the disease varies greatly. In the severest cases, especially

when of the choleraic type, death may ensue within a few hours of the first onset of symptoms. In the milder forms the acute symptoms last for two or three days, and then gradually the signs of nervous prostration disappear, and the stools assume a normal character. Not uncommonly, however, the improvement is only partial, and the child passes into a more or less maniac state with prolonged looseness of the bowels, from which recovery may only take place after some weeks. In all cases relapses, apparently from re-infection, are extremely common, and, owing to the exhaustion still remaining from the first attack, are very apt to prove fatal. They are prone to be brought on by a too early return to ordinary diet.

In children above the age of two the disease usually assumes a milder form, prostration is not so marked and the range of temperature is lower. Vomiting also is a less prominent symptom than in infants. Complete recovery more frequently takes place, and relapses and the continuance of the disease in a chronic form are comparatively rare.

In the special type of the disease commonly spoken of as "cholera infantum" the invasion is always abrupt, with a rise of temperature, followed by profuse vomiting and purging. Prostration is marked from the outset. The stools are frequent and large, and, although greenish and slimy at first, quickly become watery, colourless, and almost odourless. Thirst is extreme and wasting rapid so that the infant soon becomes quite shrivelled up. Death usually ensues, often after only a few hours, and is commonly attended by hyperpyrexia.

DIAGNOSIS.—The diagnosis has to be made from simple non-infective diarrhoea. At first the differentiation may be impossible, but a continued high temperature, marked nervous prostration, and very offensive fluid stools, point to the infective form of the disease. Epidemic prevalence also is in favour of the more severe variety. The nervous symptoms closely resemble those of meningitis, but the course of the disease and the results of a lumbar puncture will decide the diagnosis.

PROGNOSIS.—It is impossible to give any estimate of the fatality of infective diarrhoea, as it depends greatly on the type of the disease, and on the age, general condition, and surroundings of the child, besides varying greatly in different epidemics. The younger the infant and the poorer its general nutrition, the worse is the outlook. The existence of rickets also greatly aggravates the danger. The frequency of the stools is of less importance in estimating the chance of recovery in any given case than the amount of prostration and the degree of inelasticity of the skin. Cases with prolonged high temperature and those of the choleraic type usually do badly. Persistence of vomiting also is a bad sign.

TREATMENT.—The principles of treatment to be carried out in a case of epidemic diarrhoea are—(1) To stop the infection; (2) to eliminate and neutralize toxins; (3) to combat collapse; (4) to arrest the vomiting and diarrhoea.

1. In attempting to stop further infection, general hygienic measures are of great importance. Whenever it is possible, the child should be isolated in a large, airy, and well-ventilated room. Napkins should be removed and disinfected as soon as they are soiled, and the greatest cleanliness should be observed in regard to the feeding utensils and the nurse's hands.

If the case comes under observation early, an initial purge of castor-oil or, if there be much vomiting, of calomel should be given to clear away decomposed matter from the bowel. Milk, which is the vehicle of infection, should at once be stopped, and so long as the symptoms are severe, the diet should be limited to plain

water, albumin-water, barley-water, rice-water, or weak broths, all of which may be given freely.

2. In order to favour the elimination of toxins, the action of the skin and kidneys should be promoted as much as possible. To this end, if the skin be very inelastic, tepid packs are useful, and these may be continued, if necessary, for several hours on end. If the urine be scanty, a few drops of sweet spirits of nitre may be administered, and water should be given freely.

In the early stages of the disease no attempt should be made to arrest the diarrhoea entirely, as the retention of poisons would then be favoured. In addition to the initial purge, irrigation of the colon is a valuable aid in removing toxic material. Normal saline administered at a temperature of 100° F. through a long rectal tube or large-sized red rubber catheter is the best solution to employ.

The direct neutralizing of the toxins is, unfortunately, not at present practicable, as no efficient antitoxic serum for the disease has yet been found.

3. Collapse has to be obviated by saline infusion and the use of stimulants. Four ounces of normal saline may be injected under the skin of the flank every six hours. The injection of "sea-water plasma," which has been so much vaunted, has not been found by the writer to give any better results than ordinary normal saline.

Of stimulants, one of the best is alcohol. Ten to fifteen drops of brandy may be given in a little water every three or four hours or oftener; it should not be added to the feeds. Camphor is also useful, and is best given dissolved in olive-oil (1 in 15 or 1 in 30). Five minims of this solution may be injected under the skin as often as is thought necessary, or a few drops of spirits of camphor may be given by the mouth. If the collapse be extreme, recourse may be had to the mustard bath.

4. The arrest of the vomiting and especially of the diarrhoea is really a secondary object of treatment. For the vomiting, lavage of the stomach is the best treatment, particularly in the early stages, and a poultice may be applied to the epigastrium. Drugs are of little help, but small repeated doses of valeret (e.g., $\frac{1}{2}$ grain every hour for six doses) are sometimes useful. Chloroform in 1-grain doses may also be used cautiously. Iced champagne diluted with an equal quantity of soda-water is sometimes of service.

As has already been pointed out, no active steps should be taken to arrest the diarrhoea at the outset. If it continues, however, and seems to be contributing to the exhaustion, one may try to reduce the frequency of the stools. In the early stages, small repeated doses of castor-oil are useful for this purpose, and later, when the tongue is clean, bismuth, chalk, catechu, tannallen, tannigen, and other astringents, as in the case of simple diarrhoea, may be used. A powder composed of 5 grains of bismuth carbonate, with $\frac{1}{2}$ grain each of Dover's powder and calomel, is also a frequent prescription at this stage. Opium should not, however, be given at the outset, and is best withheld so long as prostration is marked and the temperature high. When prescribed, it may be given in the form of the tincture in the proportion of 1 minim for every year of the child's age. It is specially indicated when the motions are frequent and attended by much colic. In cases of the choleraic type with much vomiting and profuse purging, morphine may be given hypodermically with great advantage. The dose for a child of one year is $\frac{1}{10}$ grain, with which $\frac{1}{20}$ grain of atropine may be combined. This may be repeated in an hour if the symptoms have not abated.

"Intestinal antiseptics" are of little help, and may easily be injurious. The best are salicylate of bismuth, salol, and resorcin. If the stools are very watery, nitrate of silver is the most powerful astringent, and may be prescribed in the form recommended on p. 150.

After the acuter symptoms have passed off one must return very gradually to ordinary diet. Milk should be given at first fully peptonized and freely diluted with lime-water, or condensed or desiccated milk (*e.g.*, Glaxo) or one of the patent foods (*e.g.*, Allenbury's) may be used in place of it. A feed may be given every four hours, and water between if the child is thirsty. Any return of the vomiting or diarrhoea must be the signal for again giving up milk in any form. During convalescence change of air and mild iron tonics are of service in restoring the child to complete health. Relapses, which are frequent, must be guarded against with the greatest care.

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CELIAC DISEASE.

Celiac disease may be defined as a chronic wasting disorder of childhood, characterized by diarrhoea, with large, pale, and offensive stools, running a prolonged course, with a great tendency to relapses, exhibiting often certain nervous complications, such as weakness of the legs and tetany, and ending either in death or in complete recovery, or in partial recovery with great impairment of growth and development (infantilism).

The disease was first clearly described by Gee in 1888, and in the following year R. A. Gibbons published some additional cases. Many of the cases described by Chénolle under the title "acholia" also belong to this group, and in more recent times the condition was, so to speak, rediscovered by Herter, whose cases of "infantilism from chronic intestinal infection" certainly represent a severe form of celiac disease.

ETIOLOGY.—The cause of the disease is obscure. Both sexes are affected, but, so far as the writer's experience goes, girls oftener than boys. Social surroundings seem to play little, if any, part in the etiology, for the affection is at least as common in private as in hospital practice, and, indeed, some of the severest cases the writer has met with have been in children who have been surrounded with every care and attention. Any acute attack of enteritis, whether from infection, poisoning, irritation of undigested food, or exposure to cold, seems able to serve as a starting-point of the disease. The question whether an abnormal intestinal flora may play a part in the etiology will be discussed later.

SYMPTOMS.—The disease usually begins insidiously in the second or third year of life. The child is observed to be pale, listless, and fretful, and to be losing flesh and strength. If inquiry be made into the state of the stools, they will be found to be increased in number—usually about two or three in the twenty-four hours—and still more increased in bulk. They are generally of the consistence of porridge,

pale, or even quite white in colour, and of an extremely offensive odour. Sometimes they contain streaks of visible mucus, or even of blood, and undigested food particles can often be observed in them. The appetite is poor or capricious, and there is usually considerable thirst.

Physical examination reveals some degree of anaemia, with wasting of the subcutaneous tissues and flabbiness of the muscles. The abdomen is distended and tympanitic. The temperature is often slightly elevated at night. As the disease progresses, certain nervous symptoms may become manifest. Chief of these is weakness of the lower extremities, which may be accompanied by loss of the knee-jerks. Attacks of tetany are not uncommon, and there may even be epileptiform convulsions.

Edema, especially of the extremities, may occur, without any evidence of renal disease, just as it does in other forms of chronic gastro-intestinal disorder in childhood. The mental condition is apt to be peculiar, there being often a high degree of precocity along with a tendency for the child to be unduly interested in his own symptoms, which is most unusual at this age. Sometimes, indeed, they are actual little hypochondriacs, and almost always they are spoilt.

PATHOLOGY.—On post-mortem examination nothing at all is found on inspection except some atrophy of the intestinal wall. As to this all observers are agreed. In the absence of definite anatomical findings, various theories have been put forward as to the mode of production of the symptoms. It has been supposed (1) that there may be defective activity of the pancreas, or (2) of the liver, or (3) that the condition is really one of intestinal catarrh set up, according to Heber, by an abnormal intestinal flora.

Examination of the stools might be expected to furnish evidence as to the validity of these different views. The chief abnormality found in these is the presence of a large excess of fat or its derivatives, and it is to this mainly that their pallor is due. Unaltered fat is usually present in excess, but fatty acids and soaps predominate to an even greater extent. The presence of an excess of split fats is opposed to the theory that there is any defect of pancreatic secretion, and in two of the writer's cases—one with well-marked infantilism—in which the pancreatic reaction was tested for in the urine, the result was negative. It would seem, also, that there is never a complete absence of bile pigment from the stools in these cases, although the perchloride of mercury (for stercobilin) reaction is often much fainter than normal. It is therefore difficult to say positively whether there is any defect of bile secretion, but there is certainly no other evidence of hepatic insufficiency, the urea secretion, for instance, attaining the normal level.

Microscopic examination of the stools often reveals the presence of unaltered starch, which might be regarded as a point in favour of defective pancreatic secretion; but, on the other hand, this might also be expected to occur where the intestinal contents are being passed on too rapidly into the colon. If this takes place, there would also be less time for the absorption of fats, and therefore, on the whole, the character of the stools might be explained by the mere presence of a catarrh limited to the small intestine. This is the theory of Heber, but he goes farther, and ascribes the catarrh to the presence in the bowel of an abnormal bacterial flora. He has pointed out that the Gram-positive organisms are greatly in excess in the stools in these cases, instead of the Gram-negative predominating, as in the normal. He has further isolated certain specific organisms—e.g., the *Bacillus bifidus* and the *B. infantilis*—which he believes may play a specific part in exciting the catarrh.

and in the production of toxins to which some of the nervous symptoms associated with the disease—e.g., tetany—may be due.

In two of the writer's cases, in which a careful bacteriological examination of the stools was made by Panton, it was found, in confirmation of Herrer's observations, that the Gram-positive bacilli were present in relative greater proportion than control, but no bifid or other unusual organisms were found in either case. Whether there is really a specific infection present in these cases or not, therefore, must still be regarded as doubtful, nor is there any need to invoke such an infection to explain the nervous symptoms, for, as is well known, tetany may occur in any case in which there is excessive intestinal decomposition. Langmaid, for instance, has described a series of such cases in association with dilatation of the colon.

The infantilism which occurs as a result of prolonged colic disease is easily accounted for by the interference with nutrition which the defective absorption of food constituents induces, for not only are fats and starches badly absorbed, but there is also a large wastage of proteins by putrefaction, as well as a loss of calcium salts in the form of unabsorbed soaps. Hence both the muscles and skeleton are starved. It seems probable that the infantilism of colic disease is closely related to the parietic form of infantilism described by Byrom Bramwell, in which the impairment of growth is brought about in a very similar way.

DIAGNOSIS.—The condition most apt to simulate colic disease is abdominal tuberculosis, and it is often extremely difficult to distinguish between them. In colic disease, however, there are no enlarged glands to be felt, and no thickened and rolled-up omentum, nor is there any ascites. A negative von Pirquet's test is also of value in the diagnosis. The distinction is rendered still more difficult by reason of the fact that abdominal tuberculosis seems sometimes to supervene upon colic disease after the latter has lasted some time.

PROGNOSIS AND COURSE.—It has been already pointed out that the course of the disease is an extremely chronic one, the duration extending to years, with intervals of improvement, followed by the most disheartening relapses, which may take place in spite of every care. Complete and permanent recovery, however, may certainly occur. It took place in at least six out of sixteen cases which have been under the writer's observation, and in some of these the disease had lasted for two or three years. If a fatal result ensues, it is usually the consequence of some intercurrent condition, such as broncho-pneumonia.

When the disease has lasted some time, it leads to a retardation of growth, which was noted by Gee in his original paper, and which may leave the child frail and stunted even when the intestinal symptoms have disappeared. In well-marked cases the interference with growth may amount to a condition of true infantilism, such as Herrer described. The way in which this arrest of development is brought about has already been discussed.

TREATMENT.—A suitable diet is of the first importance. Owing to the impaired absorption of fats, the amount of fatty food must be greatly curtailed, and as starches are also badly borne, these, too, must be avoided, and the diet should consist of raw or underdone meat, raw-meat juice, skim milk (preferably peptonised), gelatine in the form of jellies of different sorts, and dextrinised foods, such as Mellin's food biscuits, breakfast biscuits, grape nuts, and malted milk. A good malted bread—e.g., Bernaline—may also be given in moderation. Whey and

cocoatina (made with water) may be used as beverages. The raw meat and meat-juice should form the basis of the dietary, and a child of four may take as much as 6 ounces of the former and 10 ounces of the latter daily. As improvement sets in, a little stale white bread, boiled rice, or Benger's food, may be added to the diet, as these contain starch in a form in which it is most easily borne.

Under this regimen the stools usually lessen in number and amount, and improve in colour, whilst abdominal distension is lessened.

In the general hygienic treatment great care should be taken to avoid chill. A warm binder should be worn over the abdomen, and the legs, which are very apt to be cold, should be warmly covered. It is sometimes even necessary to wrap them in cotton-wool and bandages. Special precautions should also be taken to avoid chill in bathing the child. Change of air to a bracing seaside place is often of great help, especially when the appetite is defective, and will often start an improvement when the case is "hanging fire."

Medicinal treatment is of secondary importance, but opium is always useful. It may be given in doses of 1 minim of the tincture for every year of the child's age, along with bitters and carminatives. It tends greatly to lessen the offensiveness of the stools.

If diarrhoea be severe, nitrate of silver is the most potent astringent. It may be given in doses of $\frac{1}{2}$ grain, with a drop or two of dilute nitric acid in a little glycerine and distilled water three times daily, and may be continued for weeks if necessary. The writer has never known it produce argyria, but if such a result be feared, the administration of the drug may be suspended for a week or two from time to time. Active preparations of pancreas are also of value, especially where the stools contain much unaltered fat. Parkeson sugar tablets (two after each meal) and Heladin (one capsule three times daily) are good forms in which to give it.

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COLITIS.

Colitis may occur as part of a general intestinal infection or as a local disease confined to the colon. Pathologically one can recognize both catarrhal and ulcerative lesions. In the catarrhal cases there is swelling and injection of the mucosa of the colon and lower end of the ileum, often with small hæmorrhagic areas in places, and enlargement of the solitary glands. In the most intense forms of catarrhal inflammation ("membranous" or "pseudo-membranous") a false membrane of fibrinous exudate (often only to be recognized on microscopical examination) may form on the surface of the mucosa in places. The whole wall of the colon in these cases is greatly thickened, and the surface of the mucous membrane where there is no exudation is much injected.

Ulceration may take the form of either (1) superficial ulceration of the mucous membrane, usually confined to the colon, the ulcers sometimes coalescing to form large areas denuded of epithelium; or (2) there may be follicular ulceration in which the process is confined to the solitary glands, which become excavated, forming

small deep ulcers which look punched out, and may reach down to the muscular coat. In extreme cases the whole surface of the colon may be honeycombed with such ulcers.

The associated lesions of colitis are the same as those of infective diarrhoea, broncho-pneumonia being most frequent.

ÆTIOLOGY.—The predisposing causes are identical with those which have already been described in the section on Infective Diarrhoea. The bacteriology of the disease is still under discussion, but there is some reason to believe that the bacillus of dysentery is most commonly the exciting agent.

Clinically the symptoms of colitis are the same as those of any other severe diarrhoea, the chief distinguishing character being the passage of viscid mucus and blood. It will be sufficient for descriptive purposes to divide cases into two groups—acute and chronic—for the pathological lesions described above have no constant relation to the clinical features. Mucous-membranous colitis will be referred to separately.

Acute Colitis.—The onset is usually abrupt, and often attended by vomiting. Diarrhoea appears early. The stools at first are loose, and yellowish or greenish in colour, but very soon mucus and blood make their appearance. The motions then become very frequent, although often small in amount. Their passage is accompanied by much straining, which often leads to prolapse of part of the rectal mucous membrane. The constitutional symptoms vary in severity in different cases. When the colitis is primary, they are often comparatively slight; but where it forms part of a general intestinal infection, they are similar to those met with in infective diarrhoea (q.v.), and may equal them in severity. The temperature is usually elevated at the onset, but subsides after a few days. In the mildest cases the symptoms last about a week, and the inflammation may then pass off entirely or settle into the chronic condition described later. In the severer cases, and especially in those of the pseudo-membranous form, the constitutional symptoms are more pronounced, and there may be marked signs of collapse from the outset, with high-continuous pyrexia. Such cases are apt to prove fatal, especially in young or ill-nourished infants. Between these extremes all intermediate varieties are met with.

DIAGNOSIS.—Acute colitis, especially when primary, is apt to be mistaken for acute intussusception. The presence of fever, the absence of the characteristic sausage-shaped tumour in the abdomen, and the negative result of a rectal examination, are the points in favour of colitis.

PROGNOSIS.—The prognosis is much better in primary colitis than in secondary cases, and in older children than in young infants. In any given case the severity of the constitutional symptoms is the best index to the chance of recovery.

TREATMENT.—The general hygienic and dietetic treatment is the same as in cases of infective diarrhoea.

At the outset it is well to clear out the bowel with a dose of castor-oil, but after that has acted opium is the most useful drug. One minim of the tincture may be given for every year of the child's age along with a small dose of castor-oil—e.g.:

Tinct. opii	℥j.
Ol. ricini	℥v.
Glycerini	℥ss.
Aq. menth. pip.	℥i ℥ss.

For a child of one year.

This may be repeated every three hours at first, or *often* if the stools be very frequent. Later on bismuth in large doses—*e.g.*, 10 grains—should be substituted for the castor-oil in the above mixture. If constitutional depression be pronounced, stimulants may be required, but the indications for their use and the mode of employing them have already been described in the section on Infective Diarrhea.

Chronic Colitis.—Chronic colitis may follow upon an acute primary attack, or it may arise insidiously out of a simple dyspeptic diarrhoea. The mildest cases can only be distinguished from simple diarrhoea by the presence of quantities of visible mucus in the motions. Blood is usually absent, and tenesmus and prolapse are less common than in the acute stage. In the severer cases constitutional symptoms, such as wasting and collapse, are more pronounced; and if the disease be protracted, the patient may pass into a miserable and cachectic state, with sunken eyes and fontanelle, a distended abdomen, through which coils of inflated intestine are often visible, and a loose dry skin, sometimes showing a petechial rash in places. Not uncommonly there is some oedema of the extremities, but without any evidence of renal disease. The temperature is generally subnormal or shows only occasional elevations.

The course of the disease is protracted, often lasting several months, and relapses are extremely frequent. Death usually results from anæmia or from some intercurrent disease, such as tuberculosis or broncho-pneumonia.

DIAGNOSIS.—In severe and prolonged cases of chronic colitis general tuberculosis is apt to be simulated, and it may, indeed, be impossible to distinguish between them. In colitis, however, the intestinal symptoms precede the wasting; in tuberculosis the reverse is the case, and the cachexia is out of proportion to the diarrhoea. Fever is the rule in tuberculosis, but the exception in chronic colitis.

PROGNOSIS.—The prognosis chiefly depends upon the age and general condition of the child, and upon the character of the surroundings. The longer the disease has lasted, the less likely is recovery to take place; and if there be no improvement after three months, the case will probably prove fatal.

TREATMENT.—The hygienic and dietetic treatment are of chief importance. The general lines of the former are fully described in the section on Colic Disease (p. 169), but emphasis must be laid on the great benefit often derived from change of air to the seaside or to a bracing mountainous locality.

The diet must be supervised most strictly, and should consist of fully peptonized milk, raw-meat juice, raw white of egg, and malted foods. Alcoholic stimulants are often of service.

Drugs are of secondary importance, but small doses of opium along with castor-oil or bismuth are helpful from time to time.

Irrigation of the colon through a long rectal tube or catheter passed as high up as possible is often very useful. One may use either normal saline solution or some astringent, of which the best is $\frac{1}{2}$ per cent. protargol or argyrol. When the latter are used, the colon should first be washed out with a large quantity of plain water, and then 4 or 5 ounces of the astringent solution injected and left in as long as it can be retained.

Muco-Membranous Colitis.—This variety of colitis, which is not to be confused with the intense catarrhal form designated pseudo-membranous, to which reference has been made above, is occasionally met with in childhood, but as it differs in no

important respect from the same disease which is so well known in the adult, a brief description will suffice.

The chief symptoms are abdominal pain, constipation, and the passage of mucus and membranous shreds or "casts." The nervous symptoms, which are usually so prominent in the adult, are less conspicuous in childhood, whilst the constitutional symptoms are the same as those of chronic indigestion. Infants are very rarely attacked, most of the patients being children at or beyond the period of the second dentition. The disease is rarely primary, there being usually a history of a previous attack of acute intestinal catarrh.

The diagnosis can only be made by the discovery of the characteristic shreds of membrane in the stools.

The course of the disease is protracted, and relapses are common, but ultimate recovery usually takes place.

The treatment is the same as that of mucus-membranous colitis in the adult.

INTESTINAL OBSTRUCTION.

Intestinal obstruction in children may be congenital or acquired. The chief causes of congenital obstruction are hernia of the umbilical cord, with fixation of the intestine to the sac, or occlusion at the opening in the abdominal wall; imperfect development of the diaphragm, with prolapse of the intestines into the cavity of the thorax, and slipping of the gut at the point of passage from abdomen to thorax (Hirschsprung); partial or complete occlusion of various portions of the alimentary tract, due to arrested development, occurring more especially in the region of the rectum and anal canal, but sometimes involving large tracts of the small intestine from the duodenum downwards; and internal hernias, with the intestine entangled in an abnormal opening in the mesentery or mesentery, or in one of the colic peritoneal fossae. Cooley records a fatal case of hernia strangulated in the intersigmoid fossa in a new-born infant, discovered at the autopsy three days after birth.

Acquired obstruction is most frequently associated with the presence of a hernia with irreducible contents—the hernia may be of the external or internal variety—an intussusception, a persistent Meckel's diverticulum, compression by a tumour or constriction by a band, volvulus, and the impaction of a foreign body in the lumen of the intestine. It may also be a sequel of septic peritonitis, on account of the paralysis of the musculature of the intestinal wall produced by that condition. Occasionally it has been caused by the inclusion of a coil of intestine in the ligature of the umbilical cord after birth.

SYMPTOMS.—The early signs of intestinal obstruction, both congenital and acquired, are intermittent pains in the abdomen, associated with crying and drawing up of the legs, which cease as the spasm subsides; nausea passing on to vomiting, which progressively increases in frequency and intensity with the duration of the trouble; alteration in the character of the vomited material from gastric contents to bilious and then to stercoral matter; an absence of the passage of flatus or feces per rectum, although soon after the onset of the attack some mucus may be passed, emptying the bowel below the site of obstruction; distension of the abdomen, becoming more marked as the duration of the attack increases; slight

acceleration of pulse-rate, tending to increase in the later stages of the illness; and shrunken tissues from loss of fluid, and cold extremities. If unrelieved at this stage, the attack may end fatally from exhaustion and collapse; or, from increasing injury to the intestinal wall, with consequent permeability to septic contents, signs of general peritonitis may be added. As a rule the temperature then rises, the acceleration of the pulse-rate is great, the abdomen is "ballooned" and quite immobile, whilst the character of the pain alters from an intermittent "colicky" nature to a constant laminating type, with tenderness and involuntary rigidity all over the abdominal wall.

The character of the signs of obstruction may be modified, or additional signs may be present with the various causes producing the obstruction, so that the cause may in some cases be identified before operation is performed. This, however, is not material. It is essential that the condition of intestinal obstruction be recognized; the cause can be dealt with at the time of operation. These variations in signs may briefly be summarized as follows:

Hernia.—When the contents of a hernial sac become irreducible, increased in size, painful, tender, and when nausea and vomiting ensue, obstruction is present. In children the strangulation of a hernia is rare, while obstruction is common. Obstruction to the passage of intestinal contents through the lumen of the gut in all cases is at first purely mechanical. Later interference with the circulation in the gut-wall ensues, and paralytic obstruction is then added to the previous mechanical obstruction. *Taxis to reduce the contents of the sac should never be attempted.* The condition of the gut-wall cannot be foretold by the clinical signs, and irreparable damage may be done. The cæcum and the appendix are so frequently present in large right-sided inguinal hernias in children that differentiation between appendicitis in the sac and obstruction to the contents is quite impossible. To attempt to reduce by manipulation an inflamed appendix would be disastrous. I have met with this condition of acute appendicitis in a hernial sac in an infant seven months in age. The condition was only recognized at the operation.

With slight obstruction, reflex vomiting may be so profound in children as to produce a fatal result before strangulation has commenced. Persistent vomiting is an indication for immediate operation. Vomiting, on the other hand, is absent from severe cases of a very serious nature, and this may be due to the fact that the gut-wall has quite early become so seriously damaged that it has given way, and a relief of tension ensued; this soon manifests itself by early signs of peritonitis or cellulitis. Whenever inflammation of the surface tissues in the region of an irreducible hernia is present, immediate operation is called for. The gravity of this sign cannot be over-estimated. The surface tissues have become infected by the septic material that has traversed the gut-wall, which has been rendered permeable by the obstruction and interference with its blood-supply. In a child eleven months old, whose right inguinal hernia had been irreducible for eighteen hours, and in whom no vomiting had occurred before admission to hospital, cellulitis of the tissues of the scrotum and inguinal region were present. Immediate operation showed that a gangrenous loop of small intestine formed the contents of the sac.

The immediate treatment of an irreducible hernia is to give the child a hot bath, then put it to bed with the pelvis and legs raised, when it is an inguinal or femoral case, so that gravity tends to enable the contents of the sac to return to the abdominal cavity, and to keep an ice-bag over the site of the hernia. Unless

vomiting supervenes and persists, this treatment may be maintained for many hours, and it hardly ever fails to effect reduction. Subsequently a radical cure should be performed.

It may be difficult to differentiate between an irreducible hernia and an imperfectly descended testicle with torsion of the cord, or an ovary prolapsed into the inguinal canal with torsion of the pedicle. The absence of a previous history of hernia, together with the absence of a testicle from the scrotum, are signs in favour of the torsion of the spermatic cord, and although vomiting may be occurring, yet the bowels will be acting normally. Addison has called attention to the value of an examination *per rectum* where strangulation of a prolapsed ovary is suspected, when it may be possible to determine that the uterus is fixed or drawn over to the affected side. But whenever any doubt as to the diagnosis exists, immediate operation must be performed with a view to saving the testicle or ovary. The following case shows the insidiousness of the condition, and the rapidity with which the destruction of the testicle may occur. An infant was brought on account of a lump in the groin, which the mother had noticed for two days. It appeared to be slightly tender when touched, but otherwise there was no pain, no vomiting, and the child seemed to be in excellent health. The lump was fixed in the upper part of the inguinal canal, the outline was indelphinable on account of the exudation into the surrounding tissues, and the testicle was absent from the scrotum on that side. A tentative diagnosis of torsion of the cord was made, and operation was done a few hours later. The testicle was found to be completely gangrenous, necessitating excision. Strangulation in an internal hernia can only be recognized at the time of operation. One case of strangulated hernia within the para-diaphragmatic fossa in a girl of seven years of age was under my care. The hernia was reduced by operation, but the patient died of shock.

Intussusception.—Invagination of one portion of the gut into another produces the condition called "intussusception," and may occur at any part of the alimentary tract from the jejunum to the rectum, but is most common in the ileo-caecal region. A tumour is present, elastic, and situated most frequently somewhere along the course of the large intestine. Unless a tumour can be felt in a case in which intestinal obstruction is suspected, the diagnosis of intussusception cannot be sustained. If necessary, it must be sought for under an anæsthetic, since a complete examination of the hepatic and splenic flexures of the colon cannot be made unless the abdominal walls are flaccid. *Per rectum*, the apex of the intussusception may sometimes be felt, and in cases with a long mesentery to the large intestine the intussusception may have prolapsed so far as to be hanging between the knees of the patient. I have seen this in three cases, and in two of them a cystic tumour of the wall of the small intestine was present at the ileo-caecal angle. To distinguish this condition from a case of simple prolapse of the rectum, it is only necessary to pass the finger along the exposed mucous surface of the prolapse up to the anal margin. In a simple prolapse the finger passes continuously from the mucous membrane to the epithelial surface of the anus. In a prolapsed intussusception the finger passes up into a groove between the anal margin and the mucous surface, and this groove extends for some distance up into the rectum (Fig. 17).

Following upon the interference with the blood-supply of the gut-wall, mucus and blood make their appearance, and are passed at frequent intervals *per rectum*. Vomiting occurs, and the earlier and more frequent its onset, the higher up in the

alimentary tract is the situation of the intussusception. Thus in an exterior intussusception, as a rule, profuse vomiting sets in almost immediately, while the passage of blood and mucus occurs late. In this type, too, the long axis of the tumour tends to lie obliquely athwart the abdominal cavity.

Conditions most likely to be mistaken for intussusception on account of the association of the passages of blood and mucus with abdominal pain are tuberculosis of the caecum, acute entero-colitis, and Henock's purpura.

In tuberculosis of the caecum the tumour is fixed, does not vary in size, the discharge of blood is profuse, unlike the small quantities seen in cases of intussusception, and vomiting as a rule is absent. In acute entero-colitis profuse diarrhoea generally precedes the appearance of blood in the motion by a considerable space of time, there is no tumour, and signs of obstruction, such as the swelling of the abdomen and rigidity of the musculature, are absent. Henock's purpura, with

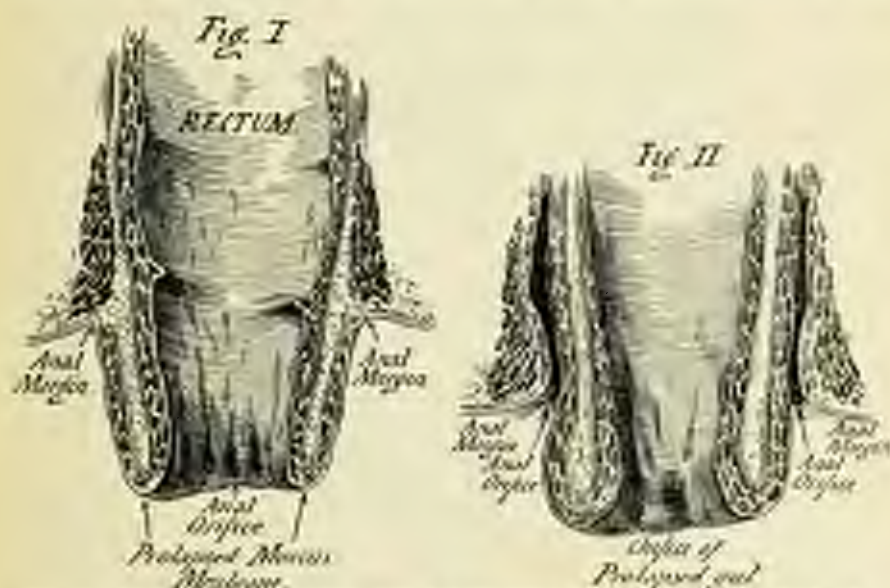


FIG. 12.—DIAGRAMS ILLUSTRATING THE RELATIONSHIP OF THE MUCOUS MEMBRANE (I.) A PROLAPSED RECTUM; (II.) A PROLAPSED INTUSSUSCEPTION, RESPECTIVELY, TO THE ANAL MARGIN.

hemorrhages into the wall of the intestinal tract, may be exceedingly difficult to distinguish from an intussusception. Purpura elsewhere, joint pains, continuous admixture of fecal material with the blood passed *per rectum*, and the absence of a tumour, are the main points in the differentiation of the condition. But, as Sutherland has pointed out, a patch of oedema or hemorrhage into the intestinal wall may well produce paralytic obstruction or become the starting-point of an intussusception, and therefore in cases of grave doubt a laparotomy should be performed. More than one intussusception may be present at the same time. In a case successfully operated upon by Steward, an exterior intussusception was found and reduced on first opening the abdomen. The condition of the wall of the intestine did not seem sufficient to account for the gravity of the condition

of the patient. A further search revealed an ileo-colic intussusception, which was then successfully reduced.

In older children, recurrent attacks of intussusception may occur. Abdominal pain is complained of in the region of the right iliac fossa, a lump is present which varies in size at frequent intervals, and eventually disappears entirely as the attack subsides. The attack may occur again in a few days' time. Excessive mobility of the rectum on account of an abnormally long mesentery to the large intestine is generally found to be present in these cases, and an operation for fixation of the rectum will prevent recurrence of the attacks. If the whole of the large intestine retains its primitive mesocolon, the apex of an intussusception may travel as far as the rectum without damage to the circulation of the gut-wall, and consequently without the onset of paralytic intestinal obstruction. Reflex vomiting, however, may be so severe as to threaten the life of the patient. In three cases with this persistence of a primitive mesentery severe pain referred to the end of the penis occurred at each spasm. This appeared to be so characteristic that in the third case I diagnosed the presence of a primitive mesentery to the large gut before operation, and confirmed it at the subsequent laparotomy. In this type of case alone does reduction of the intussusception by irrigation or insufflation appear to be justifiable, the reason being that the long mesentery of the large intestine can stretch sufficiently during the travelling of the apex of the intussusception so that no interference with the blood-supply of the gut-wall occurs, and consequently there is no damage to the coats of the intestine. Otherwise, the only treatment for intussusception is operation. The death-rate rapidly rises with delay in the performance of operation after the onset of the attack. Since the condition of the gut-wall cannot be estimated by clinical evidence beforehand, except in very rare instances, many deaths have been recorded from rupture of the intestine following attempts to reduce it by rectal irrigation. A further objection to this method is that it is impossible to determine when the intussusception has been completely reduced. Subsequent laparotomy has had to be performed in a number of cases, with the disadvantage to the patient of an added period of delay.

Meckel's Diverticulum may persist as a complete tube, forming a channel between the small intestine close to the termination of the superior mesenteric artery and the umbilicus, or it may be closed externally, so that its presence is not discovered until some pathological condition has necessitated the opening of the abdominal cavity. Between complete persistence and complete occlusion all possible varieties of partial obliteration have been recorded (Fig. 13). When it is patent at its umbilical attachment, escape of intestinal contents is sufficient to indicate the nature of the condition. But with a patent umbilical attachment the part of the duct attached to the intestine may be represented only by a solid coil. A macroscopical examination of the mucous membrane lining the external pouch will reveal its nature by showing the typical structure of lining membrane of the alimentary tract. Whenever the persistence of the duct can be recognized beforehand, it should be ablated down to its intestinal attachment, with closure of the intestinal wall, so soon as the general health of the infant permits. Apart from the unhealthiness of a discharging umbilical wound, it is so frequent a cause of intestinal obstruction that the simple operation for its removal should always be performed. Obstruction may be produced by the diverticulum acting as a fixed point, around which rotation of the intestine may occur, or as a band underneath which a coil of intestine may be tipped, or from inflammatory changes

occurring in its own structure perforations with septic peritonitis may ensue. A rare condition which has been recorded is an intussusception starting at its enteric attachment, with prolapse through the umbilical opening. When intestinal obstruction has followed on the persistence of the diverticulum, the cause may be suspected from the absence of signs peculiar to the other causes of intestinal obstruction. As a rule, however, the diagnosis cannot be made until a laparotomy has been performed.

Tumours and Adhesions are not common causes of intestinal obstruction in children. Mesenteric cysts, omental cysts, retroperitoneal sarcomata, and cysts of the intestinal wall, are the most frequently occurring tumours that may lead to this condition. Bands and adhesions are generally the sequelae of repeated slight

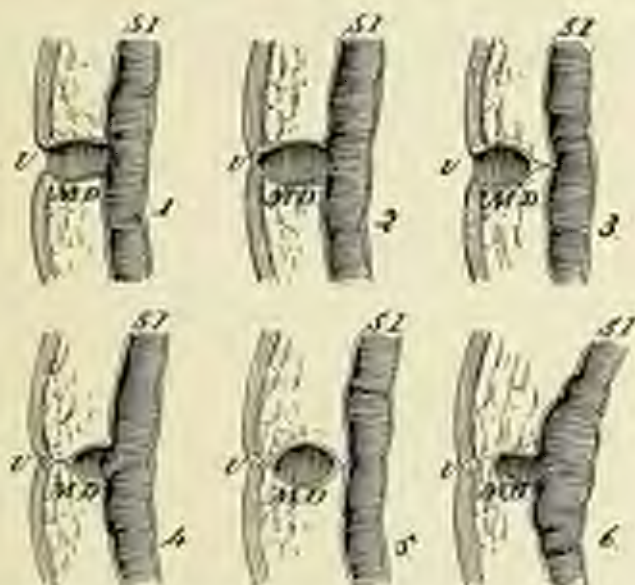


FIG. 12.—DIAGRAMS ILLUSTRATING TYPES OF MECKEL'S DIVERTICULUM.

U., Umbilicus; S.I., small intestine; M.D., Meckel's Diverticulum.

attacks of appendicitis, of inflammation of a persistent Meckel's diverticulum, and of abdominal tuberculosis. After an operation in the abdomen of a child for the relief of an inflammatory condition, the omentum as a rule becomes rapidly adherent to the immediate site of the operation. By acting as a constriction to some underlying portion of intestine, occlusion may occur, with the subsequent onset of obstruction. In a case under my care, in which an intussusception had been reduced by laparotomy, acute obstruction supervened three days later. This was cured by releasing the omentum that was found to be adherent to the site of the previous operation-wound, bending down the terminal portion of the ileum.

In abdominal tuberculosis the matting of the intestines may be so intimate that any attempt to relieve the condition by detaching the coils from one another is hopeless. To attempt to do so would probably result only in the rupture of some

vibrated portion of the gut-wall, and the consequent production of septic peritonitis. The mere performance of a laparotomy, with closure of the abdomen without attempting to deal with the causes of obstruction, is often beneficial in these cases.

The Impaction of a Foreign Body in the lumen of the intestine as a cause of obstruction may be suspected where there is a previous history of one having been swallowed. Sometimes it may be detected by a screen examination with X-rays. Fish may perforate the intestinal wall, leading to peritonitis, with subsequent paralytic obstruction of the intestine. In a case successfully operated upon for acute obstruction by Kellock a silver anchor, a plum-stone, some cherry-stones, and dead leaves, were removed from the lumen of the small intestine of a boy aged eight years.

DIFFERENTIAL DIAGNOSIS.—Very few conditions in children closely simulate that of intestinal obstruction. The late stages of cyclic vomiting, vomiting of cerebral origin, sometimes as a sequel of nephritis, and paralysis of the intestines associated with hæmorrhage into the suprarenal bodies, present some difficulties in distinction. In the individual sections conditions leading to intestinal obstruction, and requiring operative treatment, have been already indicated.

In the late stages of "cyclic vomiting" the abdomen may be motionless during respiration, and slightly rigid all over, whilst there is an absence of the passage of feces and flatus. Dulness on percussion, shifting from flank to flank with change of position, is often present, and helps to render the diagnosis obscure. Careful analysis of the history of the case will reveal that in the early stages of the attack the bowels acted normally. They have ceased to act, because there is nothing to pass. The immobility and slight rigidity of the abdominal muscles are due to exhaustion from repeated vomiting, and as a rule, after rubbing and kneading them with the hand, the patient can be induced to move them quite freely. The shifting dulness, with the absence of a fluid thrill, is caused by the empty collapsed coils of intestine flopping from side to side as the patient is turned over. This total emptiness of the intestinal tract also accounts for the absence of the passage of feces and flatus. Combined abdominal and rectal examination reveal nothing else abnormal, while the smell of acetone in the breath and its detection in the urine help to establish the diagnosis. In vomiting of cerebral origin *verruca* examination of the nervous system and of the urine is generally sufficient to explain the condition. Abdominal examination yields a negative result, but the retention of the musculature so frequently present in meningitis must not be misinterpreted.

Congenital stenosis of the pylorus can hardly give rise to confusion, on account of the characteristic association of the vomiting with the ingestion of food, the dilatation of the stomach with its wave of visible peristalsis, and the frequent presence of a palpable tumour in the situation of the pylorus.

TREATMENT.—The treatment of intestinal obstruction is immediate operation. The pre-operative treatment is to avoid the use of purges or enemata, from which nothing but harm can arise. Early diagnosis of the condition is of the utmost importance, and when this has been made a hypodermic injection of a small dose of morphia— $\frac{1}{4}$ to $\frac{1}{2}$ grain—combined with $\frac{1}{100}$ grain of atropin, is of great value. The patient is relieved of distress, shock is minimized, and the subsequent amount of anæsthetic necessary for the operation is reduced in quantity. Spinal anæsthesia

induced by Tyrrell Gray's method should be used by preference in all cases of intestinal obstruction. It produces complete relaxation and immobility both of the abdominal wall and intestinal tract, so that protrusion of intestines does not occur. Manipulation of the viscera can be freely carried out, since the peripheral impulses arising from that procedure cannot pass the block induced in the spinal cord by the local anæsthetic, and therefore general shock is prevented. Under these conditions the writer successfully performed a lateral anastomosis after resection of the intestine in a case of acute obstruction occurring in an infant twenty-four hours old. This is the only recorded instance of survival after so severe an operation among infants.

The details of the operative procedure will vary with the conditions found on opening the abdomen and with the individuality of the surgeon. But the problem of what to do with extensive areas of gangrenous intestine in children should rarely be presented to him for solution, since it indicates as a rule failure to recognize the seriousness of the condition in the early stages of the illness.

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APPENDICITIS.

Appendicitis in children differs from appendicitis in adults in two main respects. Its presence is more easily recognized, since children but rarely suffer from inflammatory or ulcerative lesions of the stomach, duodenum, biliary apparatus, pancreas, and Fallopian tubes—conditions that in the adult may be indistinguishable from an attack of or a sequela of appendicitis until a laparotomy has been performed. Secondly, the disease is far more fatal, owing to the slight resistance children are capable of offering to an acute bacterial invasion of their tissues.

The appendix is a tapering cul-de-sac opening out of that part of the alimentary tract called the caecum. It receives its main blood-supply by a branch of the posterior ileo-caecal artery, which reaches the base of the appendix by passing behind the terminal portion of the ileum. In many instances this appendicular artery, which runs in the free border of the meso-appendix, terminates at some distance from the free end of the appendix, so that the apex has to rely almost entirely for its blood-supply upon a few small anastomotic twigs. The veins pursue a similar course, and are equally exposed to the chance of occlusion by the pressure of an overlying viscus. It has been shown experimentally that partial occlusion of the venous system of the appendix inducing a passive hyperæmia is associated with a rapid intensification of the virulence of the bacteria present within its lumen. The same result follows from the blocking of the lumen of the canal—so that a stagnation of its normal contents ensues—by a stercolith or foreign body, or, as Still has shown, by thread-worms. The position of the appendix within the abdominal cavity is variable. The surgical significance of this variability is the

consequent liability of the appendix to be occluded by pressure or kinked by traction or adhesions. Lastly, Peyton and Paine have shown that invasion of the walls of the appendix may occur by bacteria which have been introduced experimentally into the circulation, resulting in intense tissue-destruction of its various coats.

To enumerate, then, these features of the appendix—that it is a hollow, blind diverticulum, with a narrow lumen opening out of a septic cavity; that its variable position and its accessibility to semi-solid material render easy the partial occlusion of its canal; that its blood-supply, often barely efficient towards its apical portion, can readily be interfered with by slight external pressure or traction; that any stagnation of its contents intensifies rapidly the virulence of its bacterial contents; and that its walls may be invaded by bacteria circulating in the blood of the patient—makes one wonder, not so much that the appendix is so often diseased, as that it ever escapes. No one factor can be held responsible for all cases of appendicitis, and to seek a common explanation in the dangerous properties of red rubber stoppers in lemonade bottles, or to weave a fanciful analogy with the faucial tonsil can only arise from an imperfect appreciation of the significance of the above-mentioned facts. A truer analogy may be found with the other normal and abnormal hollow blind diverticula from the alimentary tract—i.e. the gall-bladder and bile-ducts, the pancreatic duct, a persistent pharyngeal pouch, and a Meckel's diverticulum. These share with the appendix an extreme liability to disease of bacterial origin.

PATHOLOGY.—The commonest infective agents present in cases of appendicitis are the *B. coli communis* and streptococci, more rarely staphylococci and pneumococci, whilst instances of invasion by the bacillus of enteric fever, the tubercle bacillus, and actinomycotic fungi, have also been recorded. Occasionally both simple and malignant neoplasms have been found. After the removal of an appendix, in which induration is present, and the local signs of inflammation are not marked, it is of the utmost importance that a microscopical investigation of sections should be made. Sarcoma and carcinoma have thus been discovered in children in whom the previous attacks of appendicitis had been clinically indistinguishable from a simple inflammatory invasion. Sarcoma has been recorded in a boy of three years of age, and carcinoma in a boy of five years. If "appendicitis" literally means inflammation of the appendix, confined to that structure only, then such a condition cannot be truly diagnosed, but can only be surmised from an experience of the commonest cause of a characteristic association of a certain group of signs and symptoms. The structure of a healthy appendix cannot be determined by palpation of the abdomen, and therefore alteration of its structure due to disease cannot be estimated. The function of the appendix is unknown; therefore, the impairment of function by disease cannot be appreciated. To locate accurately the site of a lesion in any part of the body, evidence of alteration of structure or of impairment of function, or of both, must be forthcoming. If the peritoneal coat of the appendix has become involved, the disease has ceased to be appendicitis, and has become a localized peritonitis, giving rise to the characteristic signs and symptoms of that trouble. For the peritoneal covering of the appendix is no more an intrinsic part of the structure of the appendix than is the peritoneal covering of any other viscus in the abdomen. But wherever inflammation of the peritoneum is present, certain signs and symptoms peculiar to that condition make their appearance, and from their localization in some particular portion

of the abdominal cavity we may hazard a conclusion as to what underlying viscus is involved. Signs of peritonitis beginning in the right iliac fossa suggest strongly that the starting-point of the lesion is the appendix. But a diseased Meckel's diverticulum, a Fallopiian tube, a breaking-down gland in the ileo-caecal angle, or a primary perimesocolic, rheumatic, or tuberculous peritonitis starting in that region, may give rise to a similar grouping of signs and symptoms, so that a precise diagnosis cannot be arrived at until a laparotomy has been performed.

Since absorption of septic material from an inflamed appendix readily occurs via the lymphatics and blood-stream, producing a varying degree of toxæmia, another group of signs and symptoms will be associated with appendicitis. These manifest themselves as variations of the pulse-rate, temperature, and leucocyte content of the blood-stream, occasionally with rigors, attacks of convulsions, and modifications of the mental faculties. Upon these signs an estimate of the virulence of the lesion may be based, but not an estimate of the site of the lesion. The essential difference between these two problems cannot be too sharply emphasized if we wish to appreciate the significance of the wide variation of the signs and symptoms so commonly associated with appendicitis.

Lastly, the distension of a viscus by its own secretion or by a mildly inflammatory exudation is generally associated with an intermittent "colicky" pain characteristically referred to some definite area, associated with nausea and vomiting, and unaccompanied by any febrile reaction. This is well seen in the case of the kidney with an obstruction to the lumen of the ureter from a kink, a twist, or an impacted calculus; in the gall-bladder, with the cystic or common duct obstructed; in commencing intestinal obstruction; and in distension of a Meckel's diverticulum. Probably distension of the appendix gives rise to a similar train of events. An intermittent colicky pain, referred generally to the umbilicus, frequently to the gall-bladder region, more rarely to the gastric region or left hypochondrium, and sometimes to the left of the umbilicus in the left iliac fossa, associated with nausea and subsequently with vomiting, and unaccompanied by any marked modification of the pulse-rate or temperature, is very commonly produced by a dilated appendix. In operating upon cases presenting these clinical features, the writer has been much struck by the enormous distension of the appendix with an opalescent viscid fluid, and with the absence of any signs of inflammation of its peritoneal coat. The lumen has nearly always been obstructed close to its opening into the caecum by a stercolith or a stricture behind which the contents of the appendix have accumulated, but their virulence has not yet become sufficiently intense to attack the structure of the appendix. Mansell Moullin has suggested as an explanation of these signs that excessive peristaltic efforts on the part of the appendix to get rid of its contents transmits the tug by means of adhesions to various areas of the splanchnic plexus. This cannot account for those cases in which no adhesions are present—a condition frequent in children; but the variability of the distribution of the referred pain may perhaps be partially accounted for by the presence of adhesions, partially by the variable situation of the appendix.

Microscopical examination of appendices after removal has shown that all degrees of inflammatory change may be present, from a simple leucocytic invasion of its mucous and submucous coats to a complete gangrene involving the whole structure. At one end of the scale is the dilated appendix, containing a more or less clear viscid fluid, at the other the appendix issuing pus through perforations where localized sloughs have separated, associated with inevitable involvement of

the peritoneal cavity. The train of events may be a direct sequence from the former to the latter, but not necessarily. The dilated appendix may discharge its contents through its natural opening into the cæcum, when the attack subsides. Or the bacterial invasion of the structure may be so virulent from the outset, without any preliminary dilatation, that signs of involvement of the peritoneum manifest themselves within a few hours of the beginning of the attack. The extent to which the general peritoneal cavity is involved will be indicated by a diffusion of signs of inflammation over the abdomen, and the evidence of a high grade of toxæmia. But even though the clinical evidence indicates the implication of only a minute area, if the signs of toxæmia are postponed early in the course of the disease, the tissue destruction of the appendix may be found to be rapid and complete on account of the virulence of the infective agent.

DIAGNOSIS.—After these preliminary observations, an attempt may be made to correlate the clinical manifestations of appendicular disease with the underlying pathological changes:

1. *Simple Appendicitis*, in which the inflammation is limited to the appendix proper, and its peritoneal covering is not involved.

Pain of an intermittent "colicky" nature is experienced, as a rule first intensely in the right iliac fossa, and referred thence to the umbilicus. Slight deep-seated tenderness and fulness in the region of the appendix may be found, but these are not marked features of this stage of the attack. There is no tenderness, rigidity or immobility of the abdominal muscles. Nausea ensues, and subsequently vomiting. The pulse-rate and temperature hardly vary from the normal. After a period of twenty-four to forty-eight hours the attack subsides, or else passes on to the stage in which signs of the involvement of the peritoneal coat of the appendix begin to manifest themselves. During the attack, or subsequently, one or two offensive motions may be passed containing a little mucus, affording evidence of excessive intestinal decomposition. Occasionally frequency of micturition, with pain referred to the end of the penis on completion of the act, is present. This condition is very commonly found in children suffering from slight recurrent attacks of appendicular trouble.

2. *Appendicitis with Involvement of the Peritoneal Coat.*—This may follow on the previous condition, if a natural relief to the accumulated contents of the appendix has not occurred. When this happens, a relief of tension may be brought about in the distended appendix by the spread of the inflammation through its coats, with a consequent subsidence of the signs pointing to distension. Or the inflammatory reaction may be so severe from the outset of the attack that within a very few hours' time all the coats of the appendix, including its peritoneal covering, have become inflamed.

The intermittent colicky pain, referred to various sites, is then replaced by a constant sharp pain localized to the site of the appendix, generally in the right iliac fossa. There is great tenderness over this area, and involuntary rigidity of the overlying muscles is now present. At the same time signs of commencing toxæmia begin to manifest themselves. The pulse-rate rises rapidly, the temperature is generally raised, but may be subnormal, and an examination of the blood at this stage will reveal as a rule an increased leucocytosis, with an excess of polymorphonuclear cells. The sequence of the signs and symptoms in this order is one of the most characteristic features of an attack of appendicitis. Vari-

tions in their intensity are due to variations in the virulence of the inflammatory process.

If surgical relief has not been afforded to the patient at this stage, one of two things generally occurs. The inflammation of the peritoneum may become localized, with the formation of pus, which will be recognized by formation of a mass in the affected region. Or if the peritoneum is not capable of limiting the spread of the inflammation, general peritonitis, with its characteristic signs, ensues. Quite rarely an attack may subside spontaneously. But there can no longer be any justification, in the face of accumulated evidence and experience, in delaying surgical procedure in the hope of such an event. There are no signs in the mode of onset of any attack of appendicitis that point to the probability of an attack subsiding spontaneously, while the death-rate doubles with every twenty-four hours' delay in adopting surgical treatment after the first forty hours of illness. Quite rarely, within a few hours of the onset there is a catastrophic involvement of the whole peritoneal area. But to watch an attack until a localized abscess can be diagnosed by fluctuation or until general peritonitis has ensued is deliberately to risk the life of the patient.

3. Gangrenous Appendicitis.—In cases of the type to which reference has already been made, in which all the coats of the appendix are destroyed and gangrenous within a few hours of the onset, a suggestive group of signs and symptoms present themselves. The pain in the right side is generally sharp, brief, and followed shortly by a period of quiescence. Palpation reveals only a slight amount of localized deep-seated tenderness, while the general appearance of well-being of the patient is illusive. But the pulse-rate is markedly accelerated, often being as high as 120 to 150 within a few hours after the pain has been first experienced. The temperature as a rule is elevated to 102° to 103° F. Variations of temperature, however, are less constant and less reliable as evidence of a serious toxæmia than the pulse-rate. Changes in the blood-count also can generally be found. The acceleration of the pulse-rate is of the gravest significance, and indicates that from a small localized lesion septic absorption of virulent toxins is proceeding. In this group of cases I have most commonly found that the appendix is retro-colic in position, running up to the right kidney, often as high as the place where it is crossed by the transverse mesocolon. The gangrene as a rule involves its distal portion, and a commencing oedema of the retroperitoneal cellular tissue in that region is frequently present. The protected situation of the appendix accounts for the absence of signs of peritonitis, such as tenderness and rigidity of the abdominal muscles, and, indeed, inflammation of the peritoneum, except of the peritoneal coat of the appendix, is not present. But that most dangerous of all complications—a cellulitis of the retroperitoneal cellular tissue—has frequently already been initiated, and in part accounts for the evidence of general toxæmia observed so early in the course of the disease.

DIFFERENTIAL DIAGNOSIS.—1. Intermittent "colicky" pains in the abdomen, associated with nausea, and subsequently with vomiting, and with an absence of febrile reaction, may be caused by many intra-abdominal conditions in children; but a close analysis reveals the fact that the distension of a viscus is in nearly all instances the underlying cause. Intestinal obstruction invariably has this mode of onset, no matter what condition has brought it about. The subsequent course of events will make the diagnosis quite clear. The cause of the obstruction may not be determined until a laparotomy has been performed; but it is immaterial,

as immediate surgical interference is the only treatment of such a condition, from whatever cause it may have arisen. Distension of the bladder, so frequently associated with stenosis of the meatus urinarius, but occasionally brought about by impacted foreign bodies, produces a similar train of symptoms, although vomiting is rare. An examination of the abdomen and the meatus urinarius immediately reveals the cause of the condition. Obstruction to the outflow of the secretion of the kidney, or occlusion of its blood-supply by torsion of the pedicle, often a far more difficult problem in diagnosis. The site and distribution of the pain in the attack may be characteristically referred to the penis, scrotum, and inner side of the thighs; but this may be difficult to determine with precision in a child. Tenderness and swelling of a kidney may be present. As the attack passes off, a large quantity of urine may be passed, and, on microscopical examination, blood-cells be discovered. In this group of cases it is of the utmost importance that a complete quantitative and qualitative examination of the urine should be made, a cystoscopic examination of the bladder performed with a view to detecting asymmetry of the ureters and the stream of fluid exuding from them, and at least two radiographic views of the whole of the urinary tract on both sides obtained. By these means, out of the last twelve patients who were admitted to hospital under my care with the provisional diagnosis of "chronic appendicular trouble," I have been able to prove that two were suffering from stones in the right kidney, three from a stone in the right ureter, and one from distension of the bladder, due to stenosis of the meatus urinarius. All have been entirely cured of their troubles by operation. Abdominal tuberculosis, with extensive enlargement of the mesenteric glands, may often have this mode of onset, but the mass formation and its wide distribution throughout the abdomen generally makes the diagnosis clear. Later on in the course of the disease, obstruction due to the presence of bands and adhesions may occur, but at this stage, as a rule, there can be no doubt about the diagnosis. Hirschsprung's disease, or congenital dilatation of the colon, whilst producing the same symptoms, is easily recognizable by the enormous size of the large gut, with its visible waves of peristalsis. Constipation of a simple functional nature in a child seldom produces pain, nausea, or vomiting. Fecal impaction is exceedingly rare, and if it occurs in an infant functional or structural stenosis of the sphincter ani should be sought for, and treated by dilatation. In acute enterocolitis with diffuse intermittent abdominal pain, the early profuse diarrhea establishes the diagnosis. Intermittent pain may be referred to one or both sides of the abdomen with early disease of the vertebral column. The fact that pain is only produced by movement of the spine indicates the necessity for a careful examination of the vertebral column. In all cases of intermittent abdominal pain the spine must be examined as a routine. To diagnose "biliousness" in a child who is suffering from abdominal pain, nausea, and vomiting, without an examination of the abdomen, is to court disaster. When an examination is made as a routine procedure, the diagnosis of "biliousness" will probably cease to exist.

2. Constant pain in the right iliac fossa, tenderness corresponding with the area of the pain, involuntary muscular rigidity, an accelerated pulse-rate, and an abnormal temperature, are pathognomonic of peritonitis in that region; but the appendix is not necessarily the starting-point. Tuberculous peritonitis, with the whole of the peritoneal cavity studded by miliary tubercles, may appear to commence there, and may only be revealed at the laparotomy. This condition is generally greatly benefited by mere abdominal section. Its presence may be suspected

beforehand where the clinical signs of involvement of an extensive area of the peritoneum are present, and yet there is little alteration of the pulse-rate, temperature, and blood-count. Pneumococcal and rheumatic peritonitis as a rule cannot be diagnosed beforehand, unless the patient has suffered for some time from known pneumococcal or rheumatic lesion. Laparotomy is the best treatment for these conditions. Peritonitis may also follow upon unrecognized intestinal obstruction, but, with the advance of diagnosis, this condition is becoming fortunately rare. A perforated Meckel's diverticulum, infection through the stump of a septic umbilical cord, and a leaking pyosalpinx, are other starting-points of peritonitis that may be suspected beforehand, but only proven by means of a laparotomy. In a child aged eighteen months, with signs of general peritonitis of the lower half of the abdomen, after evacuating about $1\frac{1}{2}$ pints of pus, a leaking pyosalpinx was found on the left side; this was removed, the pelvis drained, and the patient made an uninterrupted recovery. She had suffered from persistent vaginal discharge for some months previously, and on account of this history the underlying cause was suspected before operation.

In abdominal tuberculosis, with breaking-down glands and a consequent involvement of their overlying peritoneal coats, this group of signs and symptoms may be produced. The size of the mass, and the absence of signs of toxæmia, which are almost necessarily present where such an extensive lesion has been produced by appendicular trouble, are valuable signs indicating the type of lesion present; but the condition requires operation for the removal or evacuation of the glands, so as to prevent dissemination of their contents throughout the abdomen.

Extrapertoneal lesions, such as abscesses arising from osteitis of the pelvic girdle or in the glands along the external iliac vessels, may present great difficulties in diagnosis. When there is any doubt in such a case, an extraperitoneal operation, such as that used for the approach to a psoas abscess, must be performed. In one such instance an abscess in the glands situated along the external iliac artery was opened and drained successfully. In another case, in which the presence of a fluctuating mass along the outer half of Poupart's ligament suggested the possibility of such a condition, approach by that route revealed a healthy extraperitoneal space. The peritoneum was therefore opened through the same incision, 2 ounces of offensive pus were evacuated, and a gangrenous appendix, which was adherent by its apex to Poupart's ligament, removed. The patient made an uninterrupted recovery.

Rectal examination may be of some value in a suspected case of intussusception or of a stone impacted in the pelvic portion of the ureter.

The type of case in which it is most difficult to arrive at a correct diagnosis is that in which pain, experienced in the right iliac fossa, is not due to a lesion situated there, but is conducted or referred from a lesion elsewhere. Such lesions are, early pneumonia, with involvement of the lower thoracic nerves in a pleuritic inflammation, acute anterior poliomyelitis of the lower thoracic region, and herpes zoster of the same spinal segments. In the first-named trouble the fact may generally be elicited, after winning the confidence of the child, that tenderness does not coincide with the site of abdominal pain, and that therefore the lesion cannot be situated there. This is a sign of great value. Secondly, when the patient has been convinced that pressure on the abdomen will not aggravate the pain, the spasm of the abdominal muscles relaxes, showing that it is a voluntary and not an involuntary rigidity. Thirdly, pressure behind over the yielding costal margin of

a child will often elicit great tenderness and aggravation of the conducted pain; whilst of supreme importance is the fact that the pulse-respiration ratio is disturbed, so that a marked acceleration of the respiration-rate out of all proportion to the pulse-rate and temperature is observed. The diagnosis is quite clear if abnormal signs in the chest can be detected; but, unfortunately, in the early stages these are seldom present. In poliomyelitis and herpes zoster, extreme pain in the region of the spine is generally experienced, in addition to the pain in the ill-region, cutaneous hyperæsthesia is often present, and there is a striking absence of involuntary muscular rigidity and of deep-seated tenderness.

TREATMENT.—Kühnelt at the German Surgical Congress, 1910, recorded his operative experience in cases of appendicitis. In 374 cases operated on within forty hours of the onset, 12 were fatal, a mortality of 3 per cent. In 262 cases in which abscess formation had occurred, 39 were fatal, a mortality of 14.8 per cent. In 296 cases in which diffuse peritonitis was present, 98 were fatal, a mortality of 48 per cent.

Schäufeler's results, from the operative treatment of 937 acute cases, are as follows:

Within forty hours of the onset, 444 cases operated on, death-rate 6.5 per cent.; later than forty hours after the onset, 281 cases operated on, death-rate 21 per cent.

C. A. McWilliams records the results of 687 operations in the acute stage. Within a short time of the onset the mortality in 264 cases was 1.6 per cent. In the cases operated upon later the mortality rises to 42 per cent., and for children under ten years is 50 per cent.

Deaver records 165 cases operated on within forty hours of the onset, with a death-rate 1.9 per cent. Murphy, recording the early operative mortality of himself, Deaver, Ochsner, and the Mayo brothers, in several thousand cases, estimates it at 2.5 per cent. He also quotes Krimmson and Guimbellot, recording 35 cases of appendicitis with abscess formation in children under two years of age; only 7 recovered.

In 79 cases under my care in children under twelve years of age, all of whom were operated upon, there were 9 deaths. An abscess was present in all the fatal cases, and surgical advice had not been sought for any of them until at least four days had elapsed from the onset of the illness. In three of these cases general peritonitis was present at the time of operation.

A consideration of these figures alone is sufficient to indicate the early treatment of appendicitis; Operate immediately, and operate speedily. Deaver states that "the mortality remains more than it should be for four causes: (1) Failure to diagnose the disease sufficiently early; (2) failure to recognize the gravity; (3) postponement of prompt surgical intervention; (4) incorrect treatment in the later stages of the disease." Pre-operative and post-operative treatment also play important rôles in bringing about a successful issue. The administration of purgatives and enemata, in the first few hours of the attack, before the diagnosis has been established, cannot be too strongly condemned. In the conditions likely to be mistaken for appendicitis their value is small; whilst if appendicitis is present, their action in increasing peristalsis not only tends to spread the infection to other parts of the peritoneal cavity, but also so promotes the absorption of material by the peritoneal lymphatics that the paralysis of the intestine is rapidly increased, and the general powers of resistance of the patient by auto-inoculation progressively depressed. Deaver states: "Purging does no

good, but positive harm." Th. Kocher writes that "the castor-oil treatment does much harm." Kistner states that "castor-oil should be abandoned in the treatment." Kinsell expresses himself "in accord with these views." The experience of the writer is entirely in agreement with these opinions. To obtain rest for an inflamed area is the aim of all surgical procedure, and in no part of the body is it so urgently needed as in the abdominal cavity. The administration of purgatives in such cases is a violation of that principle, and will make its influence distinctly felt over a large series of cases. Distension and paralysis of the intestine are frequent, and urgent symptoms arise in those cases in which purgatives have been early administered. A gratifying freedom from such complications is the reward of abstinence from this procedure. From the onset of the attack until operation can be performed, the patient should be propped up in the upright (Powell) position, hot fomentations applied to the painful area, $\frac{5}{16}$ to $\frac{1}{16}$ grain morphia with $\frac{1}{16}$ grain atropin given hypodermically, a complete abstinence from all food by mouth, including fluids, insisted upon, and warm continuous saline solution run into the rectum. In this way thirst is quenched, the strength of the patient maintained, his general condition improved by the relief of pain, and cessation of all peristalsis—since this is initiated by any ingestion of stomach contents into the alimentary tract—insures rest to the peritoneum. The details of the operative procedure will vary with the surgeon; but I strongly advocate the routine removal of the appendix in all stages except in those in which the unfortunate patient has been allowed to become profoundly toxic from the presence of a large abscess or general peritonitis; then simple rapid drainage must suffice, unless the appendix obviously presents in the line of incision. In the majority of cases in which the appendix is not obviously seen, it is embedded behind the ascending colon, and with this knowledge it can be expeditiously removed without excessive manipulation or prolongation of the operation. The choice of anæsthetic in children is a matter of importance. A general anæsthetic may be given in the early stage operation if the constitutional disturbance is slight, but open ether, for choice, after the immediate induction of unconsciousness by chloroform. On the other hand, if the pulse-rate is early markedly accelerated, or when a late stage operation with an abscess present has to be performed, I unhesitatingly use spinal anæsthesia, induced after the well-known method of Tyrrell Gray. By this method all shock is avoided, complete flaccidity of the abdominal muscles and intestines produced, the after-risk of a fatal acetonaemia, so frequently associated with the administration of chloroform to children, is minimized, and the patient invariably leaves the operating-table with his general condition in no way impaired as the immediate sequel of the operation. On the completion of the operation the patient is put to bed in the Fowler position, and the continuous administration per rectum of warm normal saline solution begins. This may be maintained for a week if necessary as the only source of alimentation; but the absence of readily assimilable carbohydrate from a child's dietary has been shown by Fox to result in the appearance of acetonaemia in 100 per cent. of all children between the ages of two and five years, irrespective of any disease from which they happen to be suffering. I therefore have the saline for rectal feeding prepared so that it contains 20 per cent. of glucose, and even then, should acetonaemia be found to be present subsequently, any quantity of a 2 per cent. solution of glucose, from $\frac{1}{2}$ to $1\frac{1}{2}$ pints, must be immediately run into a vein or subcutaneously, and if necessary this must be augmented by the administration

by mouth of ounce doses every hour of a concentrated solution of glucose. At the end of every twenty-four hours the rectum must be washed out with warm boracic lotion, introduced through a channelled catheter to overcome the slightly-irritant action of the glucose solution. Small doses of morphia and atropia are given hypodermically to relieve pain and to insure sleep. By rigid adherence to this procedure as a routine I have never yet seen post-operative distention of the intestines occur so as to be a source of anxiety. The greatest accumulation of flatus that occurs is readily removed by the passage of a long rectal tube, and this is only necessary on rare occasions. Vomiting is best treated by gastric lavage. No food should be given by mouth until the pulse-rate is approximately normal. The bowels should be left to act spontaneously, even if this means waiting for a week. The injudiciousness of any attempt to make them act artificially is well emphasized by the temporary rise of the temperature that is associated with it. A. E. Barker pointed this out twenty years ago, and my experience is fully in accord with the valuable principles in the after-treatment of abdominal surgical cases which he laid down so long ago. When the appendix has not been removed, a second operation for its removal should always be performed, as soon as possible after the wound of the first operation has healed. This necessity of a second operation should become increasingly rare. It only arises because of the dangerous delay in seeking surgical relief in the first attack, or on account of lack of skill or experience on the part of the operator in finding a hidden appendix when the illness has only been in progress for a few hours.

COMPLICATIONS.—A fecal fistula but rarely follows in children, and only occurs when the operation is done late in an acute attack or when the appendix has not been removed. Similarly, subphrenic, hepatic, and pleural abscesses may develop, but as a rule they are the direct outcome of delay in surgical interference. Bacilluria, with the presence of *B. coli* communis in the urine, is often present, but disappears as healing ensues. Urethra may be given. I have met with no instances of intestinal obstruction or general peritonitis following on the operation in the acute stage, although they have been recorded. Bronchitis or pneumonia occasionally supervene. The most dreaded after-complication is the onset of acetonaemia, but the adoption of the measure for forestalling and treating it, already alluded to, has so far been rewarded with a complete freedom from it. The signs of its onset are drowsiness, slight cyanosis, large, sluggish pupils, a dangerously accelerated pulse-rate, vomiting (often only very slight), and the presence of acetone in the urine, as revealed by Rother's method of testing. At the same time there is an absence of all signs of spreading inflammation from the operation area, and post-mortem examinations on recorded fatal cases have shown the correctness of the clinical diagnosis.

ANOMALIES.—The appendix may be present in the sac of an inguinal or femoral hernia on either side, and may there undergo inflammation. Such an attack can hardly be differentiated from strangulation of the hernial contents, and is a reason for early operation in such cases. It is sometimes present on the left side of the abdomen, but in that situation it will manifest the same signs of disorder although with a different distribution of the characteristic pain and tenderness. Rarely the appendix is the starting-point of an intussusception. A neglected abscess may track upwards and point in the loin as a periophrictic abscess, or may pass

downwards and inwards into the pelvis, forming a pararectal abscess, or, passing downwards and outwards from the right iliac fossa, may escape beneath Poupert's ligament, and eventually invade the hip-joint.

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DISEASES OF THE STOMACH.

Congenital Pyloric Stenosis.—**SYNONYMS**.—Congenital gastric spasm; Congenital spastic hypertrophy of the pylorus, etc.

The first description of a case of this disease was published by H Ezekiah Beardsley in the year 1788. In the middle of last century cases were described by Williamson and by Dawson, but it was not until the year 1888 that a paper by Hirschsprung first attracted any serious attention to it. Even after that date some time elapsed before the disease attained any wide recognition, but a large number of cases have now been described in the English-speaking countries and in Germany, although in France and in the Latin countries it appears to be either not so often met with or not so well recognised.

ÆTIOLOGY.—But little is known of the ætiology of the affection. It occurs much more frequently in boys than in girls, 80 per cent. of the cases being in the male sex. First-born children also are relatively much more often affected. Thus, out of nineteen cases recorded by Davidson, no fewer than ten were in first children. It is rare to find more than one case occurring in the same family. The way in which the child is fed appears to have no influence in determining the onset of the disease; indeed, in a majority of the cases the child has been at the breast till the first symptoms appeared. Social surroundings also appear to have nothing to do with the ætiology, the affection being quite as common in private as in hospital practice.

SYMPTOMS.—The symptoms are few and suggestive, the most prominent being persistent vomiting. It should be specially noted that the vomiting rarely dates from birth, but usually makes its appearance between the second and fourth weeks. It is often what is called "projectile" in character, and waste material is apt to be brought up than was swallowed at the last meal. It usually comes on soon after a feed, the vomited material consisting of curdled milk, mixed sooner or later with mucus, and occasionally with streaks of blood. There is one curious fact about the

vomiting—namely, its tendency to cease for some hours with any change of food and to recur again as violently as ever.

At the outset, at least, the vomiting is accompanied by constipation, which helps to distinguish it from dyspeptic vomiting, in which diarrhea is usually present. The constipation is sometimes spoken of as a "pseudo constipation," inasmuch as it is not due to any disorder of the intestine, but simply to a failure of the food to find its way into the bowel at all.

Meanwhile the child rapidly loses weight, and soon becomes extremely emaciated. The wasting, however, is not like that met with in cachexia. The child is not



FIG. 14.



FIG. 15.

FIGS. 14 AND 15.—PHOTOGRAPHS OF TWO INFANTS, SHOWING WELL-MARKED PROTRUSION OF THE STOMACH IN CONGENITAL PYLOPIC STENOSIS.

anæmic, but simply looks starved, and often remains surprisingly bright in spite of it. In this respect it differs from the wasting of most forms of organic disease, and to the experienced eye the difference often suggests a correct diagnosis.

The PHYSICAL SIGNS are few, but sufficiently characteristic. In the first place the stomach is dilated, and may cause a swelling in the upper part of the abdomen, which is all the more striking in contrast with the attenuation of the rest of the

trunk. On light percussion it can often be found that the greater curvature reaches down to, or even much below, the level of the umbilicus. Most characteristic of all is the appearance of waves of visible peristalsis (Figs. 14 and 15). These are best seen shortly after a feed has been given, and if they are lazy in making their appearance, they can often be called forth by a little gentle massage of the upper abdomen. The waves of contraction begin at the left side, where a rounded swelling about the size of a golf-ball appears below the left costal margin, and travels somewhat leisurely across the abdomen in an oblique direction, and downwards and to the right. Before one wave has disappeared another makes its appearance, so that an hourglass-shaped swelling can be seen reaching across the abdomen. Sometimes three waves are present simultaneously.

This vigorous peristalsis is pathognomonic in the child, as in the adult, of some obstruction at the outlet of the stomach, and is therefore of great diagnostic value.

Curiously enough, it does not seem to cause much, if any, pain, and the child will often lie quite placidly whilst the waves course in succession across the organ.

The third sign is the presence of a pyloric tumour. It should be felt for to the right of the middle line, and one should try to get underneath the stomach and compress the pylorus against the right side of the vertebral column. As the late George Carpenter pointed out, it is often best felt by trying to pinch up the deeper structures. Sometimes the pylorus is so tucked up under the liver that no tumour can be felt, but if one is successful it feels like a hard lump almost the size of a hazelnut, but somewhat elongated.

Headlin has pointed out that there is often a pronounced distasis of the recti muscles in these cases, but this is no doubt due simply to the distension of the upper abdomen, and is of no special value in diagnosis.

The combination of symptoms and signs, then, on which one relies for establishing the existence of congenital pyloric stenosis is—(1) Vomiting of the character already mentioned coming on within the first few weeks of life, and accompanied by "pseudo-constipation," and wasting; (2) the presence of visible gastric peristalsis; (3) the existence of a pyloric tumour, if it can be felt.

PATOLOGY.—1. MORBID ANATOMY.—The stomach, in early life, exhibits very clearly the division of the pyloric portion into the two segments known as the "pyloric vestibule" and the "pyloric canal." The latter is particularly well marked, forming a tube about 3 inches long, which begins at the *sulkus intermedius*, and ends at the pyloro-duodenal junction. If one examines the stomach from a case of congenital pyloric stenosis after death, one is at once struck by the enormous



FIG. 14.—STOMACH WITH CONGENITAL PYLORIC STENOSIS. FROM AN INFANT AGED SIX WEEKS.

The stomach capacity was $\frac{1}{2}$ ounces; the normal capacity is about 2 ounces. (From St. Bartholomew's Hospital Museum.)

development of the whole pyloric canal, which forms a dense, almost cartilaginous tube, the extremity of which projects into the duodenum in much the same way, it has been remarked, as the cervix uteri projects into the vagina. In addition, the wall of the pyloric vestibule is also thickened, and, indeed, that of the whole stomach to some extent, including even in some cases the lower end of the oesophagus.

It will be observed, too, that the mucous membrane of the pyloric canal is thrown into longitudinal folds, the ends of which often project into the vestibule.

On microscopic examination, it is found that the thickening of the wall of the pyloric canal is due almost entirely to an enormous development of the circular muscular fibres, although the longitudinal layer is also thickened, and the mucous and submucous coats to some extent. More careful examination shows that the muscle fibres are not only increased in number, but that each individual fibre is also broader than normal (Davidson).

2. *Pathogeny*.—So far all observers are agreed, but it is when one passes on to inquire how these changes are brought about that differences of opinion begin. Two distinct views are held:

(1) That the thickening of the pyloric canal is to be regarded as a congenital malformation.

(2) That it is due to overwork, the consequence of a long-continued spastic contraction.

We shall consider immediately which of these views best fits in with the clinical history and symptoms of the disease, but meanwhile we may examine them in greater detail.

(1) A study of the "architecture" of the thickened muscle layer of the pyloric canal shows that it is certainly not to be regarded as a tumour formation (Wernstedt), but rather as a hyperplasia. As Cantley has put it: "Nature in her extreme anxiety to provide an efficient pyloric sphincter has overexerted herself, and produced too great a quantity of muscular tissue." Granted that this were so, it is difficult to see why obstruction should result, for the mere thickness of the muscular coat should not prevent its relaxing to allow the escape of the stomach contents when required. It has also been pointed out as an *a priori* objection to this hypothesis that infants who suffer from congenital pyloric stenosis have no special tendency to exhibit malformations elsewhere, although it is well known that, as a rule, a malformation in one part of the body is apt to be accompanied by congenital abnormalities in others.

(2) The supporters of the spasm hypothesis have got to show—(a) How spasm may be set up; (b) that it is capable of leading to consecutive hypertrophy.

(a) As regards the cause of the spasm, it has been suggested that it is due to an inco-ordination in the pyloric mechanism—a sort of "stammer" of the pylorus—beginning, perhaps, in intra-uterine life (Thomson), whereby, when the waves of peristalsis reach it, instead of opening, as it should do, it remains closed. It has also been attributed (Shattock) to a hyperæsthesia of the mucous membrane in the neighbourhood of the pylorus, which induces reflex contraction—an "hysterical" pylorus, as it has been called. A third group of writers would attribute the spasm to a disorder of the chemical mechanism of the pylorus. They have tried to show that there is hyperacidity of the gastric contents in these cases, and that this hyperacidity induces the spasm (Engel, Pfamöller). This hypothesis, however, will certainly not hold water, for (1.) it has been shown that there is no real hyper-

acidity of the gastric juice in these cases at all (Müller and Wilcox). (ii.) When hyperacidity exists it is almost certainly the result of the pyloric obstruction, and not the cause of it (Ibrahim, Feer), being comparable to the hyperacidity found in cases of benign stenosis of the pylorus in adults, which is now admitted to be due to the continuous stimulation of the gastric mucous membrane by the retained contents. (iii.) In any case experimental evidence (Cannon) goes to show that acid on the gastric side of the pylorus induces relaxation of the pyloric sphincter, and not contraction of it.

(b) The assumption that prolonged spasm of the pyloric canal can lead to hypertrophy of its muscular coat also involves certain difficulties. It would be contrary to pathological law that this should happen, unless the muscle is not merely contracted, but contracted *spasmodically*, so that its fibres are kept in a state of "extension" at the same time. If it be admitted, however, that the pyloric canal in infancy is a regulating mechanism which controls the passage of the gastric contents from the stomach to the duodenum, this difficulty disappears, for in that case it would be comparable to the arterioles which regulate the flow of blood from the arteries to the capillaries; and it is admitted that prolonged contraction of the arterioles can lead to hypertrophy of their muscular coat.

A greater difficulty is to explain the rapid development of the hypertrophy by this hypothesis. Operation within as short a time as three weeks from the first appearance of symptoms has shown the hypertrophy to be already present in a marked degree, and it is difficult to conceive how "overwork" of the muscle could have led to so great an overgrowth of it so quickly.

It will be observed that there are great difficulties in the way of accepting either the congenital hyperplasia or the spasm hypothesis, whilst the compromise suggested by some—that there is both hyperplasia and spasm—has the difficulties of both combined.

Putting aside these *prima facie* objections, let us now inquire which hypothesis best fits the clinical facts.

We have already seen that there is some difficulty in understanding how a mere hyperplasia of the sphincter can produce obstruction at all; but, waiving this objection, any acceptable interpretation of the pathological appearances must explain these two important facts in the clinical history of the disease:

1. That the symptoms are not truly congenital, but often only come on some weeks after birth.
2. That the condition is capable of being recovered from without operation.

Now, it is obvious that the spasm hypothesis fits in best with both of these facts. Granted that spasm can be brought about *somehow*, it is really easier to understand its supervention some time after birth than its occurrence congenitally or *in utero*, whilst there is no reason why, when it passes off, a return to a normal state should not be possible. On the other hand, no plausible reason has been advanced for the delay in the appearance of the symptoms on the assumption that the obstruction is due to a congenital abnormality of structure, nor can we understand how, if that were the true pathology, cures without operation would be possible.

The *a priori* difficulties, therefore, are all in the way of spasm; the clinical difficulties, in that of hyperplasia.

Diagnosis.—Congenital pyloric stenosis is distinguished from ordinary indigestion in infancy by the projectile character of the vomiting and by the presence of visible gastric peristalsis. The co-existence of constipation is also strongly in

favour of pyloric stenosis, most cases of dyspepsia in babies being accompanied by diarrhoea. The sex of the child must also be taken into account, and one should always be reluctant to diagnose pyloric stenosis in a girl. Congenital stricture of the duodenum gives exactly the same symptoms and signs as a case of pyloric stenosis, except that no tumour can be felt. The distinction of the two conditions, however, may be impossible during life.

PROGNOSIS AND COURSE.—It is difficult to arrive at an accurate estimate of the mortality in pyloric stenosis, as the statistics given by different writers vary considerably, and seem to depend to a large extent on the method of treatment adopted. The writer has treated twenty consecutive cases in private practice by medical means, with seventeen recoveries. This is perhaps an exceptionally fortunate experience, but others have obtained even better results. Thus, Heibner records nineteen recoveries out of twenty-one cases treated medically, and Starck eleven out of twelve; whilst of six cases reported by Bloch, and not operated upon, all did well; and Benda has had no fewer than thirty recoveries out of thirty-two treated by medical measures alone.

The results of cases treated in hospital, however, are, in this country at least, much less favourable. At Great Ormond Street the mortality has been 78 per cent. in a series of sixty-four cases all treated medically. One may contrast with these figures the results yielded by operative treatment. Of these, the most favourable, so far as the writer knows, are those of Burghard, who had eleven recoveries out of sixteen cases, a mortality of 31 per cent. Cautley reports six recoveries out of seven cases operated upon in private, but a mortality of four out of five hospital cases. Of twenty-three cases under the care of Thomson, which were treated by operation (most of them by Stiles), fifteen died, a mortality of 65 per cent. We shall probably not be far from the truth if we accept Brakin's estimate of 50 per cent. as the probable mortality amongst all cases submitted to operation. It must be admitted that such statistics do not do justice to the surgeon, for no doubt in many of the cases operation was only performed as a last resort, and when the chances of recovery in any event were but small, but they are the only material available for forming a judgment. In the great majority of cases treated successfully by medical means, the recovery appears to be complete and permanent, and no impairment of the general health or gastric functions remains. In exceptional cases, however, some degree of dilatation of the stomach may persist.

The usual course of the disease under medical treatment is that the vomiting ceases quite soon, but in spite of this the infant does not usually gain weight; indeed, it may even go on losing, and there may be little or no progress for weeks, sometimes not until the fifth month. With perseverance, however, improvement finally sets in, and often quite suddenly the child starts to gain weight. This sudden change for the better is often very striking, and has also been remarked by Heibner.

Sometimes, on the other hand, and all too often in hospital cases, the issue is not so happy, and, in spite of the cessation of vomiting and the resumption of a fair amount of food, the weight refuses to rise, and the child eventually succumbs, apparently as the result of an "alimentary toxæmia," which seems to be the consequence of an atrophy of the bowel, with impairment of the power of absorption, probably brought about by the long period of virtual starvation through which

the infant has passed. In other cases, again, one is disappointed by a sudden and inexplicable collapse just when all seemed to be going well.

TREATMENT.—So soon as the diagnosis of pyloric stenosis has been made, the question as to whether the case is to be treated medically or by operation will arise. This is an extremely difficult question to answer, and opinion upon it is sharply divided. The writer's own view is that in the great majority of cases operation is not required. Others, however, hold that if, after three, or at most six, weeks of medical treatment, no decided improvement has occurred, operation should be performed without further delay. The type of case with which one has to deal may also afford some indications as to the line to be adopted. Thus, if the symptoms have set in very early and with great severity, operation is more likely to be necessary than in one in which the symptoms are milder and later in appearing. There is no case, however, in which it can certainly be predicted that its recovery without operation is impossible.

1. Medical Treatment.—The essentials are careful feeding and daily lavage of the stomach. As regards the kind of food, breast milk is probably the most suitable, but, owing to the frequent intervals at which food must be given, suckling is impracticable. The breast milk may be drawn off and given by a spoon, but usually this practice results in a speedy cessation of the appearance of milk, and has to be abandoned. In that case, or if, as often happens, the child has already been weaned before the diagnosis has been made, thoroughly peptonized and diluted cow's milk is the best substitute; but dried milk, such as Glaxo, or mixtures of whey, egg-albumin, and lactose, supplemented occasionally by somatose, may also be used. The marked fat retention which has been found to take place where there is a block at the pylorus has induced some to exclude all fat as much as possible from the food. It has not been found, however, that this is of any great benefit in practice, and if the stomach is being regularly washed out it is probably unnecessary.

One must feed one's way as to the amount to be given at a time, beginning, perhaps, with an ounce every hour, and gradually increasing the quantity and intervals as the vomiting subsides. This plan is much better than feeding at long intervals, as some advise. After the feed the child should be kept lying flat, and not moved about, in order to minimize the chance of vomiting. The stomach should be regularly washed out once or twice a day, according to the frequency of the vomiting, with a solution of bicarbonate of soda (1 drachm to the pint). That it is possible to treat cases successfully without this, the results obtained by Heubner, who does not employ it, clearly show; but it certainly adds to the comfort of the patient, and, by lessening the frequency of the vomiting, diminishes exhaustion and facilitates sleep. By removing retention products and mucus it also lessens the catarrh and swelling of the mucous membrane, and may in this way favour the escape of the contents through the narrowed pylorus.

The technique of gastric lavage in infancy is described elsewhere. As subsidiary measures, the German plan of applying continuous pressure to the epigastrium is of value, and great care should be taken, as in all underfed babies, to maintain the body heat.

If vomiting be severe, and the amount of food which passes out of the stomach very small, fluid should be supplied *per rectum* in the form of emulsions of normal saline, 2 to 3 ounces being given at a time twice or three daily. These are of great value in supplying a sufficiency of fluid to the tissues, and in promoting elimination of waste products by the kidneys.

Drugs are of little help, but minute doses of opium (say $\frac{1}{16}$ grain) at the bedtime before each feed) are strongly recommended by some. The bowels may be kept open by the use of small enemas or soap suppositories, or by the administration of phenolphthalein ($\frac{1}{4}$ grain) by the mouth.

2. *Surgical Treatment.*—If operation be decided upon, the choice lies between pyloroplasty and posterior gastroenterostomy, but the relative merits of these procedures cannot be discussed here.

REFERENCES.

The complete literature down to 1900 will be found in the monograph of Fiedet and Guillemet presented to the Sixth Congress of Gynecology, Obstetrics, and Pediatrics, held at Toulouse in September, 1909. Toulouse: Imprimerie et Librairie Edouard Privat, Librairie de l'Université, 1910.

Dilatation of the Stomach.—Organic dilatation is met with in cases of congenital stenosis of the pylorus or duodenum; acquired stenosis of the pylorus leading to dilatation of the stomach is practically unknown in early life. The symptoms and signs of dilatation are described in the section on Congenital Pyloric Stenosis. In these patients who survive, the dilatation usually disappears before the end of the first year, but in rare cases it may persist to a greater or lesser degree. The symptoms and signs are then the same as those of obstructive dilatation in the adult, and the best treatment is by operation.

Atony or Primary Dilatation of the stomach is not uncommon in a minor degree in children who suffer from chronic indigestion (p. 154), especially in those who are addicted to overeating or to drinking large quantities of liquid with their meals. It is sometimes met with also in rickety infants, although the importance which has been attached to it as a factor in the production of rickets by some French writers is greatly exaggerated.

The symptoms of atonic dilatation are the same as those of chronic indigestion, and the chief sign of it is the presence of an epigastric splash, which can be elicited several hours after a meal.

Treatment must proceed on the lines laid down for cases of chronic indigestion (p. 155), but it is especially important that the meals should be as dry as possible. Bitter tonics, strychnine, massage, and exercises, are all of help. Lavage is never called for.

Acute Dilatation may occur after abdominal operations in children, just as it may in the adult, and it is also sometimes met with as a terminal event in children who are dying from exhaustion particularly associated with respiratory disease. In these cases the intestines also become greatly distended with gas, and the pushing up of the diaphragm which results may contribute to the fatal issue.

The best treatment in such cases is the passage of a stomach-tube to relieve tension, and the hypodermic injection of strychnine.

Hair-Ball.—In children who habitually chew hair, a matted mass forming a hair-ball may form in the stomach. The diagnosis can be made from the history, in conjunction with the presence in the abdomen of a tumour which outlines the stomach, and produces a crackling sensation on palpation. Confirmatory evidence is furnished by the passage of a stomach-tube, when it will be found that the tube cannot be passed beyond the cardiac orifice, and that it is not possible to get much fluid into the stomach. Small bundles of hair may also be detected in the stool. The treatment is by operation, and the prognosis is good.

Ulcer of the Stomach.—Superficial erosions of the mucous membrane of the stomach are met with in cases of the hemorrhagic disease (p. 92), in septic conditions, and also in acute gastritis. They may lead to more or less hæmatemesis. Extensive ulceration occurs as the result of the swallowing of corrosives, or it may follow injury by foreign bodies. Tuberculous ulcers are also met with occasionally in children who have succumbed to general tuberculosis, but they do not produce any symptoms which can be recognized during life. The peptic ulcer, so common in the adult, is very rare in childhood. Cases have been collected by Jacobi and others. Perforation and peritonitis have sometimes resulted from the ulcer, but the diagnosis can rarely be made during life unless at operation.

REFERENCE.

Jacobi: *New York Med. Journ.*, 1909, no. 837.

Tumors of the Stomach.—Sarcoma and carcinoma of the stomach may occur in childhood, but both are excessively rare, and are almost incapable of being diagnosed during life.

DISEASES OF THE RECTUM AND ANUS.

Congenital Malformations.—Imperfect development of the hinder end of the primitive gut from which the rectum is derived, or of the proctodæum or anal dimple from which the anal canal proper is formed, or of both, results in many types of so-called "imperforate anus." This term should only be used, however, when the development of the proctodæum is imperfect. There may then be found no trace of an anus, or it may be represented by a dimple, by a solid fibrous cord, or by a blind cul-de-sac, with a septum of varying density intervening between it and the lumen of the rectum. Sometimes merely narrowing of an apparently normal anal canal is present. Atresia of the rectum as a congenital anomaly of varied extent may be associated with any of the foregoing anal imperfections or with a normal anus. When the anal canal is not properly formed, the rectum may end blindly, or open into the vagina, into the uterus, into the bladder, or even at the umbilicus, if the atresia involves the whole extent of the large gut. Lastly, into an apparently normal rectum and anus the uterus, vagina, or ureters, may have openings.

DIAGNOSIS.—The deformity may be obvious when the child is born, or may not be noticed until signs of intestinal obstruction begin, or meconium and feces are passed through abnormal openings.

TREATMENT.—Operative treatment is the only available means. For congenital narrowing of the anal canal dilatation with the finger is all that is necessary to secure a good result. As a possible cause of chronic constipation in children this should not be overlooked. For the lesser grades of deformity, such as the persistence of a thin septum between the anal canal and rectum, perforation must be performed, and the opening, which in healing tends to form a stricture, must be kept patent by daily dilatation. For the more serious deformities plastic operations are often resorted to, but in the majority of cases the best result that can be achieved is associated with a permanent incontinence of feces. Fortunately, the vitality of all such patients is exceedingly small, and when they survive

their immediate troubles, they tend to die within the first year of life. It cannot be urged that the proper province of surgery is to enable individuals to attain a sensitive age accompanied by all the horrors of hopeless fecal incontinence. The sufferings of intestinal obstruction may be relieved, where the deformity permits, by a perineal opening, but where this is impossible an inguinal colostomy should be made.

Incontinence of Feces.—This functional disorder may be present rarely in association with the delayed development of an otherwise healthy child; it is more particularly seen in first children, and sometimes in cases of very early rickets. It need give rise to no particular anxiety, as the condition will disappear as the child develops and responds to educational efforts. In association with lesions of the central nervous system it is a far graver disorder. When present in cases of idiocy and imbecility or of recovered diffuse poliomyelitis, very little can be done beyond fitting the patient with a rubber receptacle. It may be present in lesions of the spinal cord, such as spina bifida, spina bifida occulta, syringomyelia, and extensive poliomyelitis of the lumbar region. It occurs as a pressure symptom in Pott's disease and in acute osteomyelitis of the vertebral column. As a transitory trouble it makes its appearance in many acute febrile illnesses.

The restoration of the function in all these cases depends upon the degree of recovery of which the spinal cord is capable. In Pott's disease, weight extension to the spine is the most effective treatment of pressure paraplegia, and when successful fecal incontinence is one of the earliest signs to disappear. Laminectomy is seldom indicated in Pott's disease, and is still less frequently of any value. In acute osteomyelitis of the vertebral column in a boy of ten years old, with complete pressure paraplegia of the lower thoracic region, laminectomy resulted in a complete cure within a year, and he regained control of his feces within two months from the date of operation.

Removal of the sac of a spina bifida has no effect upon fecal incontinence, which is due to the associated syringomyelia of the cord present in all these cases. The ultimate prognosis is bad. In spina bifida occulta, in which a small dimple of the skin in the lumbar region may be the only evidence of the cause of the permanent fecal incontinence, a plastic operation may be done to fashion a functioning sphincter by interlacing large opposed portions of each gluteus maximus muscle around the anal opening. In these cases a skiagram of the lumbar vertebrae is useful in confirming the diagnosis. In extensive poliomyelitis fecal incontinence may be present for a year or two, but generally tends to spontaneous recovery. When there is no hope of this, a similar plastic operation may be done, provided the gluteal muscles are unaffected.

Prolapse.—Eversion of the mucous membrane at the anal orifice may occur as the result of local irritation, and must be distinguished from true prolapse of the rectum, in which all coats of the bowel are involved.

This latter condition may be congenital in cases of spina bifida, and in the female may be associated with prolapse of the uterus. The treatment is purely palliative. In the production of acquired prolapse, two factors are active—(1) debility or paralysis of the pelvic musculature supporting the lower part of the gut; (2) excessive or frequent straining at defecation or micturition.

General malnutrition, wasting diseases, and rickets, with profound muscular

ability, account for the former factor. The latter may be produced as the result of local irritation of the lower part of the intestine or from general functional disorders of the alimentary tract. The most active local conditions which cause a frequent desire to empty the bowel, and therefore in any twenty-four hours produce a sum total of excessive strain, are worms, rectal polyp or fibro-adenoma, hæmorrhoids, small necroid tumours, and more rarely anal fissures and proctitis. Diarrhoea and constipation, from whatever cause, are the two general conditions tending to the production of prolapse. Any condition causing straining during micturition may lead indirectly to prolapse of the rectum.

DIAGNOSIS.—It is of the utmost importance to distinguish between simple rectal prolapse and a prolapsed intussusception. Examination of the relation of the outer surface of the tumour to the anal margin readily establishes the diagnosis (*vide* Intussusception, p. 174 and accompanying diagram).

TREATMENT.—Both factors which together produce prolapse must be sought for and receive appropriate treatment if a successful result is to be obtained in these cases. It is useless to treat a rectal prolapse by posture and reposition of the tumour only, whilst ignoring the presence of rickets or of any other cause of muscular enfeeblement. Appropriate diet, regulation of the action of the bowels, and good hygienic surroundings, are necessary in all cases. Sources of local irritation must be removed, such as a polypus or hæmorrhoid, and worms must be got rid of in the ordinary way. When the prolapse is present, it should be bathed with cold water, and then dusted with boiled iodoform powder or with iodoform ointment, before reposition. Iodoform is an excellent local anæsthetic and antiseptic to the rectal mucosa, and, provided that it is sterilized before using, signs of so-called "iodoform-poisoning" do not make their appearance. Local irritation is speedily allayed by its use. The child should be taught to pass its motions while lying on one side, and afterwards, if necessary, the buttocks may be strapped together with Beiersdorf's Urin's paste strapping. In intractable cases, with several inches of prolapse, the child must be kept in bed, with the pelvis raised and the feet up in the air. All strain is thereby taken off the pelvic musculature. There are few cases that do not in the end recover with this mode of treatment. It may be necessary to persist for six to eight weeks. In my experience, operative treatment has never been necessary; it should be a last resource.

The treatment of general causes of straining, such as diarrhoea and constipation, must be carried out on the ordinary lines. Irritating motions, in children who, apart from diarrhoea, experience a scalding sensation in the lower part of the intestine, are generally due to dietary defects. The local irritation is best relieved by washing out the lower part of the rectum with a warm solution of bicarbonate of soda of the strength of 2 drachms to the pint of water.

Inflammatory Lesions.—Proctitis may occur as the result of local irritation produced by decomposing motions, from the introduction of a foreign body, such as a piece of soap, or glycerine enemata too frequently administered, from the presence of worms, from a prolapsed and neglected prolapse of the rectum, from trauma following a blow on the perineum accompanying a fall, and from vulvovaginitis, accompanied by a profuse discharge, which has spread backwards by contact with soiled linen to the anal orifice. There may be a simple or a gonorrhœal infection. Treatment must be directed to removing the cause. Local irrigation with warm solutions of boric acid, permanganate of potash, tincture of iodine

$\frac{1}{2}$ drachm to a pint of water, several times a day, to be followed by the use of an iodoform suppository, is most effective in relieving the symptoms.

Perianal Abscess may follow upon a neglected proctitis, or may be the result of a simple abrasion of the epithelial surface in that region. In children such abscesses are usually situated superficially, and very seldom involve the ischio-rectal fossa. Their onset is insidious, local symptoms are not complained of by the child as a rule, and a high temperature is a frequent accompaniment of the condition. It is a sound rule, in children in whom the temperature is raised without any obvious cause, to examine both ends of the alimentary tract. A pharyngeal abscess or a perianal abscess may be found to account for the fever. Simple incision, followed by hot fomentations, is the only treatment necessary. Healing takes place so rapidly that it is almost superfluous to state that if it be delayed *fistula* may ensue. This condition is exceedingly rare, and as a rule is the outcome of neglect or of delayed treatment. If it does occur, it must be treated by free incision and setting in the usual way.

True *Ischio-Rectal Abscess* may be caused by the spread of anal infection into the fossa, and in the early stages appears as a hard, red, brawny, tender, and painful swelling between the anal margin and the ischial tuberosity. It is accompanied by a rapid pulse and a raised temperature. This, however, is a rare mode of formation. More commonly lesions of the epiphyses of the pelvic girdle are the starting-points of ischio-rectal abscesses in children, and are frequently tuberculous in nature. The ischial tuberosity, the ischial spine, the acetabulum on its pelvic aspect, and even the sacro-iliac articulation, must be carefully examined in all cases of ischio-rectal abscess in which there are no signs of anal irritation, and in which the illness is not accompanied by much febrile reaction. Excision of the abscess, setting of the base bare, and immediate closure of the wound by stitching is the most effective treatment. This may have to be repeated several times, but by so doing there is a good chance of avoiding secondary infection. Tubercula may be given, but I have not found it of much value.

Solitary Hemorrhoids are rare conditions in children, and if they give rise to trouble should be ligatured and excised. Compression of the whole of the "pile-bearing" area is frequently seen in association with retroperitoneal sarcomata, especially when situated in the pelvis; with large kidney tumours, both sarcomata and hypernephromata; and with cirrhosis of the liver. Sedative treatment may be applied if necessary. A fissure may result from local trauma, from the tearing of the base of one of the columns of Morgagni, or of the base of a pile. This condition is not common, and should be treated by dilatation of the sphincter and under an anæsthetic, with incision of the fissure. In congenital syphilis cracks may radiate out around the anal margin. These are not classed as "fissures" in the accepted meaning of the term, but they often give rise to the same local troubles. Antisyphilitic treatment is the proper remedy.

Tumours.—Condylomata are common in congenital syphilis, and appear as greyish plaques of sodden epithelium starting on one side of the anal margin, and spreading by contact to the opposite side. In neglected cases they appear as warty, verrugated, and slightly raised masses. The presence of other stigmata and a positive Wassermann reaction establish the diagnosis. They may make their appearance in children of all ages. The warty masses, when evidence of syphilis is not forthcoming, are sometimes found to be in reality tubercular. In such cases excision is then necessary. In other cases antisyphilitic remedies must be used.

Rectal Polypus is either a myxoma or a fibro-adenoma. It is a frequent cause of straining, of prolapse of the rectum, and of hæmorrhage from the bowel. Its presence must be suspected in any of these conditions, and a rectal examination made. Digital examination alone is not reliable, and before deciding that a polypus is not present it must be excluded by examination with a rectal speculum and a good head-light. A small sterilized silk ligature must be tied round the base, and the polypus snipped off, leaving just enough pedicle to prevent the ligature from slipping. Iodoform ointment introduced at the time of the operation relieves subsequent irritation and prevents inflammatory reaction. Polypi of the fibro-adenomatous variety are frequently multiple. They involve almost the whole area of the large intestine, and treatment is unavailing.

Rare tumours are *angiomas* and *lipomas*. Other tumours in this region which may encroach upon the rectum, although not originating from it, are *retro-peritoneal sarcomata* originating in glands, or from the sacrum and coccyx; *teratomata*; *cysts of a persistent neuroenteric canal*; and occasionally true *rectal dermoids*. Removal of such tumours is indicated whenever possible.

INTESTINAL PARASITES.

The intestinal worms most commonly met with in children in this country are the *Oxyuris vermicularis*, *Ascaris lumbricoides*, and, of the tapeworms, *Tænia mediocanellata* (beef tapeworm), *T. solium* (pork tapeworm), and more rarely *T. cucumerina* or *elliptica*.

The characters of most of these worms are described in all textbooks of medicine. The *T. cucumerina* is derived from the feces of dogs and cats, and children become infected by playing with these animals. It is a short worm not exceeding 60 inches in length, with small oval segments.

The *T. solium*, or dwarf tapeworm, is sometimes met with also, but is very rare in this country.

Tapeworms and roundworms have their habitat in the small intestine; threadworms in the colon. There is usually but one *Tænia* present, but roundworms are often multiple, and threadworms always so.

Symptoms.—The general symptoms produced by intestinal worms are not characteristic, and it is never possible to arrive at an exact diagnosis unless the parasite or a portion of it has been seen in the stools. The general symptoms comprise such signs as anæmia and loss of weight. Various gastro-intestinal symptoms are also described, such as colicky pains, nausea or vomiting, and disturbances of appetite. Amongst nervous symptoms may be mentioned itching of the nose, irritability, dizziness, disturbed sleep, functional paralysis, and convulsions. They are commonest in cases in which roundworms are present. Local symptoms, such as rectal irritation, vulvitis, and enuresis, are met with almost exclusively in cases of threadworms. Certain cutaneous manifestations—e.g., erythema and urticaria may occur where round or tape worms are present. Eosinophilia of various degrees is apt to occur with all forms, but tends to disappear when the infection is of long standing, and is very inconstant. Roundworms, owing to their migratory tendency, occasionally give rise to such symptoms as are produced by blocking of the common bile-duct, or they may find their way into the air-passages and produce symptoms of tracheal obstruc-

tion. Cases of intestinal obstruction brought about by masses of ascariæ have also been reported.

There is reason to believe that the vermiform appendix may serve as a breeding-ground for *Oxyuris*, from which a re-infection of the bowel commonly takes place, and in this way the disease is apt to be perpetuated. Appendicitis has sometimes been induced by its presence.

TREATMENT.—Tapeworms.—Suitable preparation of the patient is of the first importance if treatment for tapeworms is to be satisfactory, and for two days prior to the administration of the anthelmintic the child should be kept in bed on a spare liquid diet, and the bowels thoroughly opened each day by a suitable aperient. Oil of male fern is the parasiticide most in favour in this country, 15 minims of the extract being given in capsules at intervals of half an hour for three doses and followed after the lapse of another hour by a smart purge. The proprietary preparation, Oil of Filmaron, which is the active principle of male fern dissolved in castor-oil, is also very useful, and may be given in doses of from 2 to 4 drachms. It does not need to be followed by any other aperient. The motions should be passed into warm water in a vessel lined with black crepe to facilitate the recognition of the head.

If the head be not found, it is useless to repeat the treatment until after the lapse of six or eight weeks, when the worm will have grown again.

Ascariæ.—These are best treated with xanthonin. It should be given with an aperient such as santonin, calomel, or castor-oil, the following being a useful formula:

Santonin	grs. ii ss.
Compound santonin powder	grs. ss.
Calomel	grs. ss.

Threadworms.—The presence of threadworms is usually an indication of an unhealthy state of the large bowel, and especially of the presence of an excess of mucus. This condition of things should be treated on the lines laid down in the section on chronic dyspepsia in later childhood. In many cases these measures are sufficient to effect a cure, but in the more obstinate cases one may be obliged to have recourse to the use of vermifuge drugs or local treatment. Of drugs, xanthonin is useful, given in the same way as for roundworms, or sulphur in $\frac{1}{2}$ -grain doses three times a day. Petroleum emulsion (B.P.C.) in drachm doses three daily has also been recommended.

Local treatment by enemata of salt and water, 1 tablespoonful to $\frac{1}{2}$ pint, is an old-established method. The solution should be run in through a funnel and tube after the bowels have first been emptied by an enema of soap and water. Infusion of quassa or garlic may be used in the same way, or an injection consisting of 2 drachms of oil of turpentine and 2 grains of xanthonin in 4 ounces of starch-mucilage. Suppositories are also of service; they may contain 3 grains of santonin or 4 to 7 grains of mercurial nitrate ointment, one being inserted every night at bedtime. Nitrate of mercury or blue ointment may also be applied locally for the relief of itching around the folds of the anus. Strict cleanliness should be practised in order to prevent re-infection, attention being especially devoted to the nails. Salads, watercress, and other raw vegetables, should be banished from the diet.

CHAPTER V

DISEASES OF THE LIVER, PANCREAS, AND PERITONEUM

H. MORLEY FLETCHER

INTRODUCTION.

JAVENOUS:

- CATAHRAL JAUNDICE.
- ACUTE YELLOW ATROPHY.
- ORIENTAL FAMILIAL JAUNDICE.

CYRROSES OF THE LIVER:

- MULTILOBULAR OR PORTAL CYRROSES.
- MONO- OR INTERLOBULAR CYRROSES (BILIOUS CYRROSES).
- SYNTHETIC CYRROSES.
- CYRROSES IN CHRONIC CARDIAC DISEASE, AND PNEUMONITIS.

TUBERCULOSIS OF THE LIVER.

SYRRIALS OF THE LIVER.

TONORS OF THE LIVER:

- MALIGNANT TONORS.
- INNOCENT TONORS.
- CYSTS.

PATTY LIVER.

LARDACEOUS LIVER.

ANEROS OF THE LIVER.

DISEASES OF THE GALL-BLADDER AND BILE-DUCTS.

DISEASES OF THE LIVER.

THE liver is a functionally active organ from an early period of intra-uterine life, and its importance is represented by its great weight relative to that of the foetus. At three months its weight is as much as one-half of the foetal body-weight, and then gradually falls, so that at birth the relative weight is only about one-twentieth of the body-weight.

The chief functions of the liver *in utero* are hæmatopoietic, glycogenic, and secretory.

The part played by the liver in blood-formation (*hæmatopoietic*) is still uncertain, but there is little doubt that the organ is concerned in the production of blood-corpuscles up to the end of intra-uterine life.

The *glycogenic function* develops about the fifth month, and glycogen is present in considerable quantity in the liver of the new-born child.

The *biliary secretion* about the third month consists of a colourless mucus containing granules of bile-pigment; bile is definitely formed at the fifth or sixth month of intra-uterine life.

The liver in the foetus is at first a symmetrical organ, with right and left lobes of equal size. After the fourth month (Schäfer) the right lobe increases more rapidly than the left, so that at birth it is considerably the larger. Pressure exerted by neighbouring organs probably plays a part in retarding the growth of the left lobe and causing the asymmetry. The liver is very vascular in the foetus and new-born infant, and contains a higher percentage of iron than is found in the adult liver. To these factors the dark colour of the liver of the infant is due. The greater vascularity of the child's liver as compared with that of the adult accounts for the

more rapid and marked response to active or passive congestive states of the organ in childhood, as shown by an early increase in size, especially in acute febrile conditions.

After birth the weight of the liver relatively to the body-weight steadily diminishes, and falls from one-twentieth of the body-weight at birth to about one-thirty-fourth in the adult.

In the infant the liver, owing to its large size, can be generally readily felt one to two fingers' breadth below the costal margin. Thoracic deformities, such as are commonly associated with rickets, often cause displacement downwards of the organ, which may be mistaken for enlargement. The liver is also readily displaced downwards by pleural and pericardial effusions. Displacement upwards may be brought about as the result of paralysis and atrophy of the right side of the diaphragm, two examples of which I have met with following severe attacks of acute poliomyelitis in children. Cases of undue mobility of the liver (*hepatoptosis*) have been described, but are much rarer than in adult life. Undue laxity of the abdominal wall may bring about a diminution of the support normally offered to the liver by the cushions formed by the intestines, so that the organ descends lower than normal. Loss of tone in the abdominal muscles is caused by various conditions accompanied by impairment of general nutrition. Hirtzel points out that violent coughing, as in pertussis, may be a determinant cause of the ptosis in children. In such cases physical examination shows displacement downwards of the area of hepatic dulness, thus distinguishing the condition from mere enlargement of the liver. Owing to defective movement of the diaphragm, which is dragged down by the displaced liver, the breath-sounds over the right lung may be less marked than on the left side. By pressing the liver upwards into its normal situation, the respiratory murmur becomes normal. *Hepatoptosis* is stated to occur also in cases of functional albuminuria in adolescence. In transposition of the viscera the liver is situated on the left side, and the arrangement of the lobes exactly reversed, the left lobe being the larger. It will be sufficient to mention briefly the malformations of the liver met with in children; these are as follows: Abnormal arrangement of lobes—*trigone-like lobes* (Riedel's)—have been described in infants. *Ectopia hepatis* or *hepatomphalos* is a condition in which there is a defect in the abdominal wall, so that the liver forms a projection covered only by the skin, in the right hypochondriac and epigastric regions.

JAUNDICE.

Certain forms of jaundice are peculiar to earliest infancy. These constitute a special group, and will be dealt with in the section devoted to the diseases of the newly-born. It will suffice to give here an enumeration of those types which occur only in the newly-born or quite young infants:

I. **ICTERUS NEONATORUM.**—A common and transient form of jaundice occurring very shortly after birth in a large proportion of infants, and due to physiological rather than pathological factors (see Chapter I, p. 78).

II. **CONGENITAL OBSTRUCTION OF THE BILE-DUCT.**—Chronic obstructive jaundice with *schistic stools*, due to occlusion of the bile-ducts (vide p. 230).

III. TOXI-INFECTIVE FORMS.—(a) *Jaundice due to Umbilical Infection.*—Usually streptococcal in nature. The umbilical vein contains suppurating thrombi. The jaundice is due to a portal pyæmia (see Chapter I., p. 81).

(b) *Septicæmic Fever.*—These may be caused by infective conditions, such as erysipelas, etc.

(c) *Epidemic Forms.*—Under this heading are included forms of jaundice in the newly-born attended by hæmorrhages from the navel, bronzing of the skin, and hæmaturia, which have an epidemic character. These have been described by various writers, such as Winckel, Buhl, and others. They are probably due to a septicæmia resulting from intestinal infection (see Chapter I., p. 91).

While the foregoing should be regarded as types of jaundice to which the newly-born are specially liable, it should be pointed out that several of the varieties met with in later infancy and childhood, such as catarrhal jaundice, jaundice due to gall-stones, syphilis and other hepatic disease, etc., may occur also in early infancy. We shall now proceed to give a general classification of the causes of jaundice occurring in children.

I. Obstructive Forms.—Those due to obstruction of the extrahepatic or larger bile-ducts.

II. Non-Obstructive Forms.—Those not due to obstruction of the extrahepatic bile-ducts.

I. Obstructive Forms of Jaundice.—1. *Due to Pressure on the Bile-Ducts.*—(a) By enlarged glands (tuberculous, lymphadenomatous, malignant, leikæmic).

(b) By hydatid cysts.

(c) By bands or adhesions, the result of peritonitis.

2. *Due to Changes in the Wall of the Ducts.*—(a) Swelling of the mucous membrane, as in catarrhal jaundice, cholangitis.

(b) Obliteration of the bile-ducts in infants.

3. *Due to Obstructions within the Duct.*—(a) Gall-stones.

(b) Parasites.

In *obstructive jaundice* the urine is not uncommonly noticed to have an unusually dark colour, due to the presence of bile pigment, before any yellow tint has been observed either of the conjunctiva or skin, though this rapidly develops. In infants, staining of the napkins by the bile pigments present in the urine is not infrequently the first thing to attract attention. The urine contains bile pigment and bile salts. The bile salts are said to disappear after the first few days of obstructive jaundice as a result of diminished production of the bile acids. Urobilin is not present in the urine unless bile is entering the intestine. The feces contain no bile pigment if the obstruction is complete, and become putty- or clay-coloured; in infants with complete obstruction the stools are often quite white, and resemble white of egg or curds.

Obstructive jaundice of long duration very rarely occurs in children, apart from congenital obliteration of the bile-ducts, and even in this condition it is most unusual to find the dark olive-green tint met with in chronic obstructive forms in adults. Itching of the skin and slowing of the pulse, common manifestations in the jaundice of adults, are generally admitted to be most uncommon occurrences in children, and the writer has never met with either of them. Drowsiness is common in cases of jaundice in young infants. In the chronic jaundice of infants the earliest teeth erupted may be yellow or green. This is sometimes seen in babies

who were jaundiced at birth or shortly afterwards, and in whom the jaundice has persisted for many weeks or months. The teeth when first erupted are yellow, but later become green; the coloration gradually fades. It is probably due to a deposition of bile pigment in the dentine in the first weeks of life (note cases described by Thursfield and Langmead, *Proceedings of the Royal Society of Medicine, section of Diseases of Children*, 1912, v. 147).

II. Non-Obstructive Forms of Jaundice.—These are of greater frequency in childhood than are the obstructive forms, and occur in the following conditions:

1. *Hepatic Disease*.—
 - (a) Various forms of cirrhosis.
 - (b) Congestion: in cardiac disease and polyserositis.
 - (c) Congenital syphilis.
 - (d) Tuberculosis.
 - (e) Tumours, cysts, granular, abscess, pyklophlebitis, portal pyaemia.
2. *Toxi-Infective Conditions*.—
 - (a) Specific fevers: pneumonia, septicæmia, Weil's disease, etc.
 - (b) Icterus gravis and acute yellow atrophy.
 - (c) Infective forms in the newly-born: umbilical infection, Wiedel's disease.
3. *Toxic States*.—Phosphorus and carbolic acid poisoning.
4. *Familial Type*.—Acholeuro family jaundice.
5. *After Severe Haemorrhage*.
6. *Cyclic Vomiting*.
7. *Emotional Jaundice*.

Several of these forms of non-obstructive jaundice are rare, and require little further notice. Cases of jaundice have been described in infants which have resulted from applications of carbolic dressings. Severe hæmorrhage from a wound in a young infant may be followed by jaundice. The variety called "emotional jaundice" includes those cases in which jaundice definitely appears to follow, and to be due to, a severe fright or other mental shock.

Non-Obstructive Forms.—In these the jaundice is usually less marked than in the obstructive forms; it may be so slight as to cause nothing more obvious than a slight yellow tint of the conjunctivæ.

The urine as a rule contains little or no bile pigment, even when the jaundice is distinct, though it is often much darker than normal owing to the presence of urobilin. Bile salts are usually absent. In the toxi-infective forms and in acute yellow atrophy, albumin and casts are generally present, and in the latter disease leucin and tyrosin may be detected, and in these acute forms uræa is often considerably diminished in amount.

DIAGNOSIS.—In newly-born babies the presence of slight jaundice may escape notice owing to the pink colour of the skin. Firm pressure of the finger by emptying the cutaneous vessels produces momentarily a pale area in which a yellow tint may be distinctly visible.

The first point in the diagnosis of the nature of the disease in a case of jaundice is to determine to which of the two forms—obstructive or non-obstructive—it belongs. This does not present much difficulty if the urine and stool are carefully examined; the appearance presented by them in both forms of jaundice has been already described.

In infants persistent obstructive jaundice, dating from birth or very shortly afterwards, is most likely to be due to obliteration of the bile ducts. Catarrhal jaundice may occur in infants, though it is rare in the first year, and usually passes off in a week or two. Chronic obstructive jaundice should therefore give rise to grave anxiety in early infancy as a symptom of serious organic disease.

In older children catarrhal jaundice is by far the most common cause of obstructive jaundice, most of the other forms being distinctly rare. The diagnosis of catarrhal jaundice, with its early gastro-intestinal symptoms, is discussed elsewhere; but it must be remembered that acute yellow atrophy may have an initial course closely resembling that of catarrhal jaundice. In other cases of obstructive jaundice abdominal examination may reveal the presence of enlarged glands or a distended gall bladder, or there may be an antecedent history of peritonitis. Irregular or nodular enlargement of the liver would suggest some cirrhotic condition, granular, multiple abscesses, hydatid cyst, or new growth.

In the non-obstructive forms of jaundice the diagnosis is liable to be attended with much more difficulty than in the obstructive forms. Let us first consider this in early infancy. The transient jaundice due to physiological causes, known as "*icterus neonatorum*," appearing shortly after birth, is usually slight and of short duration, and need cause no anxiety. On the other hand, jaundice in the new-born is a most serious portent if accompanied by pyrexia, umbilical inflammation, erysipelas, hæmorrhage into the skin or from the navel or bowel.

The fatal type of jaundice is met with in infants, and careful inquiry should be made into the family history. In such cases a great diminution of the red corpuscles may be present due to hæmalysis.

"Non-obstructive" forms of jaundice in older children, due to chronic forms of hepatic disease, are generally afebrile; but pyrexia may supervene in the terminal and fatal condition known as "*icterus gravis*," which is brought about by some toxic or infective cause acting on the diseased liver.

Catarrhal Jaundice.—Catarrhal jaundice is common at all ages of childhood, and may occur even in quite young infants. Its clinical manifestations in children differ in no essential respect from those met with in the adult. The onset of the attack is characterized by gastro-intestinal disturbance of varying degrees of severity, such as constipation or diarrhoea, with offensive stools, and later by vomiting. Jaundice appears two or three days after the onset, and is usually first noticed in the sclerotics; the coloration of the skin rapidly follows. The urine becomes dark brown owing to the presence of bile pigments, and the stools are usually clay-coloured.

The child is peevish and fretful, the tongue is furred, and the breath offensive. Slowing of the pulse-rate is much more rarely met with in children than in adults. At the onset, especially in young children, some degree of pyrexia is not unusual while the gastro-intestinal symptoms are present, but this disappears as the vomiting and diarrhoea cease, and the temperature afterwards remains normal. Persistence of pyrexia is an unfavourable sign, as it would indicate, in the absence of other complications, that the case is not one of simple catarrhal jaundice.

The degree of jaundice varies greatly, from a slight yellowness of the sclerotics to a bright yellow coloration of the skin. It is rarely accompanied by itching.

The liver becomes enlarged early, and its lower border may extend two or three fingers' breadth below the costal margin, and not infrequently it is tender on palpation. Abdominal pain, usually located in the upper abdomen, is not uncommon,

and is sometimes severe. In a few days the symptoms rapidly subside. Vomiting and diarrhea cease, and the hepatic enlargement and tenderness diminish. The urine and stools gradually resume their normal colour, and the jaundice fades. Recovery is usually complete in a week to a fortnight after the onset of the jaundice, though sometimes cases are met with in which the jaundice persists for several weeks, with occasional slight remissions in intensity.

Prognosis.—The course of the disease is almost invariably favourable, but attention must be drawn to certain points which may affect the prognosis adversely, and give rise to anxiety. (1) The age of the child. In very young infants the occurrence of jaundice, apart from the form known as "icterus neonatorum," may be attended with considerable uncertainty as to the nature of the case, and it may be difficult to exclude obliteration of the bile-ducts or the infective form of jaundice until the favourable course of the case has cleared up the diagnosis. (2) The persistence of pyrexia after the subsidence of the initial gastro-intestinal symptoms, the presence of cerebral disturbances such as drowsiness or delirium, and the appearance of hemorrhages either in the skin or from the mucous membranes, must be regarded as very unfavourable signs, as they would indicate a grave toxic or infective condition, such as acute yellow atrophy or icterus gravis.

Splenic enlargement is unusual, and its occurrence should arouse suspicion that the case is not one of simple catarrhal jaundice. For the further consideration of these points and of the differential diagnosis of the disease, reference should be made to pp. 214, 215.

Pathology.—The jaundice is due to an inflammation of the papilla of the common bile-duct or ampulla of Vater, the result of gastro-duodenal catarrh. The biliary papilla becomes swollen and the flow of bile obstructed. In the ordinary mild type of case the inflammation probably does not extend far up the common bile-duct, but is limited to the papilla, though in the more severe forms, such as may be associated with the specific fevers, the biliary passages are more extensively involved.

The causes which may give rise to the gastro-duodenal catarrh are many and various: errors of diet, such as overeating or unsuitable articles of food, possibly impure milk or water, and acute febrile disorders which in childhood are not infrequently attended by gastro-intestinal disturbances.

As to the nature or cause of the inflammation, little is known, but it is probably due to some intestinal infection or to the action of septic toxin of bacterial origin. In this connection it is interesting to note the not infrequent occurrence of epidemics of what appear to be simple catarrhal jaundice. Several such epidemics were recorded during the hot summer of 1911, in which groups of cases occurred in the same locality, institution, or family. These cases differ from those known as "epidemic jaundice" or "Well's disease," which run a course of greater severity, and possess special clinical features. Still has observed, in some years, a seasonal variation in the number of cases attending the Children's Hospital, the maximum frequency occurring in October and November.

Treatment.—In all cases of catarrhal jaundice the child should be kept in bed, and, as exposure to cold is not uncommonly a factor in the causation of the condition, care should be taken to avoid any risk of chills. Treatment is directed to the removal of the gastro-intestinal catarrh. If vomiting be severe, nothing should be given by the mouth but water or whey. As the condition improves,

milk, veal, or chicken broth, can be taken, and the diet gradually increased by the addition of milk-puddings, beaten-up egg, rusks, etc.; but it is essential that nothing but food of this light nature be given until the jaundice has entirely disappeared. Undue haste in increasing the dietary is liable to be followed by an increase or return of the jaundice.

In cases after the vomiting has subsided there is nothing better in the writer's experience than the usual rhubarb-and-soda mixture, with the addition of sodium salicylate. The following prescription is suitable for a child of five:

Sodii salicylate	gr. v.
Sodii bicarb.	gr. v.
Infant's tea	℥ss.
Sp. anemol (Aerolat)	℥ss.
Mixt. saccharin	℥ss.
Aquæ anethi	ad ℥℥.

This dose to be given three daily in water.

If the vomiting be troublesome, 10-grain doses of oxycarbonate of bismuth should be given in an alkaline mixture. The bowels should be cleared by a dose of calomel. If the actions continue loose and offensive, β -naphthol, salol, or naphthalene tetrachloride, are of use; but they are not often required if the diet is carefully regulated.

When the jaundice has nearly disappeared and the gastro-intestinal symptoms have quite cleared up, a mixture containing 3 to 5 drops of dilute nitro-hydrochloric acid and 1 to 2 drops of tincture of nux vomica may be substituted for the alkaline mixture with advantage.

Acute Yellow Atrophy.—This is a rare condition at all ages, and especially so in childhood, though perhaps the small number of cases recorded in children leads one to form a somewhat exaggerated view of its rarity. Icterus gravis and acute yellow atrophy are sometimes wrongly used as synonymous terms for the same condition. Icterus gravis, or malignant jaundice, should be employed as a general term to designate the terminal jaundice occurring in cases of hepatic disease, such as cirrhosis, cardiac congestion, and phosphorus-poisoning. As Rolleston clearly states it: "Icterus gravis should be regarded not as a specific disease, but as a group of symptoms due to the rapid development of hepatic insufficiency, eventually becoming absolute, which may be due to many different causes." On the other hand, acute yellow atrophy is a special form of icterus gravis affecting a previously healthy liver, and constitutes a definite clinical type.

Etiology.—Age.—There does not seem to be any special age-liability to the disease. Cases have been described in children of all ages, even in earliest infancy. The youngest case is that recorded by Politzer in a new-born infant which died on the eighteenth day.

Sex.—Male children are more liable than female. Phillips, in a recently-published series of cases, gives the proportion as 2 males to 1 female. This is interesting, for in adult cases females are more liable than males in the ratio of 2 to 1. The greater frequency in adult females is probably due to the influence of pregnancy.

Influence of Other Diseases.—Syphilis is an important predisposing factor in the disease. It may act by bringing about a lowering of resistance to infection, but most often by giving rise to a pericellular cirrhosis of the liver.

Several cases have been recorded following the administration of chloroform.

SYMPTOMATOLOGY.—At the onset the symptoms usually resemble those of an attack of ordinary catarrhal jaundice, so that for some days little or no anxiety may be felt. General malaise, with vomiting and constipation, or, more rarely, diarrhoea, and some pyrexia, are the first symptoms. Jaundice appears at the end of a day or two later. Instead of a gradual subsidence, as would be expected in catarrhal jaundice, the case now passes into a second stage, in which the following manifestations occur. Jaundice deepens, though, as the case nears its termination, it may fade. Vomiting persists or increases in severity, and the vomit and stools may contain blood. Constipation is usually present. The temperature rises, though in some cases it falls, and may become even subnormal. Cerebral symptoms develop, such as drowsiness, photophobia, delirium, convulsions, and finally coma. Twitchings, especially of the face, are not uncommon, with peculiar sucking or clamping movements of the lips and teeth. Subcutaneous hæmorrhages and epistaxis are often present. The blood generally shows a leucocytosis of 20,000 to 25,000 per cubic millimetre; its alkalinity is stated to be reduced. The liver, which at first is definitely enlarged, becomes smaller as the disease progresses, and finally the hepatic dulness may be greatly reduced or may completely disappear. The spleen is sometimes enlarged. The urine is scanty and dark-coloured, owing to the presence of bile pigment. It contains albumin and numerous bile-stained epithelial casts. The presence of leucin and tyrosin can often be demonstrated by chemical methods, though these bodies are seldom found as sediments. Urea is considerably diminished in amount. Sugar is not present in the urine. The stools are usually very offensive, and may contain bile and sometimes blood.

The duration of the disease varies. Most cases terminate with coma in two to three weeks after the onset of symptoms. In some, however, the disease runs a less acute course, and after a variable period, from two to five weeks, a gradual amelioration is noticed; the cerebral symptoms disappear, the jaundice fades, the urine gradually regains its normal characters, and the liver dulness returns. There is a slow convalescence, but the subsequent state of health must be regarded as precarious.

The following case is a girl of five under the writer's care at St. Bartholomew's Hospital illustrates the subacute form of the disease. On November 13, 1909, jaundice was first noticed; she had vomited a few days previously. Admitted to hospital on November 21 distinctly jaundiced; liver enlarged 2 inches below costal arch; spleen palpable. Temperature, 102° F. The urine contained bile and numerous bile-stained casts; leucin was present, but no tyrosin crystals were found. Leucocytes 12,000 per cubic millimetre; later 24,000. During the next few days there was repeated vomiting; the vomit was streaked with blood on one occasion. Jaundice deepened. The liver rapidly diminished in size, so that it was no longer palpable, and the hepatic dulness became less than normal. Blood cultures were sterile. On November 26 she became very drowsy, with muttering delirium and twitching and sucking movements of the lips, and finally comatose. Repeated subcutaneous injections of saline solution were given with most striking results, and each injection was followed by some subsidence in the cerebral symptoms. The condition slowly improved; the urine became normal, and the jaundice finally faded with slight remissions. The liver became just palpable. Ultimately the child recovered after a long convalescence. She has been under observation up to the present time, and appears quite well, though delicate-looking.

DIAGNOSIS.—The recognition of this serious malady is difficult, or even impossible in its early stage, when it may so closely resemble an ordinary attack of catarrhal jaundice. Increasing intensity of jaundice, the persistence of pyrexia, petechial hemorrhages, and, above all, the onset of cerebral symptoms such as drowsiness, delirium, etc., should arouse suspicions as to the real nature of the illness. The presence of leucin and tyrosin, and of bile-stained casts in the urine, and a diminished area of hepatic dulness, will confirm the diagnosis of acute yellow atrophy.

From phosphorus-poisoning and *icterus gravis* in cirrhotic conditions, acute yellow atrophy may be distinguished by the reduction in size of the liver. In phosphorus-poisoning and in cirrhosis of the liver the organ remains enlarged.

PATHOLOGY.—The disease is due to an acute and very extensive degeneration of the liver cells. "The condition is a very acute hepatitis" (Rollleston). It is the result of an acute toxic or infective process brought about by bacterial infection from the intestine via the bile-ducts or by the blood-stream. In favour of the conveyance by the blood is the frequent association of enlargement of the spleen and degenerative changes in the kidneys.

The few recorded cases of acute yellow atrophy following chloroform narcosis would suggest that toxic bodies other than those of bacterial origin may also cause the condition.

MORBED ANATOMY.—The liver is diminished in size and weight, and has a mottled pale yellow or light orange colour. The capsule is wrinkled or thrown into folds; the liver substance feels soft and flabby. On section, a striking change is apparent; the normal liver is replaced by yellow and red areas. The yellow areas represent the remains of the glandular tissue, though little or none of the normal lobulation can be made out thereon by the naked eye. The red areas which are depressed below the level of the yellow areas, and are softer than these, exhibit no lobulation. Here the most extensive destruction of the glandular elements has taken place. Microscopically, the yellow areas are found to consist of liver cells in an advanced stage of degeneration—the protoplasm vacuolated, and the nuclei stained feebly or not at all. Hemorrhages and granules of bile pigment are also present. In places collections of small cubical cells arranged in rows are seen, resembling small bile-ducts. These are known as "pseudo-bile-ducts" or "canaliculi," and are probably the result of proliferation either of cells of the bile-ducts or of hepatic cells as an attempt at regeneration. Here and there may be distinguished small groups of large and apparently normal liver cells, which show evidence of rapid growth, and also represent a regenerative process. In the red areas few, if any, of the liver cells remain, and what is seen consists of a fibrous stroma, capillaries, debris of nuclei, red blood-cells, and bile pigment. The bile-ducts usually show signs of an acute catarrhal inflammation and contain masses of desquamated cells, and the jaundice is probably due to the obstruction of the bile capillaries and smaller ducts caused thereby. Leucin and tyrosin can be sometimes recognized microscopically, or may be found in scrapings from the cut surface of the liver. Micro-organisms have been found in the liver in a very small proportion of cases.

In the subacute forms of the disease, or those of longer duration which eventually recover, the favourable result is due to a regeneration of the liver cells, some evidence of which may be found even in the acute and fatal cases, as mentioned above. This regenerative process, by active proliferation of the hepatic cells, leads to the

formation of circumscribed masses of apparently normal liver tissue, and if life be prolonged may be sufficient to compensate in part at least for the destruction which has taken place. These newly-formed areas of liver cells, with their sharply-defined outlines, may then resemble masses of new growth, as they are paler than the surrounding tissue, and may even project on the surface of the liver. They are sometimes described as adenomata. The compensation is not very stable in such cases, and subsequently some comparatively slight disorder may cause hepatic insufficiency and death.

In children the liver may afford evidence of antecedent disease, such as cirrhosis of the syphilitic type, as shown by the presence of an intercellular fibrosis. In such cases the acute degenerative process cannot be said to have taken place in a previously healthy organ, and, strictly speaking, as Rolleston has pointed out they should be classed as cases of icterus gravis rather than acute yellow atrophy, as is more generally and perhaps more conveniently done.

PROGNOSIS.—This is very unfavourable, as by far the greatest number of cases terminate fatally. The longer the duration of the case the better becomes the chance of recovery. This can take place only as the result of regeneration of the liver cells, such as occurs in the subacute form, and the formation of new liver cells must have progressed to a considerable extent to discharge the hepatic functions to an adequate degree. The life of a child who has survived an attack of acute yellow atrophy is precarious, as the liver, in spite of the compensatory formation of liver cells, may give in at a later period from some slight affection which upsets its compensation.

TREATMENT.—At the onset the treatment is that of a case of catarrhal jaundice, rest in bed with fluid diet. The bowels should be cleared with calomel and saline purges, and alkalies and large quantities of fluid should be given. When cerebral symptoms appear, the use of saline infusions may have a remarkable effect in removing drowsiness and even coma, as in the case described above. The infusion probably act by aiding the elimination of toxic substances affecting the cerebral centres.

Congenital Family Jaundice.—This condition is also known as familial cholera, acholic family jaundice, splenomegalic family jaundice. For many years past it has been recognized that there may be a tendency to the development of jaundice in members of the same family, and numerous cases of this kind have been recorded in this and in other countries. Gilbert and Lecheboullet were probably the first to draw attention to this family tendency under the term *cholémie familiale*.

There are two types of congenital family jaundice: the first that in which jaundice is present at birth or appears shortly afterwards, and the second that in which jaundice appears in later childhood or early adult life. Both these types have the following characteristics in common: There is generally a history of jaundice in other members of the family, the urine is usually free from bile (*acholuria*), the spleen is enlarged. The condition is a chronic one, and as far as is at present known does not seriously endanger life except in early infancy.

Ætiology.—Hereditary.—This is the only important ætiological factor known at present. In nearly all the cases described there is a history of other members of the family having had jaundice in infancy or later life. In the infantile type the mother may be affected, and a history may be obtained of several of her children

having been born jaundiced and dying shortly after birth. The condition has been traced for several generations in a family.

Sex.—Both sexes appear to be equally liable.

Syphilis and other infective conditions are not concerned in the production of this form of jaundice.

Exposure to cold, over-exertion or fatigue, occasionally appear to act as exciting causes for the onset of the jaundice in the older class of cases, or may bring about an exacerbation.

SYMPTOMATOLOGY.—In the infantile form jaundice is present at birth or appears shortly afterwards. The jaundice is usually slight. The urine does not contain bile, and urobilin is present. The stools contain bile. The infant is usually somewhat drowsy, as in other forms of jaundice in early life. The spleen is distinctly enlarged, and there may be some enlargement of the liver. Examination of the blood reveals some degree of anemia, which is more marked in infants than in the later cases. The anemia is sometimes very severe, and is probably the cause of death in those cases which prove fatal in the first few weeks of life. The blood count in these infants may resemble that of pernicious anemia in the great reduction of the red cells and the presence of normoblasts and megaloblasts. Traces of bilibinin are found in the blood.

If the child survives the first few weeks or months of life, the jaundice tends to fade, and in course of time may disappear. The condition may entirely subside in infancy to reappear at intervals in later childhood or early adult life, or it may persist throughout. The general health is little impaired provided that the anemia during these exacerbations is not very severe. An excellent example of this maintenance of health and strength is afforded by a case described by Hawkins and Dudgeon, of a policeman of twenty-seven, who, though jaundiced from infancy, was able to discharge his duty.

In cases of familial jaundice in older children the general health is usually good and nutrition is well maintained, although in a few cases development is retarded and puberty delayed. The jaundice is slight or may disappear altogether at intervals, to reappear or become deeper after some chill or over-exertion, or without any apparent cause. The jaundice does not give rise to itching of the skin. During the exacerbations there may be some headache and a feeling of drowsiness or malaise, and the temperature may be slightly raised. Signs of gastro-intestinal disturbance are absent. The spleen is considerably enlarged, and may reach below the level of the umbilicus; it is hard and painless. Slight enlargement of the liver is present in some cases. There is, as a rule, no abdominal pain or tenderness.

The urine is dark-colored owing to the presence of urobilin, but bile is absent, though occasionally during an exacerbation traces of bilibinin may appear. The feces are normal in character and contain bile. The blood in the older cases does not show the same degree of anemia present in infants, but during the exacerbations the red cells are diminished to about 3,000,000, and there is marked poikilocytosis and polychromatophilia. Normoblasts and megaloblasts are present. The leucocytes are not affected in any characteristic way. Hemoglobin is about 44 per cent. (Hawkins and Dudgeon). Traces of bilibinin are found in the blood in the majority of cases.

PATHOLOGY.—It is believed that this family type of jaundice is due to some inborn or congenital defect in the red corpuscles of the nature of an undue fragility, as

that they become broken up or undergo hemolysis more readily than is normal. The evidence on this point rests on observations made as to the behavior of the corpuscles in saline solutions of graduated degrees of dilution. It is found that the red cells in cases of family jaundice are readily hemolyzed in solutions of strengths which have no effect on cells from normal cases. On the other hand, it is found that in ordinary forms of chronic jaundice the red cells resist hemolysis in weaker saline solutions than is the case with red cells of normal individuals. This fragility of the red cells in familial jaundice may be possibly due to some congenital abnormality of the red marrow. Hawkins and Hodgeon suggest that it may be associated with some defect in the stability of the haemoglobin compound present in the red cells.

The haemolysis of the red corpuscles leads to the production of increased bile pigment by the liver, which causes slight jaundice and the presence of urobilin in the urine. The spleen becomes enlarged as a result of the hemolysis.

Occasionally the hemolysis may be so excessive in the exacerbations that the amount of bile pigment in the blood becomes so much increased that traces of it appear in the urine. The anemia present in these cases is the direct consequence of the hemolysis, and it may be very severe in infants, as has been already pointed out.

The presence of normoblasts and megaloblasts may be an evidence of an increased activity of the erythroblastic tissues to compensate for the hemolysis. The enlargement of the spleen is probably the result of increased activity in connection with hemolysis. On post-mortem examination this organ, as well as the liver, exhibits some degree of fibrosis.

DIAGNOSIS.—The most important points in the diagnosis are the family history, the history of recurrent attacks of jaundice or of jaundice dating from infancy, the absence of bile in the urine and its presence in the stools, enlargement of the spleen, and, finally, evidence of abnormal fragility of the red corpuscles.

It is quite possible that some cases recorded as Hanot's type of bilious cirrhosis are really cases of acholic family jaundice, and it may be difficult to distinguish between these two conditions. In familial jaundice the enlargement of the liver is slight, while in Hanot's cirrhosis it is very considerable. The exacerbations in this form of cirrhosis are severe, and are attended with pain, pyrexia, and leucocytosis, while in family jaundice these are usually absent.

In *spéno anemia* (Banti's type) jaundice may occur with enlarged spleen and acholia, but in these cases there is a definite leucopenia and a tendency to hemorrhages from the mucous membranes.

The form of anemia with enlarged spleen in infants known as "pseudo-leukemia infantum" is not attended with jaundice, the leucocytes are usually considerably increased in number, and myelocytes are present.

PROGNOSIS.—This is favourable in cases which have survived early infancy, during which period there is considerable risk to life owing to the severe anemia resulting from hemolysis in new-born infants. Cases which begin in infancy, if they survive the first few months, may do better than those which develop later. An adult affected by this abnormality may lead a fairly normal life, as the general health is little affected. The intervals between the exacerbations tend to become longer as time goes on.

TREATMENT.—The greatest care should be exercised in the case of an infant in keeping it warm and regulating the diet to prevent the recurrence of any con-

plication, such as a chill or alimentary disturbance, which might have an unfavorable influence on the condition.

In the case of older children, attention to general health is important, and they require special care in the regulation of exercise, diet, etc. Drugs, as far as is known, have no influence on the condition; arsenic in particular does not produce any effect.

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CIRRHOSIS OF THE LIVER.

The liver of a child reacts even more promptly than that of an adult to the influence of toxic substances, whether of microbic or non-microbic origin, and we may find all stages of reaction resulting from such causes, varying in degree from a state of active congestion to that of a localized or diffuse small-celled infiltration, which may ultimately lead to the production of fibrous tissue constituting one or other of the types of cirrhosis.

The forms of cirrhosis occurring in children are as follows:

1. Multilobular or portal cirrhosis.
2. Mono- or inter-lobular cirrhosis:
 - (a) Biliary cirrhosis (Hanot's cirrhosis or hypertrophic cirrhosis).
 - (b) Cirrhosis due to chronic obstruction of the larger bile-ducts.
3. Syphilitic cirrhosis.
4. Cirrhosis due to passive congestion of the liver, as in cardiac disease and polyserositis (polyserositis).

1. Multilobular or Portal Cirrhosis.—This is by no means a common disease of childhood, and in the writer's opinion it is much less common now than it used to be. It differs in no material respect from the adult type of the disease. It is caused by toxic substances conveyed to the liver by the portal circulation, and is generally due to the ingestion of alcohol for a prolonged period. Alcohol in the form of brandy, port wine, or gin, administered in excessive doses to debilitated infants or young children, is the usual cause, though in some cases this can be excluded. There is reason to believe that the liver in young animals is more susceptible to the influence of alcohol than is the adult liver. It is probable that an unsuitable and irritating form of diet may also lead to cirrhosis. As Rolleston suggests, the resisting powers of the liver may be reduced either by some congenital defect or as the result of specific fevers, and rendered more liable to the influence of toxic substances.

Focal, inflammatory, or necrotic lesions are not uncommonly found in the liver in cases of infective fevers, such as typhoid and scarlet fever, etc., and it is possible that these may be associated later with the production of some degree of cirrhosis.

SYMPTOMATOLOGY.—The earliest symptoms are those of digestive disturbance, with failure of general nutrition. The child wastes, the skin becomes sallow, and

in the later stages jaundice may appear. Ascites develops, causing abdominal enlargement, which may be the first symptom to attract attention. Hematemesis is less common than in adults. The liver, if palpable, is hard, and the surface may be made out to be uneven or nodular. The spleen is usually enlarged, and ascites is present. The superficial abdominal veins are sometimes dilated. The course of the disease resembles that in the adult. The child becomes increasingly asthenic; pulmonary complications, such as bronchitis, pneumonia, or tuberculosis, usually bring about the termination. Petechial or conjunctival hemorrhages, epistaxis, and melena, are not uncommon.

Reference must be made to a group of cases of multilobular cirrhosis of the liver occurring in children, and often familial in character, in which various forms of disturbance of the central nervous system are present, giving rise to emotionalism, tremors, muscular rigidity, contractions, and mental symptoms. Signs of hepatic disease are usually absent during life. These cases are always fatal. The average duration is four years. They have been described by Gowers, Osmond, Hornen, and others. The most complete account of this type is that by Kissar Wilson. Wilson concludes that the nervous symptoms are due to the action of a toxin, probably the result of the hepatic disease, which operates selectively on certain parts of the brain, especially the lenticular nucleus (vide p. 847).

MORBID ANATOMY.—This does not differ materially from that in the adult form. The liver is paler than normal, and it is usually reduced in size. The surface is irregular and may be "lobulated." Some degree of perihepatitis is often present. The organ feels very hard, and the cut edges are sharp. On section, pale grey bands of fibrous tissue can be seen enclosing yellow areas of liver substance. Microscopically a coarse multilobular cirrhosis is found compressing and distorting the lobules. The liver cells show fatty and atrophic changes. In places groups of apparently normal liver cells represent attempts at regeneration. "Pseudo-bile-ductules" are often present; these are narrow sinuous columns of small cubical cells. The spleen is enlarged and fibrotic. The peritoneum is usually thickened and opaque.

The **DIAGNOSIS** of portal cirrhosis is usually difficult in the absence of a history of the administration of alcohol. In a large proportion of the cases the condition is first recognized post mortem. The diagnosis of the disease must depend chiefly on the presence of signs of portal obstruction, such as ascites, distension of abdominal veins, and hematemesis. It is most liable to be mistaken for tuberculous peritonitis. Examination of the ascitic fluid is of value, as in tuberculous peritonitis the number of lymphocytes is largely increased.

A rare condition which may resemble cirrhosis, as it leads to enlargement of the liver and ascites, is that known as "primary occlusion of the ostia of the hepatic veins." Several instances of this have been recorded in children.

PROGNOSIS.—This is unfavourable, as the condition is incurable, and a child affected thereby must be regarded as in a *pessimum* state even if a collateral circulation becomes established, as shown by the absence or diminution of ascites. The child is specially liable to tuberculous infection, or some intercurrent acute illness may cause a fatal termination in early life.

TREATMENT.—The treatment of the condition in children does not differ in any essential point from that in adults.

2. **Mono- or Inter-lobular Cirrhosis.**—The two chief conditions in which a mono- or inter-lobular cirrhosis occurs are—(a) Biliary cirrhosis, also known as "Hanot's cirrhosis" and "hypertrophic cirrhosis"; (b) cirrhosis due to chronic obstruction of the larger bile ducts.

(a) **BILIARY CIRRHOSIS.**—Biliary or Hanot's cirrhosis must be regarded as a rare condition, and, judging by the number of recorded cases, it would seem to be even less common in England than in other European countries.

The clinical features of the disease, first clearly defined by Hanot in 1874, are those of a chronic form of jaundice accompanied by enlargement of the liver and spleen and by intercurrent attacks of abdominal pain and pyrexia, but not associated with persistent ascites.

Ætiology.—In children the disease is commonest between five and fifteen years, though in India and Mexico a condition closely resembling Hanot's cirrhosis affects quite young children, and even infants.

Males are more liable than females; in a series of twenty-two cases collected by the writer, thirteen were male and nine female children. Neither alcohol nor syphilis appear to be important factors.

A number of cases have been recorded which have followed an infective fever, such as typhoid and scarlet fever.

Heredity.—A "familial" form of the disease has been described, in which several members of the same family have been affected. There is some risk of confusing with this the familial form of schlemic jaundice, which is a separate clinical condition.

Symptomatology.—The earliest symptoms are general malaise, headache, some abdominal pain, moderate pyrexia, and slight jaundice. Vomiting is not uncommon. The liver undergoes considerable uniform enlargement; the surface is smooth and moderately firm. The spleen also becomes enlarged; the younger the child, the greater is its relative increase in size, and it may become even larger than the liver. The jaundice becomes deeper, though it is rarely intense. The urine contains bile pigment, and bile pigment is also present in the stools, showing that bile is passing into the intestine and that the bile-ducts are not completely obstructed. A moderate leucocytosis is present during the febrile period. After a period varying in length from a few days to one or two weeks, the pyrexia and most of the symptoms subside, but the jaundice persists. One of the chief characteristics of the disease is the occurrence of exacerbations or "crises," which take place at intervals. During these "crises" the symptoms are renewed, and the liver and spleen become tender and exhibit further enlargement. A "crisis" lasts a few days, and then subsides, leaving the liver and the spleen larger than before its onset. Clubbing of the fingers, deformities of the nails, and thickening of the bones about the wrist and ankle-joints, resembling the condition known as "osteo-arthralgia," have been described in children. Arthritis is sometimes present. Brown pigmentation of the skin of the extremities, resembling that in Addison's disease, also occurs. In this as in other forms of cirrhosis, bodily development may be retarded, puberty become delayed, and a condition of infantilism may result.

The duration of a case of biliary cirrhosis is very variable; it may continue for several years, but generally speaking the course of the disease in children is more severe and of shorter duration than in adults. The crises tend to become more frequent, and the child gradually becomes weaker. Towards the close haemorrhages

in the skin or from the mucous membranes make their appearance. The case usually terminates in a comatose state resembling an *icterus gravis*, or from some intercurrent pulmonary disease.

Pethology.—The liver is considerably enlarged; the surface is smooth, though some roughening of the capsule due to perihepatitis is often present. The liver substance is firm, and the colour deep yellow or dark green. Microscopically the chief characteristic is a fibrosis with a monolobular or interlobular distribution, with fine fibrille of fibrous tissue invading the outer zone of the lobules. In a certain number of cases the cirrhosis does not follow a purely mono- or interlobular distribution, but may be partly multilobular in type, and these are therefore classed as "mixed" types. "Pseudo-bile-canaliculi," formed by sinuous columns of small cubical cells, are usually present in the fibrous tissue. The liver cells do not as a rule exhibit extensive degenerative changes. The bile-capillaries and small bile-ducts show distinct signs of catarrhal inflammation, and their lumens contain desquamated epithelium and plugs of inspissated bile. The larger bile-ducts are little, if at all, affected.

The spleen is enlarged and firm, the result of a diffuse fibrosis. The lymphatic glands, especially those in the hilum of the liver and spleen, are sometimes swollen and softened.

The cause of the disease is most probably some chronic form of infection, the nature of which is at present obscure. The recurrent exacerbations or "crises," the accompanying leucocytosis, and the enlargement of the lymphatic glands, are in favour of an infective origin. The changes found in the liver strongly suggest that the primary cause of the hepatic condition is a cholangitis, or inflammation of the smaller bile-ducts brought about by some irritant. This was the view originally advanced by Hanot. The question naturally arises as to how the irritant, whether of bacterial or non-bacterial origin, reaches the bile-ducts. Two routes are possible: (a) The irritant may ascend the bile-ducts from the intestine, setting up an ascending cholangitis; or (b) it may be conveyed to the smaller intrahepatic ducts by the general blood-supply, giving rise to a descending cholangitis. The evidence favours the second hypothesis—namely, that the irritant reaches the liver by the blood-stream; for there are no signs of inflammation of the larger bile-ducts, such as might be expected if an ascending cholangitis had occurred, and, further, the appearances presented by the smaller bile-ducts closely resemble that produced experimentally by the injection of toluylene diamine. The enlargement of the spleen, sometimes to a greater degree than of the liver, is also in favour of a general blood infection. As to the nature of the infection, nothing is definitely known at present.

Special Forms of Biliary Cirrhosis.—Mention must be made of certain forms of hepatic disease occurring in hot climates, notably in India and Mexico, resembling Hanot's cirrhosis. In India, native infants are frequently affected by a fatal form of jaundice with enlargement of the liver, which appears to be due to irritating or unsuitable articles of diet and bad hygienic conditions. The duration, both in India and Mexico, where it appears to be endemic, is much shorter than in Hanot's cirrhosis. The cirrhosis begins as a pericellular fibrosis, and later is interlobular in type. In the Mexican form the spleen is usually not enlarged, and ascites is often present.

(b) **OBSTRUCTIVE BILIARY CIRRHOSIS.**—A form of interlobular cirrhosis of the liver occurs also as the result of chronic obstruction of the large bile-ducts. Apart

from obliteration of the bile-ducts in infants, complete obstruction is a very rare event in childhood, so that this form of cirrhosis is hardly ever met with in children after the first year. The liver in cases of chronic obstruction of the extrahepatic bile-ducts is enlarged, hard, and has a dark olive-green colour. On section the bile-ducts are found distended and full of dark viscous bile. Microscopical examination shows an interlobular cirrhosis closely resembling that in Hanot's cirrhosis. In the more chronic cases the fibrosis is sometimes of a mixed type, in that it may have a multilobular as well as a monolobular distribution. "Pseudo-bile-canaliculi" are often present in the fibrous tissue. The smaller intrahepatic bile-ducts exhibit inflammatory changes. It is probable that this cholangitis and the associated cirrhosis are caused, as in Hanot's cirrhosis, by toxic bodies brought to the liver by the blood supply. It has been suggested that these bodies may be produced in the intestinal canal as a result of the absence of bile therein.

DIAGNOSIS OF BILIARY CIRRHOSIS.—This must depend on the presence of chronic jaundice of the non-obstructive type with enlargement of the liver and spleen, and the occurrence of exacerbations or "crises" attended by abdominal pain and pyrexia. Cases of splenic anaemia (Banti's type) in the later stage may resemble biliary cirrhosis, owing to the presence of a large liver and spleen, and occasionally jaundice. In such cases a leucopenia is usual, in contrast with the leucocytosis in the crises of biliary cirrhosis, and the jaundice is slighter.

In the condition known as "acholic jaundice," with enlarged spleen, the urine is bile-free, and the liver is in many cases little enlarged. There is an absence of pyrexia, pain, and leucocytosis. There is some degree of anaemia, and the red corpuscles may be unduly fragile. In syphilitic cirrhosis other evidence of syphilitic infection may be obtained.

3. Syphilitic Cirrhosis.—This will be discussed in the section on Syphilis of the Liver.

4. Cirrhosis in Chronic Cardiac Disease and Polyserositis (Polyserositis).—The condition known under various names, such as "inflammatory mediastino-pericarditis," "polyserositis," "polyserositis," etc., is separately described (see Chapter VIII, pp. 440 and 473).

In chronic venous engorgement due to cardiac disease the liver undergoes changes similar to those found in adults. It is usually enlarged and tender. Post mortem it has a dark purple colour; the surface is sometimes roughened, owing to some degree of perihepatitis. On section the characteristic "nutmeg" appearance is found. Microscopical examination shows some apparent increase of the fibrous tissue in the portal spaces, which is probably due to the atrophy of liver cells rendering existing fibrous tissue more obvious than normal (replacement fibrosis) rather than to an actual new formation of fibrous tissue.

In the condition known under various names, such as "inflammatory mediastino-pericarditis," "polyserositis," "polyserositis," "cardio-tuberculous cirrhosis," etc. (Chapter VIII, p. 473), the liver presents certain distinctive features which differ from those in ordinary cardiac disease; it is greatly enlarged, and ascites is usually present. Post mortem the capsule of the liver is thickened and shows numerous adhesions to neighbouring structures, or it may be covered with a thick white membrane, giving it the appearance described as "foul liver." On section the nutmeg appearance due to chronic venous engorgement is seen. Microscopically, in addition to a considerable amount of replacement fibrosis, there is usually present

a definite new formation of fibrous tissue. This fibrosis is well marked under the capsule, and may be present not only in the portal spaces, but also irregularly distributed throughout the liver. Some of these areas of fibrous tissue are probably the result of tuberculous infection, giant cells are occasionally present, but tubercle bacilli are not often found. The venous congestion of the liver resulting in these cases is much greater than in simple cardiac disease, and it has been suggested that this is due to dilatation of the hepatic veins, the result of inflammation spreading from the pericardium and involving their walls. Limitation of the movements of the diaphragm by pleural and peritoneal adhesions increase further the stasis in the liver.

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TUBERCULOSIS OF THE LIVER.

The possible routes by which tubercle bacilli may reach the liver are as follows: (1) The umbilical vein; (2) the hepatic artery; (3) the portal vein; (4) the lymphatics; (5) the bile-ducts.

Infection by the umbilical vein from the placenta during foetal life may theoretically take place, but examples of it are of extreme rarity. In acute miliary tuberculosis the hepatic artery is the channel, while the portal vein conveys the infection to the liver in abdominal tuberculosis. Tubercle bacilli may pass into the liver by the lymphatics from a tuberculous focus in its immediate vicinity, such as caseous glands in the hilum. The bile-ducts in all probability do not serve as channels for tubercle bacilli to reach the liver from the intestine, but if some part of the intrahepatic ducts becomes locally infected it is possible that the infection may spread along the biliary canals as a tuberculous cholangitis (vide p. 221).

As the result of one or other of these modes of infection, the following forms of tuberculous disease of the liver are found in children:

1. Miliary tuberculosis.
2. Massive form of tuberculosis.
3. Tuberculosis of the bile-ducts.

None of these forms have any clinical importance, though they possess considerable interest from the pathological aspect.

1. Miliary Tuberculosis.—This is the commonest form of tuberculous disease of the liver in children, and is usually due to a generalized systemic infection, bacilli being conveyed to the liver by the hepatic artery. The liver is enlarged and fatty; minute grey or yellow tubercles are visible in the capsule or scattered throughout the substance of the liver, resembling those found in the spleen, pleura, lungs, etc. Microscopically the tubercles are found to be most abundant within the lobules, and in the earliest stage consist of clusters of small round cells. If the duration of the case has been sufficiently long, giant cells and caseation may be present in their central parts.

Tubercle bacilli may be brought to the liver by the portal vein in cases of abdominal tuberculosis. Owing to the more chronic type of infection in these cases

the tubercles are more yellow and larger than in the case in generalized milary tuberculosis. They are abundant in the portal spaces, and where they are adjacent to a bile-duct they may cause a softening and breaking down of its wall, so that a small bile-stained caseous cavity results. These are sometimes described as tuberculous cavities, and will be referred to later under Tuberculosis of the Bile-Ducts.

Milary tuberculosis of the liver gives rise to no clinical signs of its presence beyond some increase in the size of the organ and very rarely slight jaundice.

2. Massive Tuberculosis.—Large caseous masses are occasionally found in the liver, and this form of tuberculosis appears to be more common in children than in adults, though at all ages it is of rare occurrence. The condition cannot be recognized during life unless the masses are superficially situated and of sufficient size to be palpated. They consist of white caseous material within a thin capsule. They are sometimes described as "solitary tubercle" of the liver, though the masses are usually multiple. Occasionally the central portion may soften and break down, forming an abscess cavity as the result of infection by pyogenic organisms. These caseous masses may resemble gummata, adenomata, or secondary nodules of malignant growth, but microscopical examination will reveal their true nature.

3. Tuberculosis of the Bile-Ducts.—Tuberculosis of the bile-ducts is not uncommon in children, though rare in adults. It is nearly always associated with some chronic form of abdominal tuberculosis, such as tuberculous peritonitis or intestinal ulceration. It cannot be said to be attended by any special clinical signs or symptoms. Jaundice is not present. Post mortem the liver is somewhat enlarged and fatty. Small greenish nodules are sometimes visible on the surface. On section numerous bile-stained cavities, surrounded by caseous material, are found in the portal spaces, as well as many yellow tubercles. The cavities vary considerably in size: some are no larger than a pin's head, while others are the size of a pea, or even larger than this.

The process of formation of the cavities is dependent on a tuberculous infection of the portal spaces of a chronic type. Extensive caseation takes place in these spaces, in the course of which the wall of the bile-duct therein becomes involved, and eventually gives way as the result of the inflammatory process attacking it from without, and a cavity is thus formed containing bile-stained caseous material and disorganized columnar epithelial cells. The lesion is a localized inflammation of the duct, beginning as a pericholangitis, and there is not sufficient evidence to prove that anything of the nature of a diffuse cholangitis is set up, as apart from these focal lesions the bile-ducts appear to be not involved.

On the other hand, Sergeant has shown experimentally that tubercle bacilli are unaffected by bile, so that it is possible that a localized infection of an intra-hepatic duct may give rise to a diffuse tuberculous cholangitis. The fact that jaundice is generally absent in these cases with tuberculous cavities in the liver is of importance; if a diffuse form of cholangitis were set up, jaundice most probably would be present. (For a further account of these tuberculous cavities of the liver, see Trans. Path. Soc. Lond., 1880, p. 158.)

SYPHILIS OF THE LIVER.

Syphilitic affections of the liver in children are practically always due to the congenital form of this disease. The route of infection is by the umbilical vein, and the syphilitic virus thus conveyed to the liver of the foetus produces changes generally diffused throughout that organ; whereas in the acquired form of syphilis the infection is brought to the liver by the hepatic artery, and gives rise to lesions having a more focal distribution. Further, as Hutinel and Hudele have pointed out, in the foetus the syphilitic infection is brought in contact with a liver the cells of which are rapidly developing and immature, and therefore less able to resist than in the adult.

Syphilis may be acquired by an infant suckled by an infected wet-nurse, and in such a case the liver may escape serious damage, or, if it be involved, the lesions produced will resemble in their character and distribution those of the acquired form in adults.

In the most severe forms of inherited syphilis, the infant is stillborn or survives birth for only a few days. As a rule the signs of congenital syphilitic disease of the liver make their appearance during the first few weeks or months of life, but sometimes, in what is known as "delayed" congenital syphilis, the signs of hepatic disease are postponed till late childhood or early adult life. This form will be considered later. In infancy the chief signs of syphilitic disease of the liver may be described as follows. Both the liver and spleen are enlarged and feel distinctly hard. As a general rule there is little abdominal tenderness, but in some cases pressure causes pain, and occasionally friction may be felt over the enlarged liver, due to the presence of peritonitis. In these cases ascites may be present, but this is a rare complication of the disease. Jaundice is not uncommon, and is due to inflammatory changes in the smaller intrahepatic bile-ducts. The jaundice may be present at birth, and persist for several weeks, and then gradually clear up, sometimes with remissions. In such cases bile is usually present in the stools.

Other evidences of congenital syphilis are generally manifest, such as impairment of nutrition, anaemia, skin eruptions, epiphyseitis, gastro-intestinal disturbance, etc.

In favourable cases the hepatic enlargement and the attendant symptoms subside under antisyphilitic treatment, and it is possible that in some cases the liver completely recovers. Rolleston has suggested that the resistance of the liver may be permanently impaired, and that there is a liability to a subsequent development of cirrhosis of the multilobular type.

MORBED ANATOMY.—The liver is large and heavy and feels tough. The colour varies considerably; sometimes it is dark red or purple, suggesting a condition of great congestion, while in other cases it is a mottled yellow. Delicate adhesions are sometimes present on the capsule.

On section it presents a curiously tough resistance to the knife. The cut surface is smooth, with clean-cut edges with a characteristic shiny appearance, not unlike that of a lardaceous liver. The section usually appears uniform or homogeneous, but sometimes minute greyish specks can be distinguished. These are described as "silvery punctata"; they are not caseous, however, but are little masses of cells or granulomata.

More rarely, circumscribed pale yellow areas are seen on the surface or in the substance of the liver which are caseous and are true gummata. They vary greatly in size, but may be as much as an inch in diameter, or even larger.

It should be borne in mind that not uncommonly the naked-eye appearance of a syphilitic liver shows little abnormality, though microscopical examination may reveal very extensive changes.

Microscopical Changes.—These vary to a considerable extent with the age of the child. Three main types can be distinguished, which represent stages in the progress of the disease, and probably depend on the severity of the infection. These types are as follows:

1. Diffuse infiltrative form.
2. Gummatous form.
3. Cirrhotic form.

Quite commonly more than one of these forms is present in the same specimen.

1. *Diffuse Infiltrative Form.*—This corresponds to the "embryonal infiltrative" form of Hutinel and Hodelo, and may be regarded as a diffuse hepatitis of special character. It is the earliest stage in the disease. There is a diffuse and dense infiltration with small round cells of the portal spaces and between or among the liver cells. These cells consist of leucocytes and proliferating connective-tissue cells. This small-celled infiltration ("diffuse gummatosis") may be so extensive as to resemble a sarcomatous growth, and it is probable that some cases described as diffuse sarcoma of the liver in infants are really syphilitic in origin. There is great vascularity of the organ. This diffuse infiltration is only found in infants who die shortly after birth.

2. *Gummatous Form.*—Sometimes definitely caseous gummatous areas are found in addition to the changes described above. They do not differ essentially from those found in acquired syphilis of the adult.

3. *Cirrhotic Form.*—Congenital syphilis causes a very typical form of hepatic cirrhosis. As a result of the proliferation of the connective-tissue cells, delicate fibrils of fibrous tissue are formed between the hepatic cells, thus giving rise to a pericellular cirrhosis, which is most marked in the periphery of the lobules. The hepatic cells become compressed, deformed, and degenerate. Numerous solitary gummata or granulomata formed by masses of small round cells are seen irregularly distributed throughout the section. Fibrous tissue is also found in the portal spaces. Pseudo-bile-canaliculi are usually present in considerable numbers. The development of fibrous tissue in some areas may be so extensive that the original structure of the liver has almost entirely vanished.

DIAGNOSIS.—This will depend on the history and the presence of other signs of syphilis, and as a rule does not present much difficulty. The presence of a positive Wassermann reaction and the beneficial result of mercurial treatment would confirm the diagnosis.

PROGNOSIS.—This depends largely on the severity of the manifestation of the disease and the state of general nutrition of the infant. The greater the enlargement of the liver and spleen, the worse the prognosis. Cases with ascites almost invariably are fatal, and jaundice is an unfavourable sign.

TREATMENT.—This consists in active antisyphilitic treatment, details of which are discussed elsewhere.

Delayed Form of Congenital Syphilis of the Liver (*Hérisé-Syphilis hépatique tardive*).—In the great majority of cases of inherited syphilis the signs of the disease manifest themselves in infancy, but sometimes the infection appears to be dormant or inactive until later childhood or early adult life (between the tenth and twentieth years). In such cases the liver becomes greatly enlarged, and the spleen to a less degree. Ascites is quite common, while jaundice is rare. Bodily growth is greatly impaired, and a form of infantilism may result. Other evidences of syphilitic infection are usually found, such as keratitis, Hutchinson's teeth, etc. Extensive arterial disease is sometimes present. Lardaceous degeneration and uræmia are not infrequent causes of death.

The changes in the liver closely resemble those of acquired syphilis grafted on to congenital syphilis. Gummata with scarring and peribepatitis are present in addition to the lesions characteristic of the ordinary form of congenital syphilitic cirrhosis of earlier life. The prognosis is unfavourable.

TUMOURS OF THE LIVER.

Malignant Growths.—Primary malignant disease of the liver is very rare, but both sarcoma and carcinoma occur, the former being the more common.

SARCOMA.—*Primary Sarcoma* may occur in early infancy, and gives rise to great enlargement of the liver, so that the abdomen becomes considerably distended. The surface of the liver is irregular or bossed. Jaundice, and less commonly ascites, may be present, and there is a gradual failure of general nutrition. As is sometimes the case in sarcoma of the kidney, owing to the rapid increase in size of the growth, there may be very little, if any, diminution of the body-weight, though considerable wasting takes place, owing to interference with the alimentary system. The growth generally forms a large mass occupying the greater part of the liver, though smaller nodules may be present. Microscopically it is usually of the small round-celled variety, and it may be so generally diffused throughout the liver substance as to resemble the diffuse small-celled infiltration sometimes found in congenital syphilis, which may be mistaken for sarcoma.

Secondary Sarcoma.—Deposits are occasionally found in the liver secondary to a primary growth in the kidney, suprarenal, testicle, or elsewhere.

CARCINOMA.—Primary carcinoma is much rarer than sarcoma. Only a few cases have been recorded in early life.

ENDOTHELIOBLASTOMA.—Growths may arise from the endothelium of the bloodvessels and lymphatics. A case of hæmangioma-endothelioma has recently been described by Veeder and Anson, in an infant of ten weeks in which the surface of the liver was covered with undelimited nodules.

INNOCENT TUMOURS.—ADENOMATA.—Adenomata are sometimes found in the liver of children either as single or multiple circumscribed masses, and may form definite projections on the surface. They may be derived (a) from the ordinary hepatic cells, (b) from the cells of the bile-ducts, (c) from suprarenal "rests" which have become included. Multiple adenomata are regarded as the result of a compensa-

tery hyperplasia on the part of the liver cells in certain forms of hepatic disease, such as cirrhosis and acute yellow atrophy (q.v.). Microscopically, multiple adenomata are found to be composed of healthy liver cells irregularly arranged.

NEVUS (ANGIOMA).—This form of innocent tumour is not uncommon, and in young children nevi of the liver may be much larger than those found in adults. They have no clinical importance.

MYXOMA, LIPOMA, FIBROMA, AND TERATOMA.—Examples of these rare forms of tumour have been described in children.

CYSTS.—There are three forms of cysts of the liver:

1. *Simple Cyst*, usually single, but sometimes multiple, containing clear, occasionally bile-stained, fluid. They are due to obstruction of a small bile-duct, and are retention cysts, resembling those found in the kidney.

2. *Congenital Cystic Disease of the Liver*.—In this condition the liver contains a large number of small cysts, sometimes of microscopical size. The liver as a rule is not enlarged in children, though a case has been recorded in which it was so large as to impede delivery. In the great majority of cases the kidneys also are affected by cystic disease.

In some of the recorded cases in children congenital malformations have been observed.

The cysts vary greatly in size, but are not as large in children as in adult cases. They are sometimes so small that they can be detected only by microscopic examination. They contain a clear fluid, in which are found proteins, urea, creatinin, cholesterolin, etc., and occasionally blood-pigment. The most important microscopical change is the presence in the portal spaces of numerous tubules, lined with a low columnar epithelium which resemble bile-ducts, but do not contain bile. There is also a considerable increase in the amount of fibrous tissue in the portal spaces and between the lobules.

As an explanation of the cause of cystic disease of the liver various hypotheses have been advanced. Shattock and Still regard the condition as dependent on a developmental fault or malformation of the liver, a view which is supported to some extent by the occasional presence of other deformities or congenital defects. For a full discussion of the pathology, reference should be made to Rolleston's "Diseases of the Liver," 1912, p. 452.

3. *Hydatid Cyst*.—Rolleston states that hydatid cyst of the liver is rare under fifteen years, and that it is probable that only 5 per cent. of the total number of cases occur under ten years of age. In this country the condition is distinctly rare in childhood. There is no special clinical distinction to be drawn between hydatid of the liver in the child and in the adult, except that the cysts do not attain the same relatively large size in the young, and are generally single.

The following case in a girl of ten, under the writer's care at the East London Hospital for Children, is worth recording. She was brought to the hospital for slight dyspepsia; a mass was found in the right hypochondrium and lumbar region, lying in the situation of the right kidney. It felt hard and slightly nodular, and was about the size of an orange. It was thought to be a renal tumour, probably a sarcoma. Laparotomy was performed, and the mass proved to be a hydatid cyst arising from the lower surface of the right lobe of the liver, from which it had almost completely separated itself. It had a very thick almost cartilaginous wall.

The cyst lay immediately in front of the right kidney. It was removed without difficulty after aspiration of the fluid, and the child made a complete recovery.

Symptoms may be entirely absent, or abdominal pain and slight jaundice may attract attention. Rupture of the cyst is not uncommon in children, owing to their active movements and liability to falls, and this may be followed by hydatid infection of the peritoneum, with the formation of multiple cysts.

The diagnosis is usually easy if the cyst projects from the surface of the liver and fluctuation can be detected; but if deeply situated or in the upper part of the liver it may be very difficult.

FATTY LIVER.

This is a very common form of enlargement of the liver in children.

In many pathological conditions in childhood the liver is found to contain an excess of the normal amount of fat. If the fat normally present in an hepatic cell is increased so that a large globule is formed, and the nucleus is displaced to the periphery of the cell, but is otherwise unaltered, the condition is known as *fatty infiltration*. In *fatty degeneration* the fat is formed at the expense of the protoplasm of the cell as a result of degeneration, and the nucleus has lost its staining power or is destroyed. Of these two varieties, *fatty infiltration* is the commoner in early childhood, though it is often difficult to distinguish between them.

Fatty infiltration is usually the result of some fault in alimentation, such as overfeeding, excessive carbohydrate diet, disordered states of the digestive tract, rickets, and wasting diseases.

Fatty degeneration is produced by toxic or infective conditions. Phosphorus and delayed chloroform-poisoning are examples of toxic causes, diphtheria and septicæmia of infective causes.

The fatty liver in tuberculosis may be due partly to the action of toxins, and partly to the disordered digestion and wasting generally associated with this infection.

The CLINICAL SIGNS of fatty infiltration are enlargement of the liver, with an absence of pain or tenderness. The surface is smooth, and the edge is readily felt in a thin child. The stools are often light-coloured. In fatty degeneration the liver is usually little, if at all, enlarged.

Post mortem the liver is uniformly enlarged. The surface is smooth and glistening. The colour is generally a pale yellow. In infective or toxic conditions this pale yellow colour is often not universal, but is present in patches. The cut surface is smooth and greenish, and the liver substance is soft and friable.

The chief microscopical changes consist in the presence of fat globules filling the liver cells; in the toxic and infective conditions the nuclei of the cells are degenerate, and in advanced cases little can be seen but the network of fat cells.

The PROGNOSIS must depend on the cause of the condition, and as to whether or not this can be removed by treatment.

As Guthrie and others have pointed out, the presence of a fatty liver is a special danger to patients who have to undergo chloroform anaesthesia, as there is a liability to the onset of symptoms resembling tetanus gravis, with a fatal termination.

LARDACEOUS DISEASE.

Lardaceous disease is the result of chronic suppuration and of syphilis. Tuberculous disease of the hip and spine are the commonest surgical causes in children; to these must be added chronic empyema and bronchiectasis. Since the introduction of aseptic surgery, the frequency of lardaceous disease has very greatly diminished.

The CLINICAL EVIDENCES of the disease are as follows:—Increasing pallor of a peculiar waxy tint, and often some puffiness of the eyelids. The abdomen gradually becomes more prominent, due to the increase in size of the liver and to the presence of ascites in the later stages. The liver becomes greatly enlarged, and is painless. The lower edge may extend well below the navel, and feels hard and resistant. Jaundice is usually absent. The urine is pale and limpid and increased in amount, it has a low specific gravity, and contains much albumen and hyaline casts. The spleen is also enlarged.

Wasting is progressive. Oedema of the legs sets in and steadily increases, and the child dies of exhaustion, often hastened by diarrhoea or vomiting.

Post mortem the liver is very large, heavy, and firm, with a smooth surface. On section the cut surface resembles bacon fat and has a curiously smooth, glistening appearance, and gives the characteristic staining reaction with an iodine solution.

Microscopically the earliest change is a deposition of lardaceous material in the capillaries of the median zone of the lobules, and later in the interlobular vessels. In syphilitic cases the capillaries and small arteries are affected mostly in the neighbourhood of gummata.

PROGNOSIS.—This must always be regarded as unfavourable. By far the greater number of cases in which the condition has been recognized are suffering from some severe and incurable form of debilitating disease. The development of lardaceous disease in such cases is of serious import, as a fatal termination is most probable. Occasionally cases are seen with advanced lardaceous disease, due to some chronic disease of bone, in which, after suppuration has ceased, the greatly enlarged liver returns to its normal size, oedema and albuminuria disappear, and the child apparently recovers. It would be of great interest to learn what is the final condition of the liver in these cases of seeming recovery.

The DIAGNOSIS of the condition is usually easy, owing to the presence of chronic suppuration or of congenital syphilis.

ABSCESS OF THE LIVER.

Abscesses in the liver are either single or multiple; in children they are generally multiple. The tropical form of abscess is rare in children even in countries where dysentery is prevalent, and need not be considered further. A single abscess may be caused by a suppurating hydatid cyst, and is occasionally found both in portal and general pyæmia; it is sometimes produced by the coalescence of several smaller ones. Cases of single abscess are said also to occur as the result of injury.

Multiple abscesses are due to infection conveyed to the liver by the portal vein, the hepatic artery, or the bile-ducts. The source of the portal infection is usually

some suppurative condition, such as umbilical infection, appendix abscess, or intestinal ulceration, etc. Pyogenic abscesses are due to infection brought to the liver by the hepatic artery. Inflammation of the bile-ducts, especially suppurative cholangitis, may also lead to abscesses. A number of cases have been recorded in which such inflammation has been set up by the presence of roundworms in the duct.

SYMPTOMATOLOGY.—The child is acutely ill, and usually complains of pain and tenderness in the hepatic region and in the right shoulder, but the abscess may be latent and cause no definite symptoms. Pyrexia of the hectic type, with rigors, sweating, and vomiting, are common. A moderate degree of jaundice is frequently present. Severe abdominal pain and tenderness are constant symptoms; the pain may be localized in the hepatic area, or be referred to other parts of the abdomen and to the right side of the chest or shoulder. Pleurisy may be set up by extension of the inflammation through the diaphragm. The liver is enlarged and tender, and generally the surface is smooth, though large superficial abscesses may occasionally be recognized by palpation. In a case recently under the writer's care the lower edge of the liver extended halfway between the navel and the pubes, and on the surface numerous small nodules, the size of peas, were definitely felt, which subsequently proved to be abscesses projecting under the capsule.

Most cases of abscess of the liver in children terminate fatally, as they are usually multiple and the result of portal or general pyæmia. If the abscess is single and of considerable size, the condition may be recognized and relieved by surgical treatment, or it may rupture into the pleura, lung, or peritoneum.

In cases due to portal vein infection (pylephlebitis), the liver is usually found to contain abscess cavities so numerous that it appears to be riddled by them. The cavities have ragged irregular walls, lined by shaggy flakes of fibrin and pus, and larger cavities are formed by the coalescence of several small ones. In other cases the abscesses form small spherical cavities.

In tuberculosis of the liver, small cavities are sometimes met with containing granular bile-stained debris, which are elsewhere described. These might be mistaken for small abscesses, but are in reality due to localized tuberculosis of the bile-ducts.

The **DIAGNOSIS** of hepatic abscess depends on the presence of rigors, jaundice, hepatic pain, and enlargement, and is further aided by the presence of other signs of pyæmia or of some abdominal suppurative or intestinal ulceration.

The **PROGNOSIS** must be regarded as extremely grave in the pyogenic form, and a favourable result is only likely to take place as the result of opening and draining the cavity in the single form.

DISEASES OF THE GALL-BLADDER AND BILE-DUCTS.

ABNORMALITIES.—The gall-bladder may be completely absent, while the bile-ducts are normal. Absence of the gall-bladder as well as of the bile-ducts is probably the result of intra-uterine inflammation. Instances have been recorded of double gall bladder, each having a cystic duct.

The gall-bladder is occasionally found to the left of the hepatic duct fissure, so that it lies under the left lobe (Rolleston).

ACUTE INFLAMMATORY CONDITIONS of the gall-bladder and bile-ducts are far less common in children than in adults. They are usually caused by microbial infection from the intestinal tract, and infection may also take place by the blood-supply and the liver. Post-typhoidal cholangitis may occur in young children, though cases are not common.

A number of cases have been recorded in which acute suppurative inflammation of the bile-ducts and gall-bladder has been set up by the presence of roundworms which have entered the bile-passages from the intestine. Abscess of the liver may result in such cases.

The symptoms of acute cholangitis may be stated briefly to consist of pain and tenderness in the right hypochondrium, with pyrexia and usually jaundice. The treatment is drainage of the gall-bladder.

Chronic forms of cholangitis, apart from tuberculosis, are uncommon in childhood, and comparatively few cases have been described. In most of these the cause appears obscure. The condition leads to a great thickening of the walls of the bile-ducts, some of which become considerably dilated. Jaundice may be absent. Fig. 17 illustrates the condition.

Cholelithiasis.—It is hardly necessary to do more than point out that gallstones, though very rare in children, are occasionally found at all ages. Still in a series of twenty-three cases, found biliary calculi present in ten infants who were either still-born or who died a few weeks after birth. In infants the calculi are numerous and form very small black, friable, irregular concretions, which are probably formed during intra-uterine life. They give rise to no symptoms, and it is doubtful whether they possess much clinical or pathological significance. They are probably due to stagnation and inspissation of the bile. Similar calculi are not found in older children.

The rare cases of gallstones in later childhood present no clinical distinction from those in adult life, and are attended by the same symptoms and complications.

REFERENCE.

ROLLISTON: Diseases of the Liver. 2nd ed., 1912.



FIG. 17. — SECTION THROUGH THE LIVER OF A CHILD OF THREE AND A HALF YEARS, SHOWING CHRONIC PERICHOLANGITIS.

The bile-ducts are greatly thickened, and some are dilated, and form cyst-like spaces. This case was not tuberculous. (Case described by the writer, Trans. Path. Soc. Lond., 1901, 12, 193.)

CONGENITAL OBLITERATION OR ATRESIA OF THE BILE-DUCTS.

This condition, not so uncommon as was at one time believed, was first fully described by John Thomson in 1892, with a collected series of forty-nine cases, and since that date many other cases have been recorded.

Ætiology.—Little is known as to the ætiological factors, but in the recorded cases the condition is more common in the male than the female sex. It is possible that heredity may have some influence, as a few cases have been described in members of the same family. Several cases have been recorded in association with other malformations, such as transposition of the viscera. In the great majority of cases there is no evidence of syphilitic infection. Congenital syphilis may, however, lead to a form of atresia of the bile ducts.

SYMPTOMATOLOGY.—The first symptom is jaundice. This usually appears in the first week, though occasionally it has been present at birth. In some cases the jaundice is not noticed until as late as the third or fourth week. At first very slight in degree, the jaundice gradually deepens, but it rarely assumes the deep olive-green tint so often seen in adults with complete obstruction of the common bile duct. The jaundice not uncommonly varies in intensity, but never completely disappears. The urine contains bile-pigment, and stains the napkins. The coloration of the urine, like that of the skin, at times may vary considerably in degree. The first stools passed after birth usually resemble normal meconium, but subsequent motions are free from bile, and resemble boiled white of egg; they are offensive and often dry and constipated. Occasionally they are greenish in colour as the result of the administration of mercury or of bacterial action. The state of bodily nutrition is at first good, and may continue so for weeks, or even months, but eventually progressive wasting sets in, and some slight intercurrent affection causes a sudden and often unexpected termination. A convulsive attack, possibly of choleraic origin, may be the final event. On examination of the infant, considerable enlargement of the liver is found to be present. The lower edge may reach below the level of the navel, and the organ feels hard and smooth. The spleen also is usually definitely enlarged. Some cases run a much shorter course than that described above, and in these during the first week hæmorrhages appear in the skin, and blood comes from the navel; hæmatemesis and hæmorrhage from the bowel may also occur. The infant rapidly sinks, and dies in the course of a few days.

The duration of the disease is very variable. So far as is known, the condition is always fatal. In hæmorrhagic cases death occurs at an early stage, from a few days up to two weeks. In the more common form life is prolonged for several weeks, or even months. In no proved case of this condition has the infant lived more than eleven months (Thomson).

PATHOLOGY.—Post-mortem examination shows various abnormalities of the bile ducts and gall bladder. In some cases these structures are completely or in part absent. In others, the common bile duct or both ducts are represented by a thin filamentous cord of fibrous tissue entirely devoid of any trace of a lumen; or the lumen may be found to be greatly narrowed or obliterated, either locally or generally, as the result of an inflammatory process.

The liver is enlarged and hard, and has the dark olive-green colour characteristic of chronic obstruction of the bile ducts. Microscopically, it exhibits a definite cirrhosis, chiefly of the monolobular type, as in biliary cirrhosis, and, to a less degree, a multilobular cirrhosis. The bile capillaries contain plugs of inspissated bile. The liver cells show little, if any, sign of degeneration. In infants who have lived for some weeks the gall bladder contains colourless mucus. The spleen shows a moderate degree of fibrosis, and in some cases the pancreas is similarly affected.

The causation of the changes in the bile ducts is a matter still under discussion.

They cannot be accounted for as a mere developmental error or malformation, as there is no doubt that they are the result of inflammation. Thomson explains the process as originating in a congenital narrowing of the lumen of the common duct, which produces a partial obstruction to the flow of bile, and retention of irritating products when the liver takes on its full function after birth. This sets up a catarrh, and ultimately a cholangitis, which eventually leads to the complete occlusion of the duct as the result of inflammatory action. The hepatic cirrhosis he attributes to the cholangitis.

Rollston suggests that the process is as follows: Toxic substances in the maternal blood are conveyed to the fetal liver by the umbilical vein, and also by the hepatic artery, and set up a cholangitis, and eventually hepatic cirrhosis. The inflammation, extending to the bile-ducts as a descending cholangitis, produces the obliterative changes therein. The narrow calibre of the ducts in infancy renders them especially liable to obstruction.

This explanation presents less difficulty than that of Thomson, and appears to be better supported by the histological changes found in the fully investigated cases. It seems probable, however, that more than one pathological condition is concerned, and that all cases of atresia of the bile-ducts are not due to the same process.

DIAGNOSIS.—This depends on the presence of chronic obstructive jaundice, dating from, or shortly after, birth, not infrequently accompanied by cutaneous, gastric, and intestinal hemorrhages. The duration of the jaundice, the bile-free stools, and dark urine, should early arouse suspicion that the case is not one of simple icterus neonatorum.

TREATMENT.—This should be directed to the maintenance of nutrition. Mercurial treatment should be employed, as there is a possibility that the case may belong to the small group associated with congenital syphilis, examples of which have been recorded by Rollston and others. Surgical treatment, with the view of re-establishing communication between the liver and the intestine, at present holds out little or no hope of success, though this would seem to be the only procedure which could be expected to give relief or cure.

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DISEASES OF THE PANCREAS.

PANCREATITIS :

ACUTE.

CHRONIC.

TUBERCULOSIS.

TUMORS.

CYSTA.

PARASITES.

It is difficult, if not impossible, to give satisfactory figures as to the weight of the pancreas at different periods in the development of the child. Just as the weights of infants of the same age may widely differ, so we find great variations in the weight of the pancreas at the same periods.

The following figures are quoted from Hutinel and Nobécourt to illustrate this point:

Age	Weight of Pancreas in Grammes
5 days	5
2 months, 2 cases	1.60 and 1.50
3 "	4.50
4 " 2 cases	2.00 and 3
5 " 2 "	8 and 17
6 "	13
8 " 3 cases	2.50, 10, and 15
9 " 2 "	15 and 40
12 " 2 "	15 and 20
1 to 2 years, 6 cases	12, 13, 15, 22, 25, and 30
3 years	20
7 "	40

Briefly, we may say that at birth the pancreas weighs between 5 and 6 grammes, at six months 10 grammes, and at twelve months 15 to 20 grammes.

The functions of the pancreas in the infant are similar in kind to those in the adult, but differ in importance. In the child, as in the adult, the pancreas produces both an external and an internal secretion, and disorders of both these secretions are to be found. So far as is known, the internal secretion, with its special function in relation to carbohydrate metabolism, is the same in early as in later life, and it may be affected by changes produced in the islands of Langerhans, such as may occur in the acute and chronic forms of pancreatitis and atrophic conditions with the production of glycosuria.

The external secretion of the infant's pancreas differs considerably from that of the adult in the activity of the ferments it contains. The amylolytic ferment is absent at birth, or very feebly active; it becomes definitely present between the second and third month, and is fairly active by the sixth month; but it does not develop its full power till the end of the first year.

The fat-splitting ferment or lipase, steapsin, is present in the secretion at birth, and is then in full activity. Trypsinogen can be found in the fetal pancreas at the fifth month. Trypsin is present in the pancreatic juice very shortly after birth.

Of these three important ferments normally present in the secretion, it would seem that, particularly in children, the lipase steapsin is the one most liable to show variations in its functional activity as the result of disease of the pancreas. Disturbance of this function of the gland leads to the presence of abnormal quantities of neutral fats in the feces, giving rise to the so-called "fatty stools," or "steatorrhea," as it is termed by some authors. It is advisable to restrict the use of the name "steatorrhea" to the passage of liquid fat with the stools.

The proteolytic ferment trypsin is less commonly affected in pancreatic disease. Evidence of defective action may be afforded by the presence in the feces of numerous muscle fibres not exhibiting signs of partial digestion, and even retaining their striation. This condition is sometimes called "anotorrhea." The proteolytic action of the pancreas may be tested by Sallé's method. In this, iodoforms (0.15 gramme for children) is administered in gelatine capsules treated with formaldehyde to enable them to resist gastric digestion for a prolonged period. They are readily digested in the intestine, with the result that iodine should be detected in the urine or saliva in about five hours in a normal person. If this

period be exceeded, it would point to a reduced activity of the ptecolytic action of the pancreatic secretion. The clinical value of this test is, however, doubtful.

In addition to these clinical manifestations of disturbance of the pancreatic functions, we must include a form of infantilism, associated with fatty stools, described by Byrom Beaurell.

Complete absence of pancreatic secretion by obstruction of the pancreatic duct is extremely rare in children.

PANCREATITIS.

Both acute and chronic forms occur in children.

Acute Pancreatitis.—Although a rare complication of that disease, acute pancreatitis is most likely to be met with in children in connection with mumps, and this variety will be first described.

The frequency of occurrence of pancreatitis in mumps varies in different epidemics. Thus, Edgcombe met with five cases in an epidemic of thirty-three cases, Fales twelve in fifty-eight, Pick four in twenty. The onset of abdominal symptoms is usually within a week of the appearance of the parotid swelling, in the earliest on the second day, and in the latest on the eighth day.

The clinical history of a case may be described as follows: Four or five days after the usual parotid manifestations, the child is seized with abdominal pain, accompanied by vomiting, and not infrequently by diarrhoea. The pain is usually located in the epigastrium, but may be felt in either the right or left hypochondrium, and in one case (Barbieri's) is described as radiating round the right lumbar region to the spine at the level of the first and second lumbar vertebrae.

Fever is not a prominent feature, though the temperature is sometimes slightly raised. Neurath has drawn attention to the fact that in the cases observed by him the frequency of the pulse was not increased, and was even slower than normal. This is important, as in the other acute abdominal conditions, such as peritonitis, the pulse-rate is usually increased, and therefore the occurrence of bradycardia would be a valuable sign in the differential diagnosis.

It has been suggested that the vomiting, which may be very severe, is due to pressure excited by the swollen pancreas on the semilunar ganglion and solar plexus.

Palpation of the abdomen shows a distinctly localized area of tenderness in the epigastrium or left hypochondrium, 1 to 2 inches above the level of the umbilicus, and occasionally a diffuse resistance can be made out in this situation.

The urine in only one case in a child has been definitely described as containing sugar. In this case, recorded by Barbieri, a boy of six years, abdominal pain set in six days after the appearance of mumps, and thirst, increased appetite and polyuria, soon followed. The urine was found to contain sugar, which disappeared in twenty-five days after the onset of the parotid swelling. Complete recovery followed. Further observations are wanted on the condition of the urine in cases of abdominal pain complicating mumps. Cambridge's reaction has been found positive in the urine of several cases of mumps with abdominal symptoms, thus affording confirmatory evidence of the existence of pancreatitis.

The stools are sometimes constipated, though more commonly they are loose and frequent, and contain fat globules. In one case Neurath found an absence of trypsin.

The course of acute pancreatitis with mumps is favourable. The abdominal symptoms may be severe for two or three days, and then gradually subside. Recovery usually takes place in seven to eight days after the onset. The question

has been raised as to whether this form of pancreatitis may be followed by fibrosis of the pancreas, analogous to the testicular fibrosis and atrophy which may result from orchitis in mumps. There is no evidence on this point, but it would seem improbable that the pancreas should be more liable to undergo fibroid change after mumps than the parotid, which in many respects it so closely resembles.

Little is known as to the morbid changes present in the pancreas, as in the cases recorded in children the condition appears to have always had a favourable termination. In an autopsy on a case in a young adult, accompanied by jaundice and leucæmia, Lemaitre found inflammation of the pancreas causing swelling of the gland and pressure on the common bile-duct.

Mention must be made of cases of abdominal pain and tenderness resembling in all respects this form of pancreatitis which have been met with in epidemics of mumps, but in which the parotid glands have been apparently unaffected. These have been described as "pancitis sine parotide."

It is possible that some cases of diabetes may date their origin from inflammatory changes brought about in the pancreas by an attack of mumps.

Acute pancreatitis is rare in children apart from mumps. It may arise as the result of infection from the intestine passing up the pancreatic duct. It is not uncommon in acute inflammatory conditions of the intestinal tract in children to find some swelling and hyperæmia of the pancreatic duct. Acute pancreatitis may be also caused by a blood infection; it has been met with in smallpox of the malignant type, in scarlet fever, and diphtheria. Both hæmorrhagic and suppurative forms have been described. Several cases have been recorded in which acute inflammation has been caused by the presence of ascidides in the pancreatic duct. The symptoms of acute pancreatitis are severe abdominal pain, vomiting, and collapse. Death has usually occurred in a few hours. Post mortem the pancreas is swollen, and feels denser and harder than normal. Microscopically, the gland shows signs of acute inflammatory changes. Hæmorrhage into the substance of the gland or abscess formation may have taken place. In infants and children fat necrosis is relatively far rarer in acute lesions of the pancreas than it is in adults. The diagnosis of acute pancreatitis in children, except as a complication of mumps, must be difficult or impossible, as there is nothing characteristic beyond localized abdominal pain and tenderness. In pancreatitis with mumps no treatment is required beyond hot or cold applications to the abdomen. Fortunately, cases of pancreatitis in children apart from mumps are extremely rare, and in these surgical treatment by laparotomy and drainage would appear to afford the only possible chance of recovery.

Chronic Pancreatitis.—Chronic pancreatitis or fibrosis of the pancreas is not very rare in young children, and is most commonly the result of congenital syphilis. It is also found in cases of chronic tuberculosis, in diabetes mellitus, and associated with Banti's disease.

Syphilitic changes in the pancreas have been found in the foetus as early as the fifth month, and are often present in infants suffering from marasmus, or exhibiting definite signs of congenital syphilis. Rolleston has described syphilitic disease of the pancreas in an infant with obliteration of the bile-ducts. Syphilis usually leads to a diffuse fibrosis of the pancreas, but cases of milium gummata have been described. The gland is affected in some degree in quite a large proportion of cases of congenital syphilis; Birch-Hirschfeld puts it as high as 43 per cent.

The pancreas is hard and swollen, and on section, to the naked eye, may have lost the characteristic acinus appearance. Microscopically, there is evidence of

a diffuse pancreatitis, as shown by the presence of an interlobular, interacinous, and intercellular fibrosis. The arteries are affected. Gummata, either large or small, are very rare in children. It is interesting to note that, although syphilitic fibrosis of the pancreas is so extensive that the acini may have almost disappeared, the islands of Langerhans are not involved, and appear quite normal. This will explain the fact that glycosuria does not occur in syphilitic disease of the pancreas in children. The chief signs directly due to the pancreatic fibrosis are changes in the character of the stools, which may contain an excessive quantity of fat.

TUBERCULOSIS.

Tuberculosis of the pancreas is commoner in childhood than in later life. The infection in the majority of cases is conveyed by the blood-supply, and gives rise to the formation of miliary tubercles. It is often difficult to recognize the presence of tubercles in the pancreas by the naked eye, owing to the pale yellow colour of the glandular tissue, and this probably accounts for the reputed infrequency of their occurrence. Microscopical examination will frequently reveal their presence in cases in which, macroscopically, no tubercles are visible. The tubercles are usually situated in the acini of the gland. Kudrowetsky states that pancreatic tuberculosis is present in 44·4 per cent. of tuberculous children.

The pancreas is also sometimes infected directly from contiguous tuberculous glands, and in such cases caseous masses or abscesses may be formed. In cases of chronic tuberculosis some degree of pancreatic fibrosis may result without the formation of tubercles in the gland, and it has been suggested by Carnot that this is the result of the action of a tuberculous toxin present in the pancreatic secretion. So far as is known at present, tuberculosis of the pancreas has no clinical importance.

TUMOURS.

Tumours of the pancreas, both innocent and malignant, are so extremely rare in children that it is not necessary to say more of these pathological curiosities than that cases of primary carcinoma and sarcoma have been recorded in quite young children. Secondary sarcoma is less rare.

CYSTS.

Retention cysts are occasionally met with, due to occlusion of one of the branches of the pancreatic duct. Localized or encapsulated collections of fluid in close relation to the gland are sometimes classed as pancreatic cysts, but are strictly speaking, peri-pancreatic. They may be the result of a limited peritonitis. Multiple cysts have been found in the fetus. Hydatid cysts must also be mentioned. From their clinical aspect cysts of the pancreas in children do not present any special features beyond those found in adults. The treatment is incision and drainage; exploratory puncture is dangerous.

PARASITES.

Several cases have been recorded of the presence of small *Ascarides lumbricoides* in the pancreatic duct and pancreas, giving rise to an acute pancreatitis.

DISEASES OF THE PERITONEUM.

PERITONITIS:

ACUTE NON-TUBERCULOUS FORMS.
CHRONIC PERITONITIS.
TUBERCULOUS PERITONITIS: ACUTE
AND CHRONIC.

ACUTE.

TUMORS OF THE PERITONEUM.
CYSTS OF THE PERITONEUM.

PERITONITIS.

Many of the varieties of peritonitis met with in adults may occur also in children, but the relative frequency is strikingly different. For example, the tuberculous and pneumococcal varieties are much commoner in children than in adults. This greater frequency may be due either to a higher susceptibility or to a greater liability to these infections in the young.

Seeing that tuberculous peritonitis is by far the commonest and most important form of peritonitis in childhood, it will be considered separately, and the following classification of the varieties of peritonitis will be adopted:

- I. Acute non-tuberculous forms of peritonitis.
- II. Chronic peritonitis.
- III. Tuberculous peritonitis, acute and chronic.

I. Non-Tuberculous Forms of Peritonitis.

These may occur at any age in childhood, even in newly-born infants, and tend for the most part to run an acute course. They may be classified according to the nature of the infection causing them. Thus there are—(a) The pneumococcal form; (b) the gonococcal; (c) those due to streptococcal, staphylococcal, or *Bacillus coli* infections.

ÆTIOLOGY.—The conditions giving rise to acute peritonitis in children may be briefly stated as follows:

1. Umbilical infection in the newly-born, usually a streptococcal infection.
2. The specific fevers, especially scarlet fever, rheumatic fever, and septicæmia.
3. Pneumonia and pleurisy, or chronic lesions associated with the pneumococci, such as otitis media or partially healed empyema.
4. Infection from the intestinal canal, as in appendicitis, perforation of the bowel, strangulated hernia, vulvitis, intussusception. The causal organisms may be streptococci, *Bacillus coli*, staphylococci, or a mixed infection.
5. Vulvo-vaginitis associated with the gonococcus.
6. Blebs on the abdomen, perforating wounds, laparotomy.
7. Congenital syphilis.

SYMPTOMATOLOGY.—Since all forms of acute peritonitis in children present many features in common, it will be convenient to give a general account of the clinical manifestations met with in this condition. The onset is usually sudden, with pyrexia, abdominal pain, and tenderness, though in very young infants the abdominal symptoms may be ill-defined. The tongue is dry. Vomiting occurs early, and may be most distressing. At first the vomited matter consists of the stomach contents; later, of bile and mucus. The pulse is small and frequent. In most cases constipation is present, though in the pneumococcal form diarrhoea

frequently occurs. There is great restlessness, thirst, and often hicough. Retention and often suppression of urine may occur. The abdomen is at first retracted and rigid, with little or no respiratory movement, but soon becomes distended and tympanitic, with a shiny appearance of the skin. The intestinal distension leads to a diminution or absence of the liver dulness, and causes embarrassment of respiration by upward pressure on the diaphragm. Although free fluid may be present, it is masked by the tympanites, and there may be no sign of its presence beyond some dulness in the flanks. Examination of the blood generally shows a high leucocytosis, though this may be absent, or even a leucopenia may be found in those acute or fulminating cases in which there is little or no reaction to the infection. In the acute tuberculous variety also leucocytosis may be absent. The face early develops the characteristic "abdominal" expression, known as the "Hippocratic facies"—the eyes sunken, the features pinched and drawn. The child gradually sinks into a torpid condition, due to the increasing toxæmia, though in many cases consciousness is retained till the end. The abdominal pain and tenderness not infrequently diminish greatly towards the termination. In infants and in severe fulminating cases death occurs early (in two to three days); in the usual type and in older children the duration is from five to seven days. In favourable cases the inflammatory process may resolve or become confined with localized abscess formation.

MORBID ANATOMY.—In acute or fulminating cases the signs of peritoneal inflammation are much less marked than in the ordinary type. The peritoneum is hyperæmic, has lost its normal lustre, and is dull and opaque. Petechial hemorrhages may be present. The surface is slightly roughened, and fine fibrinous particles or filaments are adherent to it. There is usually a small quantity of turbid ascitic fluid, rich in cells. In cases of longer duration the appearances will depend on the nature of the infection. Thus, in the pneumococcal form, greenish-yellow pus, usually odourless, will be found; in the streptococcal and *Bacillus coli* infections the pus has usually a dirty grey colour, and is often offensive.

The liver and kidneys exhibit cloudy swelling or fatty change, and the spleen is usually large and soft.

PROGNOSIS.—In all forms of acute peritonitis occurring in children the prognosis is extremely grave. In infants and weakly children acute peritonitis is almost invariably fatal. The prognosis depends on the nature of the infection. Thus the gonococcal and pneumococcal forms give the best chances of life; a streptococcal infection is the most fatal.

The prognosis is also largely affected by the source of the infection. If the peritonitis is due to some localized lesion of the bowel or other organ, which can be dealt with by surgical measures, the infection may be limited. The longer the duration of a case of acute peritonitis, the more hope may be entertained of recovery, as there is a possibility that the generalized inflammation may gradually subside or become localized.

DIAGNOSIS.—As a general rule, this is not difficult, except in infants in whom, as has been already stated, the abdominal condition may be latent. The most common cause of peritonitis in children, apart from the tuberculous form, is appendicitis.

Thoracic conditions, such as pneumonia, pleurisy, pericarditis, may closely simulate acute peritonitis, and careful examination of the chest should always be made. Intestinal colic may be accompanied by vomiting, and the pain is often

very acute, but is usually intermittent, and the examination of the abdomen between the paroxysms will generally reveal little or no resistance or great tenderness, and no ascites. The pulse is not so rapid or thready as in peritonitis. Further, the absence of a high leucocytosis in such a case would practically exclude peritonitis. In doubtful cases rectal examination should always be made, as an intussusception, or some localized resistance or tenderness, may be detected by this means. The presence of blood and mucus in the stools would suggest the presence of intussusception, but these often occur in the diarrhoea of acute enterocolitis accompanied by much straining and tenesmus.

Pneumococcal Peritonitis.—Ætiology.—This is much more frequent in females than males. In a series of 234 recorded cases in children, collected by Ewing, 62 (or 27 per cent.) were males, and 172 (or 73 per cent.) females. It is difficult to account for this preponderance in the liability of females. Infection from the female genitalia cannot account for it, as in cases in which purulent vaginal discharges have existed the pneumococcus has not often been found. Further, in fatal cases the Fallopian tubes do not show greater signs of inflammation than in other forms of peritonitis. The explanation that female children may offer a lower resistance than males to pneumococcal infection seems improbable, as inequality of liability between the sexes does not exist in other forms of this infection.

Various explanations have been given as to the channels by which the peritonium becomes infected. The possible routes of infection are the blood, the lymphatics, the intestinal canal, and the Fallopian tubes or spermatic cord.

There can be little doubt that in most cases the infection is conveyed by the blood, and that the peritonitis is the result of a septicæmia associated either with an acute disease, such as pneumonia or pleurisy, or with a pneumococcal focus, such as a partially healed empyema, a chronic inflammation of the naso-pharynx, or an otitis media. Bowen and Arnold, and others, consider that the infection is derived from the intestinal tract. There is no definite evidence in support of this view, except the clinical fact that many cases of pneumococcal peritonitis are attended by diarrhoea, and that local lesions, such as follicular ulcers, occasionally have been found. The mesenteric glands in such cases do not show any obvious change, such as swelling, which might be expected if the infection arose from the intestinal canal. It would seem probable that the explanation of the diarrhoea given by Richbieth and others is correct—that it is the result of the toxæmia produced by the blood infection, as in the case of other septicæmic conditions.

Cases of pneumococcal peritonitis may be divided into three groups:

1. Those in which the peritonitis develops concurrently or simultaneously with pneumonia or pleurisy. This is the most common form. The peritonitis runs an acute course, and in fatal cases terminates about the sixth or seventh day. If the duration be prolonged, the outlook is more hopeful, as the severity of the symptoms may abate at the eighth to tenth day, and the disease become chronic and localized.
2. Cases of very acute type in which no other pneumococcal lesion or source of infection is discoverable post mortem. The duration in this group is often very short—about two to three days. Diarrhoea is frequently present. This form is less common than the preceding.
3. Cases running a subacute or more chronic course. In these there is usually some source of infection present not of an acute type; for instance, an incompletely healed empyema with a sinus, or otitis media, or the patient has had a recent attack

of pneumonia. In such cases the infection of the peritoneum is much less virulent than in Groups 1 and 2.

In the majority of cases the symptomatology is that of an acute peritonitis. Herpes not infrequently appears on the lips. In infants and in the most acute forms the abdominal symptoms may be comparatively slight, and may escape observation. A high leucocytosis rapidly develops, though in the fulminating cases and in very young or weakly children this may not take place; in such cases there is a failure of reaction to the infection, and this should be regarded as a most unfavourable indication. In Rischbeth's series of fifty-four cases, forty-eight were fatal—a mortality of 88.8 per cent.

The most favourable cases are those in which at the end of seven or eight days there is a subsidence in the acute symptoms, together with a fall of temperature. The signs of general peritonitis lessen, and the fluid begins to become localized. The pus tends to collect in the lower part of the abdominal cavity, in the iliac fossae, the umbilical region, or the pelvis, and spontaneous discharge may take place at the navel, or into the bowel or vagina. Cases rarely recover without the formation of such residual abscesses.

The peritoneum after pneumococcal infection shows the usual appearance of an acute peritonitis. The character of the exudation presents certain special features. In a very early case there is usually a moderate quantity of thin, rather turbid fluid. In later cases the exudation is thicker, and obviously purulent, and contains greenish-yellow flakes of lymph. The fluid readily forms soft, jelly-like clots. In the more chronic forms, or when the exudation has become localized, it consists of greenish-yellow pus, usually odourless; but if it has been of long duration, may have become offensive, owing to mixed infection with *Bacillus coli*. Extensive fibrous adhesions, either general or local, may be left as the result of an attack of pneumococcal peritonitis. Other lesions are often present, such as meningitis, pericarditis, arthritis.

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Gonococcal Peritonitis.—This form of peritonitis is distinctly rare. It is due to a vaginal infection, and when we consider the frequency with which gonococcal vulvo-vaginitis occurs, it is somewhat surprising that it is not more common. In children the condition is confined to the female sex. The onset is very abrupt or explosive, and attended with great prostration and pain, and the signs of acute peritonitis are early apparent. The possibility of a gonococcal infection may be unthought of unless the existence of a vulvo-vaginal discharge has been discovered and cultivations made therefrom, or until the presence of gonococci in the pus obtained from the peritoneal cavity by laparotomy has led to an examination of the genitalia. There seems every reason to believe that a gonococcal vulvo-vaginitis may infect the peritoneal cavity by an ascending infection of the Fallopian tubes. In the very rare cases of gonococcal peritonitis in the male, the infection travels up the spermatic cord. In children this form of peritonitis is attended by a higher mortality than in adults, probably because the infection of the peritoneum assumes a diffuse character, instead of tending to be localized in the lower part of the abdominal cavity. Comby gives a mortality of 20 per cent. in children. There

seems little doubt that, although a very serious affection, gonococcal peritonitis is one of the least dangerous forms of acute peritonitis in childhood. In fatal cases death is probably due to septicæmia. In favourable cases the abdominal pain and tenderness lessen towards the end of a week, and all the signs and symptoms may disappear in ten to fourteen days from the onset. It appears doubtful in cases of recovery in children, what effect the disease may ultimately produce as regards lesions of the Fallopian tubes and sterility.

TREATMENT OF ACUTE PERITONITIS.—(1) Medical treatment; (2) surgical treatment.

I. MEDICAL TREATMENT.—The child is placed in the most comfortable position, the head well supported, and the abdomen protected from pressure by the bed-clothes. All food, liquid and solid, should be withheld. Ice may be given in suck. Fluid should be supplied as saline infusions under the skin at regular intervals; rectal saline injections may cause pain, but, if well tolerated, should be given. Purgatives should not be administered, as they cause increased peristalsis and abdominal pain. The bowel should be washed out with a simple enema. For the relief of abdominal pain, turpentine stripes or ice-bags should be applied, care being taken to avoid pressure.

Opium and morphia are contra-indicated for two reasons: first, because the clinical features of the disease may be masked and the diagnosis obscured by the speedy relief from pain resulting; and, secondly, because, according to Badger and Sargent, these drugs not only increase the paralysis of the gut, but also, there is reason to believe, they may inhibit phagocytosis. If the pain is very distressing, codeine in $\frac{1}{4}$ -grain doses every four hours for a child of four years may be given, as suggested by Eustace Smith.

Vomiting is often a troublesome symptom; washing out the stomach may relieve it.

Collapse may be present at the onset of the case, and should be treated with hot applications to the limbs and saline infusions, rectal saline injections, and hypodermic injections of strychnine, camphorated oil, or adrenalin chloride.

2. SURGICAL TREATMENT.—In the majority of cases of acute peritonitis, although the recognition of its existence may present so difficulty, the diagnosis of the cause is often difficult in the extreme. A large proportion of the cases are due to appendicitis, and if there be any doubt as to the nature of the infection in a given case, laparotomy should be performed without delay, as by this means the local lesion may be dealt with and the fluid evacuated. There is some difference of opinion as to whether immediate operation should be performed in all cases of pneumococcal and gonococcal infection. In the pneumococcal form the infection is diffuse, the result of a septicæmia, and in children the mortality is very high, whether laparotomy is performed early or is delayed. Little is gained by waiting in the hope that the peritoneal infection may become localized, even when the pneumococcal nature of the case is recognized. The abdomen should be opened and thoroughly drained. In pneumococcal cases at a later stage, when localized collections of pus have formed, these should be opened and drained. The gonococcal cases should be also treated by laparotomy.

The use of an autogenous vaccine is indicated in pneumococcal cases with chronic abscesses, and in the acute stage it should be given, if time permit of its preparation. Anti-pneumococcal serum has proved useful in the writer's experience.

In the other forms of acute peritonitis—i.e., the streptococcal, staphylococcal

and *Bacillus coli* infections—little benefit is derived from the use of either vaccines or sera; the disease runs too short and acute a course in children for an effort to be produced.

If possible, laparotomy should not be performed during the initial shock of the infection; it should be delayed till collapse has passed off and reaction set in.

II. Chronic Forms of Peritonitis.

In children chronic peritonitis occurs in two forms—localized and diffuse.

1. **Localized Form.**—This is usually the result of an antecedent acute peritoneal inflammation, either generalized or localized, such as pneumococcal peritonitis, which has finally quieted down and become limited to one part of the peritoneal cavity; or an attack of appendicitis may lead to chronic peritonitis in the right iliac fossa. In such cases adhesions are formed which may or may not give rise to symptoms. Pain due to dragging on the adhesions may be present, or signs of intestinal obstruction may be caused by kinking of the gut or constriction by a band.

2. **Diffuse Form.**—The most common cause of diffuse chronic peritonitis is tuberculous peritonitis, which will be considered later, though it may occasionally follow some of the varieties of acute peritonitis already described. In such cases the effect produced is a more or less general matting of the intestines by organized adhesions. There are, however, two forms of diffuse chronic peritonitis which require special attention. The first is that sometimes described as "syphilitic" peritonitis. This variety is met with in the foetus, and in young infants, the subjects of congenital syphilis. It may be associated with ascites, and the term "fatal ascites" is given to this condition when it is present in the foetus or newly-born infant. The inflammatory process is generally unattended with pain or acute symptoms, and runs a chronic course, usually terminating fatally. The peritonitis is not always attended by effusion, but is often dry. It seems probable that the peritoneal inflammation is set up by some infection of comparatively feeble virulence, probably streptococcal, to which the syphilitic infant offers but slight resistance, owing to its debilitated condition. The condition is a rare one in this country.

The second form of chronic diffuse peritonitis is sometimes described as "chronic adhesive peritonitis," and it forms a part of the condition known as "polyserositis," or "polyserositis," as described by F. Taylor, Pick, Contato, and others. In this the peritoneal inflammation is associated with chronic fibrotic or adhesive changes in the mediastinum, pericardium, and pleura. Adhesions are most numerous in the upper part of the peritoneal cavity, and may be limited to the under-surface of the diaphragm and the hepatic and splenic regions, where the inflammatory process leads to a thickening of the capsules of the liver and spleen. The liver is usually enlarged, and ascites commonly present.

The nature of the inflammatory process in polyserositis is a question of considerable interest. There is much to support the view that the changes in the thorax and peritoneum are the result of a chronic tuberculous infection. The peritonitis is probably secondary to the mediastino-pericardial affection by extension through the diaphragm (*cf.* Chapter VIII., p. 423).

III. Tuberculous Peritonitis.

Tuberculous peritonitis is the commonest disease of the peritoneum met with in children. Although in a few rare instances it may seem to occur as a primary affection, in the vast majority of cases it is a secondary condition, the result of infection which has reached the peritoneum from some near or remote tuberculous focus. In the clinical picture presented by the common varieties of abdominal tuberculosis in children, the peritoneal signs are usually predominant, and there is some risk that the underlying factors may receive insufficient attention.

Formerly the term *tuber mesenterica* was used to denote extensive tuberculous affection of the mesenteric glands, and later was loosely applied to cases of tuberculous peritonitis with palpable glands, so that it became a matter of some uncertainty as to the precise meaning to be attached to the term. To avoid confusion, it would seem advisable to discontinue the use of the name *tuber mesenterica*.

In children tuberculous infection of the mesenteric glands in some degree is exceedingly common, and is often present without definite signs and without obvious infection of the peritoneum. It is remarkable with what frequency tuberculosis of these glands is met with in laparotomies performed in young children in acute conditions, such as intestinal obstruction, in whom the existence of tuberculous disease has been quite unsuspected.

Ætiology.—*Sex.*—Male and female children are liable to tuberculous peritonitis in almost equal proportions. Thus, in 163 consecutive cases under fourteen admitted to the East London Hospital for Children there were 82 males and 81 females. Goodhart and Still in 100 cases found 48 males and 52 females; Billiet and Barthex, 53 boys and 33 girls.

The sex liability is a matter of some importance, as the Fallopian tubes are a possible path of infection in females, and if infection reach the peritoneum by this route, we should expect to find a greater frequency in girls than in boys. Female children, however, are not more, but rather less, liable to the disease than are boys, and this must be taken as strong evidence against any infection occurring in connection with the female genital organs. On the other hand, adult females are more liable to tuberculous peritonitis than adult males, chiefly on account of the frequent occurrence of an ascending infection from tuberculous Fallopian tubes.

Age.—The disease is commonest in the second, third, and fourth years. In the series of 163 cases in children under fourteen, 48 occurred in these years. The following figures show the distribution of the cases according to age:

Age	Under 1 Year	1-2	2-3	3-4	4-5	5-6	6-7	7-8	8-9	10-11	12-13	14-15	16-17	18-19
Number of Cases	5	29	25	23	16	10	8	13	7	5	14	5	4	2

Total 161 (2 ages unknown) = 163.

The disease is comparatively rare in children over eleven years of age.

Influence of Specific Infections.—The specific fevers, especially measles, undoubtedly predispose to tuberculous peritonitis, as they may to other tuberculous infections. They act by lowering the general resistance to microbial invasion.

It seems reasonable to suppose that the existing powers of the abdominal lymphatic structures may be affected by many acute diseases, such as broncho-

pneumonia, as it is common in such conditions to find post mortem in young children swelling and injection of Peyer's patches and of the mesenteric glands.

Unhealthy States of the Alimentary Tract.—The alimentary tract of children is peculiarly exposed to tuberculous infection, as will be seen later in the consideration of the paths of peritoneal infection. Unhealthy conditions of the intestinal tract, such as chronic catarrh or enteritis, undoubtedly are factors in the production of infection of the mesenteric glands and of the peritoneum, probably by lowering the resistance normally offered to infection by the lymphatic tissues. In this respect the alimentary canal may be compared to the respiratory tract, in which chronic catarrhal conditions undoubtedly give rise to an increased liability to tuberculous infections of the lungs and bronchial glands.

Trauma.—A history of a blow on the abdomen or of a severe fall is not infrequently met with, and occasionally may have a direct relation to the onset of the disease.

Symptomatology.—Tuberculous disease of the peritoneum usually runs a slow insidious course, but cases are met with in which the disease takes an acute form.

The clinical types of the disease will be considered in two divisions: *I. Acute; II. Chronic.*

I. Acute Form.—These are divided into—

1. Diffuse or military type.
2. Localized types.

1. Diffuse or Military Type.—This is an acute infection of the peritoneum, most commonly met with as part of a general infection in cases of acute military tuberculosis, in which the abdominal symptoms are the predominant features of the case. Cases of general tuberculosis in which scattered tubercles are found in the peritoneum at the autopsy, though there were no abdominal signs or symptoms during life, are of frequent occurrence, but do not now come under consideration. It is rare to meet with cases of acute tuberculous peritonitis apart from a generalized tuberculosis, but they may occur in two conditions: first, those in which infection is set up by the more or less sudden discharge of tuberculous material into the peritoneal cavity, such as might be caused by the breaking down or rupture of a caseous mesenteric gland; and, secondly, the very rare cases of acute tuberculous peritonitis in which no other tuberculous lesions are discoverable post mortem, and which are described as primary infections of the peritoneum.

The clinical manifestations of the acute form are pyrexia, abdominal pain and tenderness, sometimes preceded by a brief period of general malaise. The face is flushed, the tongue dry and furred. The abdomen becomes distended and tender. Fluid is usually present, sometimes in considerable amount, so that a thrill may readily be obtained, or in less quantity it may cause some dulness in the flanks. Intestinal distension may be so great as to mask the presence of fluid. Enlargement of the spleen may be made out if the abdomen is not too distended to permit of examination. Diarrhoea is not infrequently present. Pyrexia usually persists throughout the course of the disease. The child sinks into a torpid, stuporous state, and death takes place usually in three to five weeks, either from exhaustion or involvement of the meninges, though in some cases the acute symptoms may gradually subside, and the disease pass into one or other of the chronic forms to be described later.

It will be seen from this description how closely a case of acute tuberculous peritonitis of the diffuse form may resemble one of typhoid fever.

2. Acute Localized Form.—In this form the appendix and ileo-caecal group of mesenteric glands form the routes by which infection reaches the peritoneum. Such cases are comparatively rare, but deserve recognition, as they may closely simulate attacks of appendicitis. Brunner, Eisendrath, and others, have recorded cases of this kind, but comparatively little attention has been paid to this clinical type in this country. Tuberculosis of the Fallopian tubes may also give rise to an acute localized form, but this source of infection is of far rarer occurrence in children than in adults.

The symptomatology of this "appendicular" or "ileo-caecal" variety is as follows: There is a sudden onset with severe abdominal pain, and sometimes vomiting, accompanied by pyrexia. Resistance is felt in the right iliac fossa, or a definite mass may be made out in this situation, and the diagnosis of appendicitis is usually arrived at. Laparotomy reveals a mass of acutely inflamed mesenteric glands, which generally contain recent caseous foci. The appendix is swollen and injected. It may contain tuberculous ulcers, or tubercles may be present in the sub-mucosa, but in some cases no definite tuberculous lesions are found in the organ. These seem every reason to believe that tubercle bacilli may pass through the wall of the appendix without producing demonstrable lesions therein.

In some cases the caecum itself is infiltrated with tubercles to such an extent as to form a palpable mass in the right iliac fossa.

This "appendicular" or "ileo-caecal" form of acute tuberculous peritonitis, if left untreated, may remain localized, or may set up a generalized acute or chronic peritonitis.

II. Chronic Form.—In the great majority of cases tuberculous infection of the peritoneum runs a slow chronic course.

The symptomatology of all forms of chronic tuberculous presents certain features in common; thus, the onset is slow and insidious as a general rule, though cases are met with which, from the sudden onset of their earlier symptoms, may resemble the acute varieties of the disease. The early symptoms of the chronic form are not infrequently resemble those of chronic gastro-intestinal disorders—general malaise, anorexia, with progressive wasting and increasing pallor. On the other hand, it is not unusual to meet with cases in which the state of general nutrition is surprisingly well maintained, although the peritoneal affection is definitely established. Abdominal pain, usually vague and slight, but sometimes severe, is frequent, and is increased by movements, especially those of walking, running, and jumping. Eustace Smith has pointed out that in the early stages of the disease the abdominal pain caused by walking downstairs may be an early and significant symptom. Pyrexia is usually present, it is generally irregular in type and variable in degree. In cases in which intestinal ulceration has occurred, diarrhoea may be a prominent symptom, though more commonly in the early stages of the chronic form, if the action of the bowels is irregular, the looseness is due to a catarrhal state of the intestine. Constipation, on the other hand, is not infrequently met with.

A sign common to most cases of chronic tuberculous peritonitis is progressive enlargement of the abdomen.

It is not uncommon to find associated with the chronic forms of tuberculous peritonitis a dry pleurisy, limited to the bases of one or both lungs. This pleurisy must be regarded as secondary to the peritoneal infection, the tubercle bacilli reaching the pleura by the lymphatics of the diaphragm. It is usually unattended by pain, and may subside simultaneously with the peritoneal affection.

Clinical Varieties.—It is found convenient to divide the clinical varieties of chronic tuberculous peritonitis into three chief types, each presenting certain special features. These are—

1. The ascitic form.
2. Adhesive, fibrinous, or plastic form.
3. Caseous (or ulcerous) form.

It must be borne in mind that intermediate and mixed types are often met with, and also that a case may begin as one variety and gradually take on the characteristics of another; for example, an ascitic case may develop into one of either of the other forms.

1. Ascitic Form.—In many cases of this type the first thing noticed is enlargement of the abdomen, and this may develop quite suddenly, so that in the course of a few days the abdomen may have become considerably distended. Slight indisposition, as shown by fretfulness and loss of appetite, may be the only symptoms preceding or accompanying the onset. Less frequently the onset of the abdominal enlargement is more acute and attended with fever. Pain is not usually a prominent symptom.

Though slight wasting may have occurred in an early stage, the state of general nutrition is often remarkably good, and there may be nothing in the appearance of the child to suggest the presence of so serious a disease. The abdomen presents a characteristic appearance; when the child is upright, there is a marked protrusion; and when recumbent, the belly has a globular form. The navel is flattened, or, when the distension is great, is everted or pointing. As the distension increases, the skin covering the belly becomes smooth and shiny, and the superficial veins may be seen unusually distinctly. The diaphragm is pushed upwards, causing an upward displacement of the hepatic and of the cardiac dulnesses. On palpation of the abdomen, a fluid thrill can be usually elicited, but this may be absent when the intestines are much distended with gas. When present in smaller amount, the fluid will give rise to shifting dulness in the flanks. Sometimes the presence of free fluid in the peritoneal cavity can be detected by placing the child in the genu-pectoral position, when a distinct fluid thrill may be obtained by palpating in the umbilical region, now the most dependent part.

It is important to bear in mind the fact that the fluid contents of distended intestines may produce a thrill, sometimes mistaken for that caused by ascitic fluid.

It may be possible, when there is no great amount of free fluid, to make out by deep palpation the presence of enlarged mesenteric glands, or the thickened, coiled-up omentum may be felt as a ridge or band passing transversely across the upper part of the abdomen.

The fluid is usually straw-coloured; occasionally, in cases with an acute onset, it may contain blood, though in the writer's experience a hæmorrhagic effusion is less commonly met with in children than in adults. The fluid has usually a specific gravity of about 1025, and contains much albumen and many cells, among which lymphocytes largely predominate.

The ascitic type is rightly regarded as the most favourable form of tuberculous peritonitis. The fluid may gradually become reabsorbed, and the child may make a good recovery. In such cases the peritoneum, after a lapse of time, may show no trace of the former affection, or localized or general adhesions may result (plastic or fibrinous type). The less favourable cases may gradually develop into

the caseous or ulcerous variety, to be described later, while others may develop further tuberculous lesions, or terminate in an acute generalized tuberculosis.

2. Adhesive or Fibrinous Type.—Cases of the adhesive type may begin with ascites, and possibly in their earlier stages the ascitic form already described, as the condition may gradually develop without the occurrence of ascites.

The onset of this form of tuberculous peritonitis does not differ to any great extent from that of the ascitic variety. Advice is generally sought because the child is ailing, or on account of abdominal pain or enlargement. In early cases there is nothing characteristic beyond the abdominal conditions. The child has a "pot-bellied" appearance, though the prominence of the abdomen is usually not so marked as in the other forms, and is chiefly due to intestinal distension. On palpation, the abdomen has a curious "doughy" feel, as if filled with some soft substance, such as porridge. There is usually little or no tenderness, though at times the child may complain of some abdominal pain.

In a considerable proportion of cases an oriental band or thickening can be felt, or enlarged glands may be made out by deep palpation.

In some cases of this adhesive variety, especially in those of long standing, the abdomen is not prominent, but, on the contrary, may be retracted. This is due to a shortening and thickening of the mesentery, met with especially in this type, which causes a general retraction of the bowel towards the spine, so that the intestines may feel unduly fixed.

Recovery may occur, though it is impossible to give accurate figures as to the chances of life. The course is prolonged, and, should recovery take place, extensive adhesions are produced, sometimes to such an extent as to cause complete obliteration of the peritoneal cavity. These adhesions may be a source of further complications by causing obstruction or kinking of the bowel. On the other hand, very extensive adhesion of the peritoneal surface may exist for years without giving rise to any untoward events.

As in the preceding variety, the fibrinous or adhesive form may be associated with a terminal generalized tuberculosis, or a fatal result may be brought about by the development of pulmonary lesions.

3. Caseous (or Ulcerous) Form.—This is the most severe type of tuberculous peritonitis. In its early stages there may be little to distinguish it from the other varieties, as it has the same gradual and insidious onset. Not a few cases begin as the ascitic form.

Abdominal pain is generally present, and sometimes is very severe and paroxysmal in character. It is aggravated by movement; even the act of turning in bed or the pressure of the bedclothes may cause discomfort.

Some degree of pyrexia is usually present, and as the disease progresses the temperature often assumes the hectic type, owing to the occurrence of a mixed infection.

Diarrhoea is a frequent symptom, and in the majority of cases of this type is associated with ulceration of the bowel. Its severity is variable. The stools are often loose, offensive, and contain undigested food and occasionally blood. In the more severe cases diarrhoea may be the cause of death, from the resulting exhaustion. On the other hand, constipation may be a troublesome symptom, and is sometimes due to obstruction of the bowel by adhesions or kinking. Extensive ulceration of the gut may be present, with little or no diarrhoea.

The appearance of the child is very characteristic. The face has a listless apathetic expression; the features are often less wasted than the body. In the

later stages an "abdominal facies" develops, due to the presence of folds or creases at the angle of the mouth.

The skin is harsh and dry, with loss of elasticity, and has a dirty white colour, as if unwashed, or may show a diffuse brownish pigmentation, most marked in the flexures of the joints.

The abdomen is prominent and globular, and looks disproportionately large compared with the wasted thorax and limbs. The increased size is chiefly due to distension of the intestines rather than to the presence of ascites, though free fluid can be often detected in the flanks.

The appearance of the navel is often significant and of some importance in the diagnosis. It may be only flattened or unduly shallow, but it is often everted, so that it appears to protrude or project outwards, and frequently shows signs of local inflammation. The navel commonly feels more fixed than normal when moved from side to side.

The skin covering the abdomen sometimes has a peculiarly shiny appearance, due to the distension. On light palpation of the surface, little nodules, like small shot, may be felt in the subcutaneous tissue; these little masses of subcutaneous fibrous tissue should not be mistaken for tubercles.

Coils of distended intestine may be made out exhibiting visible peristalsis.

Palpation reveals the presence of masses of varying size and consistency, formed by enlarged glands, omentum, or thickened adherent coils of intestine and mesentery. Localized collections of fluid can sometimes be detected; these are often purulent, owing to a mixed infection by pyogenic organisms giving rise to a localized abscess. The skin over this region becomes red and tender, and the abscess may point and discharge its contents, consisting of pus often mixed with fecal matter, and a fecal fistula may result. The commonest situation for this to occur is at the navel.

Death is due either to extension of the disease to other parts—i.e., acute miliary tuberculosis or pulmonary tuberculosis—or to gradual exhaustion brought about by diarrhea, abscess formation, fecal fistula, lardaceous disease, pyrexia, or intestinal obstruction.

PATHOLOGY.—It is important to consider first the possible channels by which the peritoneum may become infected by the tubercle bacillus.

The Blood.—The infection may be conveyed to the peritoneum by the blood-supply. This occurs frequently in cases of acute miliary tuberculosis, in which the peritoneum, in common with other organs or structures, is found to be studded with tubercles, although little or no evidence of peritonitis has existed during life. Such cases are regarded as secondary, and the primary focus from which dissemination has arisen is usually found without difficulty. A few cases of acute tuberculosis of the peritoneum have been described in which no other tuberculous lesion has been found in the body, and such cases must be regarded as due to a primary blood infection, though it is possible that the original focus may have been so insignificant as to have escaped observation.

The Intestine.—This is by far the commonest channel of infection in children. The bacilli may be acquired by means of infected food, especially milk and butter, or by sucking dirty toys and hands. In rather more than half the cases (53 per cent.) investigated by the Royal Commission on Tuberculosis, the tubercle bacillus present was of the bovine variety. Children, owing to their small stature, crawling habits, and usually open mouths, are specially liable to inhale and swallow a large amount of infected material, which ultimately reaches the intestine. Here

localized lesions may result in the form of ulcers, or, as has been experimentally proved, the bacilli may pass through the wall of the bowel without producing demonstrable changes therein. The peritoneum becomes secondarily infected, either directly from the intestine or from the mesenteric glands.

Lymphatic.—Tubercle bacilli most commonly reach the peritoneum from the intestine by the lymphatics. The lymphatics in the vicinity of a tuberculous ulcer are often recognizable as white, varicose, distended channels, studded with tubercles on the serous surface of the gut. The infected mesenteric glands in an early stage are swollen and inflamed, and may show small caseous foci. Later, caseation is more extensive, and the gland may ultimately completely soften and break down.

There is reason to believe that the resistance offered to the passage of tubercle bacilli by the intestinal wall and lymphatic glands may be lowered by unhealthy conditions, such as chronic catarrh of the bowel, or by acute febrile conditions such as measles, as is the case in the lungs and bronchial glands.

The infection may reach the peritoneum by other lymphatic channels—by the diaphragmatic lymphatics, for instance; but usually in such cases the peritoneal condition is local and latent. It is not very uncommon to meet with other evidence of local tolerance of the peritoneum to tuberculous infection; thus, in operations for hernia tubercles are occasionally found in the peritoneal lining of the sac, without giving rise to any symptom or sign of their presence.

Extension from some local focus, such as the Fallopian tubes, tuberculous kidney, etc. In children the peritoneum rarely becomes infected in this way. The part played by the Fallopian tubes has been already discussed, and need not be further dealt with.

MORBID ANATOMY.—Having considered the symptomatology of chronic tuberculous peritonitis under the three clinical forms—i.e., the ascitic, the adhesive, and the caseous (or ulcerous)—it is convenient to adopt the same division in considering the morbid anatomy of the disease.

Ascitic Form.—The peritoneum is covered with discrete grey or yellow tubercles. The omentum is usually folded or rolled up, and is also studded with tubercles.

The fluid is straw-coloured; sometimes it is brown, owing to the presence of altered blood-pigment. It has a specific gravity of 1020 to 1025, and is alkaline. The cell elements are increased in number, and show a marked predominance of lymphocytes. It is not easy to demonstrate the presence of tubercle bacilli in the fluid except by inoculation into guinea-pigs, as the bacilli are present in scanty numbers.

In early or more acute cases the natural, smooth, shiny appearance of the peritoneum is lost, and the surface becomes dull, lustreless, slightly roughened, with some capillary injection.

A brief mention must be made of the form of tuberculous infection of the peritoneum in acute miliary tuberculosis, with which as a rule no abdominal symptoms are associated. In this the peritoneum is studded with minute greyish, translucent tubercles. Generally no naked-eye manifestations of peritonitis are visible, though there may be slight ascites. The appearances closely resemble those exhibited in the pleura.

Adhesive, Fibrous or Plastic Form.—In this variety the peritoneal cavity is sometimes entirely obliterated. On opening the abdomen in the usual way, the

coils of intestine are found to be firmly adherent to each other and to the parietes without any obvious signs of tubercles or caseous material. The mesentery is thickened and shortened, so that the intestines are drawn towards the spinal column. In other cases the adhesions are more limited in their distribution, and cords or bands of varying size and thickness are present.

In the majority of such cases evidence of the tuberculous nature of the case can usually be found in the mesenteric glands, which exhibit caseous or calcareous foci.

Caseous (or Ulcerous) Form.—In the adhesive type there is a tendency to the formation of fibrous tissue, whereas the leading characteristic of that now under discussion is the production of masses of caseous material.

The peritoneum is studded with yellow caseous tubercles, and sometimes exhibits patches of dark grey pigmentation. The omentum is thickened and rolled up, and is full of similar tubercles. The coils of intestine are glued together by masses of yellow, cheesy-looking material. With care the coils can be separated, except where dense fibrous adhesion has taken place. The intestinal wall is thin and friable, and may have given way in one or more places, with formation of fecal fistule. Ascitic fluid of the same characters as in the acute form is often present in inconsiderable amount, either free or shut in by adhesions. Localized collections of pus are frequently found due to a mixed infection with pyogenic organisms.

Extensive ulceration of the bowel is characteristic of this form. This may lead to perforation into the peritoneal cavity, causing an acute peritonitis, or more usually a localized abscess with partly fecal contents; or adjacent coils of the gut may communicate by a fistulous passage, or the gut adhering to the parietes may perforate externally.

The mesenteric glands are generally enlarged and show advanced caseation.

In cases of long duration with prolonged suppuration, lardaceous degeneration of the liver, spleen, and intestines, is not uncommonly present. The Fallopian tubes are occasionally found filled with purulent caseous material, the result of a descending infection, and in a small proportion of such cases the uterine cavity may contain similar material. Tuberculous masses are sometimes found in the liver and spleen. In the liver these are situated in the portal spaces, and may form bile-stained cysts, occasionally of considerable size (vide p. 224).

Diagnosis.—The acute forms may resemble the other acute infections of the peritoneum, and there may be considerable difficulty in recognizing the tuberculous nature of the case in the absence of other tuberculous lesions. Examination of the blood may give considerable assistance, since in tuberculous peritonitis a low leucocyte count or a moderate leucocytosis, in young children, differentiates it from the pyogenic infections, such as pneumococcal, streptococcal, and other forms, in which a high leucocytosis is the rule; though if a mixed infection with pyogenic organisms has taken place a definite leucocytosis is generally present. Reference has been made to cases in which the pyrexia, distended abdomen, and palpable spleen, and the low leucocyte count, may suggest typhoid fever; in these the Widal agglutination test should be applied. It would seem almost impossible to distinguish without operation the appendicular or ileo-caecal form from an ordinary acute appendicitis, as the symptoms and signs are practically identical.

The diagnosis of the chronic forms of tuberculous peritonitis in typical cases is not difficult; the gradual onset, with abdominal distension and the characteristic doughy feel, the everted navel, palpable mesenteric glands, and thickened

emaciation, with or without the presence of ascites, make a very definite picture. In early stages, however, the diagnosis may be difficult. Careful examination should be made to ascertain the presence of enlarged glands. These are usually found to the left of the vertebral column, near the navel and in the right iliac fossa. Rectal examination sometimes reveals their presence when abdominal palpation has proved negative. Fecal masses may be mistaken for enlarged glands. Some forms of chronic enteritis simulate tuberculous peritonitis, as they are associated with general failure of nutrition, abdominal distension, and loose offensive stools. Congenital or idiopathic dilatation of the colon (Hirschsprung's disease) gives rise to abdominal distension and a feeling of fulness of the abdomen, such as is found in tuberculous peritonitis. It is not difficult to prove the existence of dilatation of the bowel by repeated enemata, which may bring away large collections of feces, or by examination by X-rays after the administration of bismuth emulsion by the mouth or rectum. Lymphadenomas, when the glands affected are confined to the abdomen, may closely resemble tuberculous peritonitis. Considerable enlargement of the spleen is in favour of the case being one of lymphadenoma. Localized abscess formation in pneumococcal peritonitis may occasion difficulty as regards the differential diagnosis, especially if seen at a late stage. Ascites in polyarthrosesitis (polyserositis), in which mediastinitis, pericarditis, pleurisy, and peritonitis are associated as the result of a chronic inflammation, is distinguished by the presence of the thoracic lesions. Cirrhosis of the liver, more particularly the syphilitic form, when it gives rise to ascites, may be mistaken for tuberculous peritonitis. The family history, the presence of other specific lesions, and a positive Wassermann reaction, should render the diagnosis clear.

Certain rare forms of peritoneal growths, especially myxosarcoma, have been diagnosed as tuberculous peritonitis. Lympho-sarcoma, lymphadenoma, sarcoma and mesenteric cysts, may also simulate tuberculous peritonitis.

The value of tuberculin tests in the diagnosis must be briefly discussed. V. Pirquet's cutaneous method is of some value, if a negative result is obtained in a mild, doubtful case, as evidence against the existence of a tuberculous lesion. It should be borne in mind that this reaction is often negative in very severe and advanced cases of abdominal tuberculosis. The subcutaneous or hypodermic method is of greater value if a negative result is obtained, and is more reliable than v. Pirquet's test; but it is superfluous in advanced cases, because in such the diagnosis should present little difficulty, and the presence of irregular pyrexia may obscure any reaction which takes place. A positive reaction produced by either method is only of value in so far that it is an indication of some tuberculous focus, and does not necessarily indicate that the disease in question is of a tuberculous nature.

Prognosis.—The question of prognosis in tuberculous peritonitis naturally is divided into a consideration, first, of the immediate outlook as regards recovery from the local condition, and, secondly, of the ultimate prospects of the case.

As regards the *first* point, the prognosis during the active course of the disease depends on—(a) the type of the peritoneal affection: the ascitic form is undoubtedly the most favourable, and the caseous the most serious type; (b) the age of the patient: the younger the child, the more unfavourable is the outlook. Still, in a series of 100 cases with autopsy, found that 53 were between one and four years. In the writer's series of 163 cases, with 49 deaths, 35 of the fatal cases occurred in children under four. Again, certain symptoms affect the prognosis adversely: progressive wasting in spite of treatment; the presence of pulmonary lesions; the

occurrence of suppuration, early indications of which may be afforded by hectic fever or inflammation of the navel; obstinate diarrhoea, as affording evidence of intestinal ulceration. Cases with local abscesses almost invariably run a fatal course. Pleurisy need not be regarded as an unfavourable association.

Secondly, what are the ultimate prospects of a case that has survived the perils of the peritonitis? A certain proportion undoubtedly recover, but it is most important to bear in mind that in the great majority of cases the peritoneal infection is only a secondary condition, and though, given suitable conditions, this may entirely clear up, the mesenteric and bronchial glands remain for a much longer period potential sources of general infection. Thus, a considerable proportion of cases of apparent recovery from tuberculous peritonitis terminate after a lapse, it may be, of some years, from acute tuberculosis, meningitis, or from deposits in the lungs or elsewhere. It is difficult to form an accurate estimate of the proportion of cases making complete recovery. Beaton made an inquiry into the after-history of a series of cases treated in hospital, which, though it dealt with a comparatively small number, and those of serious type, shows clearly the heavy mortality ultimately attending the disease.

TREATMENT.—Tuberculous peritonitis, although a very serious affection, is not to be regarded by any means as a hopeless disease of children. Much can be effected by treatment, and it is remarkable how some of the most severe cases will respond to the measures adopted. Even in cases regarded as hopeless, great alleviation of suffering and some prolongation of life, and occasionally unlooked-for recovery, may be brought about by the adoption of appropriate measures.

The subject will be considered as follows:

1. General hygienic and dietetic treatment.
2. Medicinal treatment.
3. Surgical treatment.

1. General Hygienic and Dietetic Treatment.—In all cases the child must be kept in bed until all signs of active disease have subsided. A broad flannel binder should be applied to the abdomen. Care must be taken to keep the feet warm, and for this sleeping socks are usually necessary.

The room must be large and light, with a sunny aspect, but whenever possible open-air treatment should be carried out. The results obtained by open-air treatment in tuberculous peritonitis are striking, even when carried out in crowded, smoky districts such as in the poorer neighbourhoods of London. In the open-air shelters at the East London Hospital for Children, Shadwell, the improvement is often surprising. The most delicate children support the treatment well when protected by suitable clothing, and thoroughly like and appreciate it. The appetite improves, and the irregular pyrexia so frequently present lessens, and often entirely disappears. If open-air treatment cannot be carried out either on a balcony or in a shelter, the bed should be placed near a window which is kept open night and day. Cases that can be sent away do better at the seaside than elsewhere. The climate of the South Coast resorts, and those on the Kentish coast, such as Broadstairs, Margate, etc., is suitable for most cases all the year round.

Diet.—This will largely depend on the nature of the case. The main object is to improve the state of general nutrition, but great care has to be exercised, as in many cases some degree of disorder of the alimentary system is present, and the diet will largely depend on the character of the stools. In cases with diarrhoea or

loose, offensive stools, the diet should consist largely of milk. Carbohydrates, such as bread, potatoes, rice, sago, tapioca, sugar, etc., should be withheld, or given in very small quantity. Lightly boiled or poached eggs or beaten-up egg and milk are well borne.

When the intestinal functions are more normal, the diet may be increased and pounded fish or meat added. Fat should be given either as butter, cream, or as cod-liver-oil. In many cases the starchy foods are indifferently tolerated, and give rise to flatulence and loose, offensive stools.

In some cases milk is poorly digested, or may even cause diarrhoea. The effect of pancreatization should be tried; or whey and cream, veal or chicken broth, to which plasma has been added, may be substituted for a time.

2. Medicinal Treatment.—Some preparation of iron is indicated, and the form in which it is given depends on the state of the alimentary canal. When it can be tolerated without causing diarrhoea, the iodide of iron seems to produce the best results, as it does in other forms of chronic tuberculous disease. In more delicate cases the ammonio-citrate or tartrate of iron should be substituted.

Cod-liver-oil should not be given when the stools are loose and unhealthy; when it is indicated, $\frac{1}{2}$ to 1 drachm doses are quite sufficient. The dosage of oil is often excessive in these cases, and gives rise to disturbance of the bowels. Children tolerate it best in the form of an emulsion.

Diarrhoea should be treated by a suitable modification of the diet. The bowels should be emptied by a dose of castor-oil or a rhubarb and soda powder. Astringents, such as bismuth, aromatic chalk and opium powder, paly, kino co., are required in the more severe forms. Intestinal antiseptics, such as salol, naphthalin tetra-chloride, or β -naphthol, should be given when the stools are offensive, loose, and frothy, in spite of careful regulation of the diet.

Cresote in $\frac{1}{2}$ to 1 minim doses is very useful. It can be added to the castor-oil emulsion. For older children it is best given in capsules. Guaiacol and thiozol given by the mouth have not proved so satisfactory, in my experience. Both these drugs can be administered by injection.

Pain.—In the graver forms of the disease, particularly the caseous or abscess type, abdominal pain is sometimes distressing. Laudanum stipes may give relief, but it may be necessary to give some preparation of opium by the mouth or as a suppository, or codeina or morphia by hypodermic injection.

External Applications.—Medication applied to the abdominal wall is one of the oldest forms of treatment, and still has, rightly, many adherents. The usual method consists in rubbing $\frac{1}{2}$ drachm of mercurial ointment into the wall of the abdomen at night and covering with a binder. Recent experimental work by Rachford and others has shown that the absorption of drugs by the skin largely depends on the vehicle employed. Thus, by using anhydrous lanoline as the basis various drugs can be administered efficaciously. The method adopted by the writer is that described by Rachford. The skin is well washed and dried, and \mathfrak{z} of the ointment, consisting of \mathfrak{z} . cresote or guaiacol to \mathfrak{z} i. anhydrous lanoline, is rubbed in, covered with a binder, and left undisturbed till the next application in the following evening. Burney Yee's injection of \mathfrak{z} ii. of a mixture of equal parts of iodoforn ointment and cod-liver-oil, applied twice daily, is also valuable.

Tubercle Treatment.—Tuberculous peritonitis is one of the varieties of tuberculous infection most favourably influenced by this method of treatment. It

should be tried in the more severe as well as in the milder cases which are not progressing. It is surprising in some cases how rapidly the intra-abdominal masses diminish in size. It is most important to begin with small doses; otherwise severe reaction may be set up in the mesenteric glands, with constitutional disturbances and a great increase of abdominal pain and tenderness.

For a child of two years a dose of $\frac{1}{100,000}$ milligramme, or 0.001 cubic millimetre, tuberculin T.R. may be administered as the initial dose, and, if no reaction follows, ten days later $\frac{1}{50,000}$ milligramme, or 0.002 cubic millimetre, given, and repeated at intervals of ten days.

Clive Riviere, an advocate of large doses, recommends that the dosage should be calculated according to the weight of the child, on the basis of an average adult dose being about $\frac{1}{3,000}$ milligramme, or 0.05 cubic millimetre, tuberculin T.R. for a man weighing 10 to 12 stone. Thus, for a child weighing 2 to 3 stone the dose should be approximately one-quarter of the above, or $\frac{1}{12,000}$ milligramme, or 0.012 cubic millimetre. It is advisable to begin with a dose considerably lower than this, especially in young children, and gradually to increase it until a reaction is produced, as indicated by slight malaise, loss of appetite, or alteration of temperature. When this effect is reached, the same dose should be repeated at fortnightly intervals. If at a later stage of treatment this dose becomes ineffective, it should be further increased. Success in tuberculin treatment is shown by improvement in appetite, gain in weight, and improvement in the local condition. Loss of weight, on the other hand, is an important indication that the treatment is unsuccessful or that the dosage needs revision. Hypodermic injection is preferable to administration by the mouth. Seeing that a large proportion—more than half—of the cases of tuberculous peritonitis is due to infection by the bovine bacillus, it may be ultimately found that in some cases tuberculin made from the bacilli of the bovine type is more efficacious than a human tuberculin, but on this point further investigations are necessary.

3. Surgical Treatment.—The relative advantages of surgical over medical treatment in the ascitic type of the disease have been much discussed. There is no doubt that a considerable proportion of cases of this, the most favourable form of tuberculous peritonitis, recover without operation, and it is a matter of question whether surgical treatment gives as good or better results. It is difficult to form an opinion from published statistics of results. The explanation given by Watson Cheyne in 1899 of the successful results attending laparotomy in the ascitic cases was that removal of the effusion is followed by subsequent outpouring of fluid rich in antitoxic properties, a view restated by Wright and Douglas in 1907 as follows: "That success is satisfactorily accounted for by the replacement of a stagnant lymph which has forfeited much of its antibacterial nature by a fluid of higher efficacy, freshly derived from the circulating fluid." Further arguments advanced in favour of laparotomy may be briefly stated as follows: that exposure of the peritoneum to the air, oxygen, or solutions which may be applied to it, brings about retrogressive changes in the tuberculous process; that a localized mass of infected glands may be discovered and possibly removed; that the tight bandaging necessary after the operation is of use in promoting rest to the abdominal contents.

The procedure which the writer has adopted for some years past is a compromise. If after medical treatment, carefully applied on the lines already suggested for a month to six weeks, no improvement has resulted either in the general or the abdominal condition, or there is a steady increase in the amount of ascites,

laparotomy is advised. Simple incision and removal of the fluid as completely as possible is sufficient, and is preferable to paracentesis. There does not seem to be anything gained by replacing the fluid with normal saline or other solutions, or with oxygen. The writer has recently had an opportunity of seeing the results of incision and drainage in the case of a child of three years. At the operation the peritoneum was found to be densely studded with large yellow tubercles. The child made a rapid and complete recovery, but a year later died of tuberculous meningitis. At the autopsy the peritoneum was free from tubercles and appeared absolutely normal, except for a small patch of delicate adhesions in the left iliac fossa. The mesenteric glands were small, and contained a few partially calcareous foci. The bronchial glands were caseous, and were regarded as the probable focus of the terminal tuberculous dissemination. There seems little doubt, however, that in a proportion of cases an equally favourable result may be brought about by purely medical treatment.

Surgical treatment may be required in the caseous variety. Collections of pus, generally associated with the presence of pyogenic organisms, should be dealt with by surgical measures as soon as their presence is recognized. Great improvement sometimes follows the free drainage of collections of pus and caseous material. Intestinal obstruction may occur in this or the other form from kinking of the gut or by the formation of bands of adhesions.

In the acute appendicular variety the appendix and adjacent mass of glands should be removed. Ebersole states that the immediate risk of operation is not great, though the ultimate result must remain doubtful owing to the uncertainty as to the existence of other infected foci. Operation is contra-indicated in cases in which general infection has taken place and in cases with advanced tuberculous lesions elsewhere.

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ASCITES.

Ascites may occur in intra-uterine life, and even obstruct labour, or it may manifest its presence very shortly after birth in infants affected by congenital syphilis. The ascites in such cases is the result of a peritoneal inflammation, set up probably by some pyogenic organism owing to lowered resistance caused by specific disease. The condition is rare; it has been described as "fetal ascites."

The most frequent causes of ascites in children is some form of acute or chronic peritonitis, and with this must be included the not very common condition called "polyserositis" or "polyserositis," in which there is an association of pericarditis, mediastinitis, pleurisy, and peritonitis. Nephritis and *marasmus cordis* often give rise to ascites to a very considerable degree. Cirrhosis of the liver, both

of the syphilitic and multilobular type, is another cause; in biliary cirrhosis (Hansen's type) ascites is rarely present. Pressure on the portal vein, such as may be caused by tuberculous or lymphadenomatous glands, or new growth, or blocking of the vein, as in *pylphlebitis*, may give rise to a large effusion of ascitic fluid. Free fluid in the peritoneal cavity can sometimes be detected in leukemia and severe anemia. The condition known as "essential edema of children"—namely, general edema without renal disease—is another cause. In the form met with in infants during or just after an attack of epidemic diarrhoea, ascites may accompany the general edema. New growths of the peritoneum or visceral organs may cause ascites.

The character of the fluid depends on the cause of the ascites. When the ascites is the result of a peritoneal inflammation, the fluid is classed as an "exudate"; it has a specific gravity of 1020 to 1024, and is rich in cell elements. The character of the cells depends on the infection. In tuberculous peritonitis of the ascitic form of pure or unmixed type lymphocytes predominate. With pyogenic



FIG. 18.—ASCITES IN A GIRL OF SIX YEARS, WITH CIRRHOSIS OF THE LIVER, SHOWING THE CHARACTERISTIC APPEARANCE OF THE ABDOMEN.

infections, such as the streptococcal and pneumococcal forms, the polymorphonuclear leucocytes are present in large numbers. The ascitic fluid in cases of syphilitic cirrhosis gives a positive Wassermann reaction. The fluid in non-inflammatory conditions, such as *meckel's diverticulum*, and obstruction of the portal vein, is of the nature of a "transudate," and is of somewhat lower specific gravity (1018 to 1020), and contains few cell elements, these being chiefly endothelial cells. In very acute inflammation, and in tuberculous peritonitis the fluid may contain blood.

DIAGNOSIS.—The recognition of free fluid in the peritoneal cavity is usually easy, provided that attention be paid to (1) the shape of the abdomen, (2) the position of the area dull to percussion, (3) the shifting character of the dull area. In rickety children with chronic gastro-enteritis the distended condition of the abdomen may simulate the presence of an effusion—a mistake which may be avoided by noting carefully the position and character of the dull area. Distension in the flanks due to an overloaded colon is not uncommon in such cases, but does not materially shift with change of position. Another possible source of error in

female children is the presence of a large thin-walled ovarian cyst. In such a case, with the child in the recumbent position, the flanks are usually resonant, and the central area of the abdomen is dull, with a rounded margin convex towards the epigastrium; whereas with free fluid in the peritoneal cavity the flanks are dull, the central area resonant, and the rounded margin convex towards the pubes.

Sometimes a thrill may be made out in the umbilical region by placing the child in the genu-pectoral position, that region being then the most dependent part. Some of the very rare peritoneal growths, such as myxosarcoma, which may completely fill the peritoneal cavity, can closely simulate ascites and give rise to a definite fluid thrill.

TREATMENT.—Ascites in children, unless due to an acute peritonitis, calls for paracentesis much less often than in adults. Removal of the fluid by paracentesis is not often required except in cases of morbus cordis, nephritis, or cirrhosis of the liver. The indications for paracentesis are the same as with adults: considerable and increasing distension, embarrassment of respiration or of the heart, displacement upwards of the diaphragm. "Diuretics, cardiac stimulants, purgatives, etc., often suffice without paracentesis." The method of performing paracentesis in no way differs in children from that employed in adults.

Chylous Ascites.—In this form of ascites the fluid, owing to the presence of fat, resembles milk, and on standing the fat may rise to the surface, forming a definite layer. It is due to interference with the thoracic duct or receptaculum chyli by adhesions or the pressure of tumours or glands. It may be met with in tuberculous peritonitis, or in cases of rupture of a mesenteric cyst. In true chylous ascites the fat particles are finely divided, and there are no cells undergoing fatty degeneration. In another condition, described by Batty Shaw, ascitic fluid may be found presenting a white or milky appearance, in which the white colour is due to the presence of degenerated and fatty cells derived from the peritoneum in cases of tuberculous peritonitis and chronic peritonitis; this is called chyliform or "fatty" ascites. An opalescent fluid with a milky appearance is sometimes met with in ascitic cases which owes its peculiarity, not to the presence of fat, from which it is free, but to the presence of some substance such as lecithin or nucleo-albumin. These forms are all rare in children.

PERITONEAL TUMOURS.

New growths of the peritoneum are rare in children, but both innocent and malignant forms are met with. Cysts of the peritoneum will be considered separately.

1. Innocent or Non-Malignant Tumours.—(a) *Fibroma.*—The small nodules of fibrous tissue occasionally formed as the result of a chronic peritonitis, especially of the tuberculous variety, should not be regarded as true fibromata. True fibromata occur usually in the mesentery.

(b) *Lipoma.*—Instances have been recorded of lipomata of large size, weighing as much as several pounds, removed from the abdomen of quite young infants. They appear to originate in the retroperitoneal tissue, generally on the right side of the abdomen or between the layers of the mesentery. Both fibroma and lipoma may exhibit myxomatous change.

(c) *Angioma.*—Cavernous angioma has been described.

(d) *Myoma and Sarcoma* may arise in the retroperitoneal tissue.

2. Malignant Tumours.—As Robertson points out, malignant growths of the peritoneum may arise from (1) the endothelial lining, its bloodvessels and lymphatics; (2) the serous layer of connective tissue; (3) the remains of the Wolffian bodies, ducts of Müller, accessory adrenal bodies, and teratomas. In connection with these structures, the following primary growths may arise:

- (a) Sarcoma.
- (b) Endothelioma.
- (c) Angiosarcoma.

Sarcoma usually starts in the retroperitoneal space, or between the layers of the mesentery or omentum. Retroperitoneal sarcoma may cause ascites, and thus simulate tuberculous peritonitis, or give rise to oedema of the legs by pressure on the vein cava.

Diagnosis in cases of malignant disease of the peritoneum is most difficult, and is practically impossible without laparotomy. The growth may extend into the peritoneal cavity and completely fill it up, and if the tumour has a myxomatous character it may give rise to definite fluctuation and a fluid thrill. Some of these tumours, especially if polycystic, are removable, and it is therefore advisable that an exploratory operation should be performed without delay.

Secondary Malignant Tumours.—These are usually of a sarcomatous nature, though rare instances of carcinomatous growths are met with in cases of primary growth in retroperitoneal teratomas and Wolffian bodies.

CYSTS OF THE PERITONEUM.

Encysted collections of fluid the result of an inflammatory process such as tuberculous peritonitis may closely simulate a peritoneal cyst. Such collections are most commonly found in the pelvis and in the omentum.

Mesenteric Cysts.—These are the commonest variety of peritoneal cyst met with in children. They may be single or multiple, and are generally situated close to the intestine. They usually contain clear fluid, though blood or chyle may be also present. When the contents are milky owing to the presence of chyle, they are sometimes called "chylous cysts." Mesenteric cysts are regarded as either the result of distension of lymphatics or of embryonic origin. They are of various sizes, and usually form movable tumours, more commonly on the right side of the abdomen. Resection due to the intestine can generally be made out in front of the cyst. Usually there are no symptoms except some slight discomfort unless the cysts are of large size.

Blood-Cysts.—As the result of injury effusions of blood take place, forming large cystic collections in various situations such as the retroperitoneum, especially in the neighbourhood of the pancreas, or in the mesentery, etc. The swelling sometimes does not arise till some days after the injury, and it has been suggested by Fisher and others that a localized peritonitis is first set up, and that the hæmorrhage takes place subsequently, owing to the rupture of delicate vessels in the newly-formed adhesions. It is probable that some of the so-called "pancreatic cysts" are of this nature. These blood-cysts contain altered blood; the lining is rough and fibrous.

Urachal Cysts.—These lie in front of the peritoneum, between the navel and bladder, and are due to the distension of the non-obligated urachal duct, which passes from the fundus of the bladder to the umbilicus.

Allantoic Cysts.—Cysts to which this name has been given are collections of fluid occasionally found in the pelvis or lower part of the abdomen. They are in most cases, probably, the result of former peritonitis, which has given rise to an encysted collection of fluid.

Hydatid Cysts.—These may be either single or multiple. They are found in the mesentery, omentum, or general peritoneum. They may closely resemble mesenteric cysts.

Dermoid Cysts (Teratomas) may be found in the retroperitoneal space, in the mesentery, transverse mesocolon, and omentum.

DIAGNOSIS.—The diagnosis of the nature of a peritoneal cyst may be very difficult, except in the case of a blood-cyst in which there is a definite history of injury. These are usually of large size, and it may be possible by exploration with a needle to ascertain the hæmorrhagic character of the contents. It is inadvisable to use an exploring needle in other cases, as this operation is not free from risk, especially with hydatid cysts.

Chronic peritonitis with a localized collection of fluid may simulate a cyst, and the history of the case should be carefully considered.

TREATMENT.—This is purely surgical. Laparotomy should be performed and the cyst removed. Blood-cysts should be evacuated and scraped.

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CHAPTER VI

DISEASES OF THE NOSE AND THROAT

F. A. ROSE

CONGENITAL ATRESIA OF THE CHOANÆ.
ACUTE RHINITIS (CORRUA).
MEMBRANOUS RHINITIS.
TUBERCULITIS.
CHRONIC ENLARGEMENT OF THE TUBERLS.
CHRONIC RHINITIS.
RHINITIS SIGNA.
LEPUS.
SYPHILIS.
ABSCESS OF THE NASUM.

EPISTAXIS.
FOREIGN BODIES IN THE NOSE.
ADENOIDÆ.
RATNOPHARYNGEAL ABSCESS.
TUMORS OF THE NOSE.
LARYNX.
LARYNGITIS.
CHRONIC LARYNGITIS.
FOREIGN BODIES IN THE LARYNX.
LARYNGEAL NEW GROWTHS.

CONGENITAL ATRESIA OF THE CHOANÆ.

INTRODUCTION.—The condition is congenital, and is characterized by the presence of a diaphragm in the posterior part of the nasal fossæ. The right side is obstructed more frequently than the left, but in about one-third of the cases both sides are affected.

SYMPTOMATOLOGY.—If both choanæ are affected there is complete nasal obstruction. The patient is forced to breathe through his mouth, the sense of smell is in abeyance, and speech lacks nasal resonance. Owing to the patient being unable to clear his nose by blowing, mucus collects in the nasal fossæ and runs out of the anterior nares. The constant running is apt to lead to an eczematous condition of the upper lip. When only one nostril is obstructed, the patient is able to breathe through the nose, and keeps his mouth shut; and in such a case the chief complaint is of the mucus dribbling from the affected nostril. Deafness is a common complication.

PATHOLOGY.—The diaphragm is a vertical plate situated about $\frac{1}{2}$ inch in front of the choana. Typically it consists of bone covered on each side by mucous membrane, but in many cases it is partly membranous, and a perforation is often present near its centre. Its presence undoubtedly depends on some error in development, and the most probable explanation is that the bucco-nasal membrane persists and becomes supported by an ingrowth of mesoderm (Hochstetter). It is worthy of note that the naso-pharynx is nearly always large and free from adenoids.

TREATMENT.—The partition must be perforated by means of a burr, and the opening made as large as possible by chisels and cutting forceps. There is a great

tendency for the opening to contract, so that, if there is any doubt as to the sufficiency of the aperture made in this way, it is advisable to remove the posterior part of the vomer as well.

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ACUTE RHINITIS (CORYZA).

The term "coryza" is applied to any condition of the nose in which there is excessive secretion of mucus, accompanied by swelling and other evidence of inflammation of the nasal mucous membrane. The cases can be divided into three groups, according to the supposed cause:

1. The first group contains those cases in which the cause is a bacterial invasion of the nasal mucous membrane. The symptomatic rhinitis of measles probably belongs to this group.

2. The second group contains those cases in which the irritant is a chemical substance such as iodine and arsenic, and also those cases due to the inhalation of the pollen of certain grasses.

3. The third group contains those cases in which there is no sufficient evidence of bacterial invasion nor of chemical irritation, and which are therefore believed to be due to some nervous derangement. The term applied is "vasomotor rhinitis" or "paroxysmal rhinorrhea."

Microbic Rhinitis.—Acute rhinitis due to microbial invasion. It is now generally accepted that the "common cold or the head" is due to infection of the nose.

ETIOLOGY.—Children are even more susceptible to this affection than adults. It prevails during the rapid changes of weather which occur during the spring and autumn, and is often epidemic. When the patient is an infant the mother or nurse must always be suspected as a source of infection.

SYMPTOMS.—The patient feels chilly, and complains of headache and lassitude. The temperature rises to 100° or 101° F. An itching sensation in the nose leads to attacks of sneezing, and this is followed by a flow of thin, watery secretion. The swelling of the nasal mucous membrane causes the nose to become more and more obstructed, until it is impossible to breathe through it. The sense of smell is lost entirely, and the sense of taste is much impaired. In infants the symptoms are apt to be particularly severe. Slight bleeding from the nose is frequent. The small size of the nasal passages rapidly causes complete obstruction, rendering sucking difficult, if not impossible; even feeding with a spoon may be difficult. Moreover, at night sleep is broken, and suffocative attacks are frequent, owing to the infant's reluctance to breathe through its mouth. After two or three days the discharge becomes more tenacious, from the presence of mucus, and opaque from the presence of pus cells; the proportion of pus varying much in different cases. Gradually the nose becomes patent; the discharge becomes scanty and tends to dry, leading to the formation of crusts, and in about ten days the attack subsides.

COMPLICATIONS.—In every attack of acute rhinitis there is a risk of the infection spreading to the Eustachian tubes, and so reaching the middle ear. Even more commonly the larynx becomes infected, and tracheitis and bronchitis result. The

accessory air-sinuses of the nose are very poorly developed in children. The maxillary sinus, although present at birth, remains a relatively small cavity until the eruption of the second teeth. The other sinuses are for the most part represented by small diverticula springing from the ethmoid, and do not become fully developed until after the age of ten years. Suppuration in any of the cavities as a result of nasal infection is an exceedingly rare event in a child, and the writer has never met with it under the age of nine years.

PATHOLOGY.—The mucous membrane is reddened and swollen to a degree which varies with the intensity of the inflammation. The secretion in mild cases is merely turbid from the presence of desquamated epithelium, or may be yellow from the large proportion of pus cells. It is highly probable that some bacteria are more apt than others to give rise to pus formation. The bacteria which are found most commonly are the pneumococcus, streptococcus, staphylococcus pyogenes aureus, *necrobacillus catarrhalis*, influenza bacillus, and certain diphtheroid bacilli, notably the *Bacillus septus* and *B. coryne segmentatus* of Clutley. The latter group of bacilli is particularly interesting, in that attempts have been made to show that members of it are the specific cause of the "common cold in the head."

The **DIAGNOSIS** is made without difficulty, but it is important to determine whether or not the rhinitis is part of a general infection, such as measles.

TREATMENT.—When the attack is severe enough to require treatment, the patient should be confined within doors and kept warm; this is effected most readily by confining him to bed. The air should be kept warm and as fresh as possible, but a draught aggravates the symptoms. A hot bath before going to bed is beneficial. Warm drinks add to the comfort of the patient, and for this purpose black-currant tea answers admirably. A small amount of lanoline should be smeared on the upper lip, on the alae nasi, and inside the nose. In the case of infants a few drops of menthol-oil (menthol, 2½ grains; liquid paraffin, 1 ounce) should be dropped into the nose from a pipette. If the nose is too much obstructed for the menthol to enter, it should be preceded by a few drops of hydrogen peroxide (5 volumes). In the case of older children a few drops of oil of eucalyptus should be sprinkled on cotton-wool and the vapour inhaled.

Rhinitis due to Irritants.—This is quite uncommon in children. Vapours such as chlorine gas, powders such as ipecacuanha or potassium bichromate, and drugs such as adrenalin chloride, are capable of causing acute rhinitis when applied to the nasal mucous membrane. The rhinitis which ensues rapidly follows the application of the irritant, and soon disappears, seldom lasting more than two or three days. The internal administration of iodine and arsenic compounds has a similar effect in susceptible persons.

HAY FEVER.—Hay fever is the most impressive example of acute rhinitis due to a local irritant. The pollen of certain grasses which usually flower in June is the common cause in this country. Roses act in the same way, and in America ragweed pollen is a well-recognized irritant. Association with certain animals, particularly horses, may have a similar effect.

SYMPTOMS.—The symptoms begin within a few minutes of exposure to the irritant. There is an intense itching or pricking in the nose, which leads to violent attacks of sneezing and a profuse watery discharge from the nostrils. The external nose is swollen and the interior obstructed. The conjunctiva is injected, and

sometimes in a condition of chemosis. The eyes water, and the whole face becomes puffy. The patient is reduced to a state of helpless misery. Heat and bright sunlight intensify the symptoms, which abate or are entirely absent during cool rainy weather. An examination of the nose shows the mucous membrane to be oedematous and covered by clear watery secretion.

PATHOLOGY.—The most remarkable feature of this disease is that it only attacks a small proportion of the population, the great majority being immune. Temperament is said to be more susceptible than those living in the country, and males are believed to be attacked more frequently than females. No adequate explanation of this susceptibility has yet been offered. It has been said that septal spurs and deviations, hypertrophies of the mucous membrane, polyps, and such-like, are responsible, but their removal is usually ineffectual. It is known that a small dose of arsenic administered to a susceptible person is capable of producing acute rhinitis and conjunctivitis. In a similar manner, we must regard the subjects of hay fever as having a peculiar susceptibility to the action of pollen and other microscopic dust. Whether this vulnerability resides in the nasal mucous membrane alone or in other tissues of the body is not known at the present time.

DIAGNOSIS.—The symptoms follow so rapidly on the cause that there is rarely any difficulty in diagnosis.

PROGNOSIS.—The prognosis must be guarded, particularly in a child. As will be seen later, we have no reliable means of treatment, and the progress of the disease varies widely in different cases. A mild attack one year may be followed by a much more severe attack in the following year, with recurrence year after year in spite of treatment. Moreover, the character of the attack may change, and nocturnal attacks of asthma be added to the nasal symptoms, or to some extent replace them. In other cases hay fever appears to be the starting-point of toxicopyral rhinorrhoea.

TREATMENT.—The most efficacious method of treatment is to avoid contact with the pollen. This can be accomplished by taking a sea-voyage, by residence in Heligoland, Mont Dore, or some other resort, during the pollen season. Failing this, an attempt should be made to render the nose less sensitive. Dunbar endeavours to confer a passive immunity by the application to the eyes and nose of a serum obtained from an immunized horse. Numerous successful cases are reported, but some patients obtain no benefit. Scheppegrell endeavours to obtain active immunity by making the patient inhale pollen dust several times daily for a period of six weeks before the expected attack. Attempts are also made to render the nasal mucous membrane less sensitive by the application of the galvanic current to certain sensitive areas. These areas can be readily discovered by gentle probing, and are commonly found on the upper surface of the inferior turbinate, and on the septum near the anterior end of the middle turbinate bone. A considerable amount of success has followed this treatment also, but even repeated applications fail to give relief in some cases. When the foregoing measures fail, the writer has found the application of an ointment containing 30 grains of anaesthesia and 2 grains of atropine sulphate to the nostrils to be of great service in warding off attacks, if the ointment is applied before the patient is exposed to the pollen. Two or three applications may be necessary during the day. When the symptoms are once developed, the intolerable stuffiness can be relieved by a spray containing cocaine

and adrenalin chloride; the effect is only temporary, and reaction follows, but, for the time being, comparative comfort is attained. There is serious objection to providing a patient with a spray containing cocaine, save in quite a minute quantity, and on this account great caution must be observed. Patients soon find out that they suffer less in a cool, dark room than in the hot, bright sunshine, and wearing tinted goggles or a thick veil affords some relief.

Paroxysmal Rhinorrhœa is closely allied to hay fever in its manifestations. Comparatively trivial alterations in temperature, such as passing from a hot room to a cold, or vice versa, bring on violent attacks of sneezing, accompanied by profuse watery running from the nose. Attacks are particularly likely to occur on rising in the morning, and again about four to six in the afternoon; they recur daily with great regularity, and are quite independent of the pollen season, usually being much more troublesome in the winter than in the summer. The condition is very uncommon in children, the majority of patients being over fourteen years of age. A certain amount of chronic rhinitis is usually present, and, for some reason which is not clearly understood, the nasal reflexes of these patients are unduly excitable.

MEMBRANOUS RHINITIS.

Membranous rhinitis is an infective disease due to the diphtheria bacillus; it is characterized by the formation of membranous exudate on the mucous membrane. The condition is by no means uncommon, but it is probable that a considerable number of cases remain unrecognized.

ÆTIOLOGY.—Age has a decided influence on the incidence of this disease. It is most common between the ages of three and eight years; after that age fewer and fewer cases are seen, and the writer has only seen three cases in which the age was over twenty. There are no predisposing causes known except exposure to infection, and it has been noted that it more commonly arises from contact with another case of membranous rhinitis than from contact with a case of faucial diphtheria.

SYMPTOMS.—The chief symptoms are nasal obstruction, discharge from the nose, and bleeding. At first the symptoms resemble those of a severe cold in the head. The nose becomes stuffy, and the obstruction increases until nasal respiration is impossible and there is loud snoring at night. At the same time there is a watery discharge from the nostrils, very irritating to the upper lip, and making it red and sore. Later the discharge becomes purulent, and small shreds of membrane may be blown out. Bleeding also occurs in the majority of cases, but is seldom excessive. The general health is only affected to a slight degree. The temperature rises to 98° or 100° F., seldom higher, and often not as high. The child becomes pallid, and is disinclined to take its food, but goes about as usual, and is considered to be merely "out of sorts." **Unilateral Form:** In about 20 per cent. of the cases only one side of the nose is attacked. The condition is much more persistent than an ordinary cold; from four to six weeks is the common duration, but it may last two or even three months. When the nose is examined, a thick white membrane is found blocking the nasal fossa; it covers both the septum and the outer wall, and is strictly limited to the mucous membrane. Pieces of it can be torn off quite easily, but this proceeding always causes bleeding. The pharynx is not affected. Otitis media is set up occasionally. There are no sequelæ, such as paralysis.

PATHOLOGY.—The lesion is an inflammation of the nasal mucous membrane due to the diphtheria bacillus, and resulting in the formation of a false membrane. Owing to the large area of membrane, it would seem probable that a sequela such as paralysis, due to absorption of toxin, would be common, and the conspicuous absence of such sequela has led some to doubt if the bacillus which is invariably found in these cases is really the diphtheria bacillus. Bacteriologically, however, the bacillus is identical with the Klebs-Löffler bacillus, and is equally virulent to animals.

DIAGNOSIS.—The bilateral form must be distinguished from a cold in the head. The presence of membrane, the frequent bleeding, and abundant diphtheria bacilli, make this easy. The unilateral form must be diagnosed from a foreign body in the nose (see Foreign Body, p. 293).

PROGNOSIS.—The prognosis both as regards life and local recovery is excellent and unfavourable complications are very rare.

TREATMENT.—It is important to isolate the patient, as he may infect others with the diphtheria bacillus. It is usual to inject diphtheria antitoxin, and to syringe the nose with a mild antiseptic. But, so far as is known, it is not certain that this treatment has much effect in shortening the course of the disease.

TONSILLITIS.

INTRODUCTION.—Before entering upon a description of the inflammatory diseases of the tonsil, it is necessary to make a short statement with regard to its structure and function.

1. *Structure.*—The tonsil is essentially a lymphatic gland, but with two important points of difference: first, that the fibrous tissue capsule which encloses the ordinary lymphatic gland is on the pharyngeal surface of the tonsil replaced by stratified squamous epithelium; secondly, that a number—usually about twelve—of pockets or crypts lined by the same squamous epithelium run deep into the substance of the gland. The largest of these crypts, the so-called "supratonsillar fossa," lies within, and not above, the limits of the uppermost part of the gland. The importance of these differences lies in the greater extent of absorptive surface, and in the liability of the crypts to become choked with bacteria and debris.

2. *Function.*—It is commonly believed that the main function of the tonsil is to act as specialized lymphoid tissue, so situated as to drain the mucous membrane of the larynx and naso-pharynx; this view regards the tonsil as a portion of the protective mechanism of the body. Whether this assumption is correct or not, it is certain at any rate that the tonsils, even in a healthy condition, harbour multitudes of micro-organisms, and that they are with great frequency the primary sites of bacterial infection. An interesting speculation put forward by Goss is that the crypts serve as culture tubes for bacteria, and that the absorption of the products of bacterial growth tends to establish immunization of the body tissues. In spite, however, of much investigation, it must be admitted that our knowledge of the function of the tonsil is lacking in precision.

Tonsillitis in children may be considered under two heads:

1. Acute tonsillitis.
2. Chronic enlargement of the tonsils.

Acute Tonsillitis.—Acute tonsillitis forms a prominent feature of several acute specific fevers, especially scarlet fever, diphtheria and measles. Apart from these, many cases of acute tonsillitis occur in which the infection appears to be primary in the tonsils. Among these primary infections, mention must be made of the tonsillitis which so frequently heralds an attack of acute or subacute rheumatic fever (vide Chapter XVIII., p. 253).

Lacunar Tonsillitis (Follicular).—**Ætiology.**—**Season.**—Attacks may occur at any time of the year, but particularly during dry, dusty weather.

Infectivity.—1. Contagion: The disease is contagious; several members of a family may fall ill one after the other. In many cases it is impossible to trace the source of infection. Thus, an unusually severe rhinitis in a mother was followed by a sharp attack of lacunar tonsillitis in her two sons. Similarly, the influence of contagion is often noticeable in the epidemic outbreaks which occur in hospitals and institutions.

2. Contaminated air: There are good grounds for believing that air contaminated by sewer gas or rubbish-heaps can be a source of tonsillar infection, although bacteriological evidence is difficult to obtain.

3. Contaminated milk: In several instances severe epidemic outbreaks of tonsillitis have been traced to contamination of the milk-supply.

4. Chronic inflammation of the tonsil predisposes the patient to a recurrence of acute attacks.

SYMPTOMATOLOGY.—The onset is sudden. There is sharp pain in the throat, causing intense discomfort when swallowing. Food is chewed, but only swallowed slowly, and in some cases is refused altogether. (It must be borne in mind, when dealing with children, that they may deny the existence of a sore throat, even when acute inflammation of the tonsils is present.) At the height of the inflammation liquids may be returned through the nose. The tongue is furred, the breath is foul, and tender enlarged glands can be felt at the angle of the jaw. The constitutional disturbance is severe. Pyrexia is always present, the temperature ranging from 101° to 106° F. Lassitude, amounting to prostration in the severe cases, is always present; there are aching pains in the back and limbs, constipation, and loss of appetite. It is remarkable how ill a patient may become in a short time. The tonsils are intensely red and much swollen, projecting beyond the pillars of the fauces; on the surface a number of yellowish-white patches of exudation are visible. Each is about $\frac{1}{4}$ inch in diameter, and investigation with a blunt probe shows that it corresponds with the mouth of a crypt. The exudation can be wiped away quite easily. It is sometimes grey in colour, covering a large area of the tonsil, and very tenacious, and may then have a considerable resemblance to the false membrane of diphtheria.

VARIETIES.—In some cases of acute tonsillitis no exudation is visible; the condition is then described as *parachymatous tonsillitis*. Ulceration may also be present. The ulcers may be circular and superficial, and cause little disturbance, or deep and sloughing, and accompanied by grave general symptoms.

PATHOLOGY.—The micro-organisms most commonly found are streptococci and pneumococci. In addition, other bacteria are common, such as the micrococcus catarrhalis, diptheroid bacilli, and staphylococci, while influenza bacilli have been found. It seems probable that acute tonsillitis may be caused by a number of different organisms, but our present knowledge does not allow us to associate any

particular clinical variety with any particular organism. Moreover, the same organism appears capable of giving rise to each of the different clinical varieties of tonsillitis. Thus, streptococci may appear to be the predominant infection in the lacunar form, in the parenchymatous form, and in the ulcerative variety. The present writer's belief is that the more severe the attack, the greater is the probability that streptococci are the causal organisms.

DIAGNOSIS.—As already mentioned, many children do not complain of any soreness of the throat. It is therefore imperative to examine the fauces of every sick child. It is remarkable how frequently such examination will reveal an unsuspected tonsillitis. The next step is to exclude the infectious fevers, such as scarlet fever, measles, and diphtheria. Scarlet fever and measles rarely cause trouble, and will not be discussed further. The chief difficulty arises in connection with diphtheria. The typical false membrane of diphtheria is brilliantly white; it is situated on the upper part of the tonsil, is surrounded by a narrow, acutely red zone, is tough in consistence, and cannot be wiped away, but can be lifted up and peeled off with forceps, leaving a bleeding surface. The membrane forms one continuous sheet, and commonly extends on to the pillars of the fauces and the palate. On the other hand, the exudation of lacunar tonsillitis does not extend on to the pillars of the fauces, can be wiped away, and is distributed over the surface of the tonsil in patches which correspond to the mouths of the crypts. In typical cases, therefore, no difficulty arises, but many cases both of diphtheria and tonsillitis are atypical, so that the throat may present a very baffling appearance. Hence the clinical rule has arisen that, when any exudation is present on the tonsils of a child, a bacteriological search for the diphtheria bacilli must always be made.

PROGNOSIS.—As a rule resolution occurs within three or four days, and is followed by rapid and complete recovery. Persistence of acute or progressive symptoms beyond this time must give rise to a suspicion that suppuration has occurred, and that a tonsillar or peritonsillar abscess is forming. Important and severe sequelæ have been recorded. It has often been noted that tonsillitis is an initial symptom of acute rheumatism. Acute nephritis may follow acute tonsillitis, even when diphtheria and scarlet fever can be excluded. Pericarditis and enditis, due to the *Streptococcus larynx*, have been recorded in two cases, the initial lesion being a follicular tonsillitis. Compared with the frequency of the disease, such complications are rare. A permanent enlargement of the tonsils may remain after a severe attack.

TREATMENT.—In the case of small children local treatment is difficult and keenly resented. Nevertheless, an endeavour should be made to clean out the mouth with a weak mixture of hydrogen peroxide and Listerine, and hot fomentations should be applied to the outside of the neck in order to relieve the pain. Older children can suck formalin tablets, and the throat can be painted with a mixture, containing 20 minims of Iod and 4 drachms of glycerine to the ounce, twice daily. Swallowing is less painful if ice be held in the mouth for five minutes before each meal. Of drugs, sodium salicylate is the most effective, and should be administered in doses suitable to the age of the patient, but in some cases there is very little response. Tonsillar and peritonsillar abscesses must be opened, but any other operation upon the inflamed tonsil is not advisable. The air of the room in which the patient is confined should be warmed, and a steam-kettle is often helpful for the accompanying laryngitis. Considerable debility commonly follows an

attack, and should receive treatment, a visit to the seaside being particularly beneficial.

Suppurative Peritonsillitis (Quinsy).—Although a very common sequela of lacunar tonsillitis in older patients, peritonsillar abscess is uncommon in children, and in infants it appears to be replaced by retropharyngeal abscess. The onset is that of a tonsillitis, but on the third or fourth day, when amelioration of the symptoms might be expected, the condition becomes aggravated. Swallowing is almost impossible; there is a throbbing pain in the side of the throat, shooting up to the ear, which prevents the patient from sleeping. The mouth is full of saliva and frothy mucus; the tongue is yellow with thick fur, the breath offensive, and it becomes more and more difficult to open the mouth. The soft palate in the neighbourhood of the tonsil is found to be red, swollen, and bulged forward; the tonsil is enlarged and projects beyond the middle line. The uvula is oedematous. Fluctuation may sometimes be detected in the bulged palate, but generally the tenderness is too great to permit of palpation. Left untreated, the abscess bursts spontaneously. In some cases the pus can be seen coming from a crypt at the upper pole of the tonsil; in others an opening appears in the palate just above the supratonsillar fossa; whilst in others a careful search fails to reveal the point at which the pus is escaping. Severe hæmorrhage has been known to follow spontaneous rupture of the abscess.

PATHOLOGY.—Opportunities for investigating the situation of the pus by a post-mortem dissection are very uncommon. The clinical features of the disease lead to the belief that the collection of pus lies outside the capsule of the tonsil in the loose tissue of the soft palate, and that the tonsil itself is pushed inwards by the abscess. In some cases small collections of pus are found in the substance of the tonsil or locked up in a crypt; the term "tonsillar abscess" is then applied.

TREATMENT.—The pus should be evacuated at the earliest possible moment. It may be present on the fourth day of illness, and is almost invariably present on the fifth day. If circumstances permit, an attempt should be made to open the abscess through a crypt at the upper end of the tonsil, or through the supratonsillar fossa, by means of a bent probe. Later, when a large abscess is bulging, it should be opened by plunging a pair of sinus forceps through the soft palate into the abscess, and separating the blades as they are withdrawn. In this way a free opening is made, and within a few hours of the escape of the pus the patient experiences great relief.

Vincent's Angina—INTRODUCTION.—In 1896 Vincent called attention to the presence of certain micro-organisms in cases of hospital gangrene, and it is owing to his researches that we are able to differentiate the particular variety of ulceration of the tonsil that goes by his name.

ETIOLOGY.—The disease is mildly infectious; somewhat intimate contact appears to be necessary for its transference from one person to another. The majority of cases are seen in children. Of thirty-two cases reported by J. D. Balleston, twenty-seven were in patients under ten years of age, and the oldest patient was sixteen.

SYMPTOMATOLOGY.—Complaint is made of soreness of the throat, running from the nose, and headache. As a rule only one tonsil is attacked; its surface is covered by a white membrane, which frequently spreads on to the pillars of the fauces and

uvula. The membrane bears a strong resemblance to that of diphtheria, but is usually softer. When the membrane is removed, a deep excavation of the tonsil is exposed, the cavity being filled by soft friable material. Apart from the tonsil, separate ulcers may be found on the gums and on the mucous membrane of the inner surface of the cheek. The breath may be very offensive. The cervical glands show a moderate amount of enlargement, but never suppurate. The constitutional disturbance is slight; the temperature is raised moderately for a few days, and then remains normal during the remainder of the illness. The progress of the disease is slow, and, although healing may occur in a week, the duration is commonly longer; three to six weeks may elapse before the ulcers disappear.



FIG. 24.—VINCENT'S ANGINA. (Gow.)

PATHOLOGY.—Scrapings taken from the ulcer show an abundance of long narrow bacilli with pointed ends (*Bacillus fusiformis*), mingled with large spirochetes. Few other organisms are seen.

DIAGNOSIS.—The depth of the ulceration and the presence of lesions on the cheeks or gums help to distinguish the affection from diphtheria. A film preparation must be made immediately, when the presence of the characteristic fusiform bacilli and spirochetes establish the diagnosis, which is completed by the failure of diphtheria bacilli to grow upon suitable culture media.

PROGNOSIS.—The course is benign, and complications are rare. Recurrence is frequent. A few deaths have been reported. Two cases in which the larynx and trachea were attacked have been reported, both in adults.

TREATMENT.—The mouth must be kept clean and tincture of iodine should be applied to the ulcers. Iodol or methylene blue powder may be used instead and give excellent results.

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CHRONIC ENLARGEMENT OF THE TONSILS.

ETIOLOGY.—Chronic enlargement of the tonsils is an exceedingly widespread affection. It is frequently met with in the second year of life, and from that age onwards is one of the commonest affections of childhood. A strong family history is usually obtainable; all classes of society are affected, and sex has no influence.

The causes which lead to enlargement of the tonsils are the same as give rise to adenoids, and so we find that adenoids are accompanied by enlarged tonsils in about 70 per cent. of the cases; and when the patient is under the age of eight years enlarged tonsils are almost invariably associated with adenoids. Wright believes that an enlargement of the tonsils without infection occurs during dentition at each period of molar eruption. What is more certain is that the presence of carious teeth is a potent cause of chronic tonsillar infection.

SYMPTOMATOLOGY.—The actual enlargement of the tonsils causes but few symptoms, and in many cases is only discovered during a routine examination. When symptoms are present, the voice is altered, and the patient speaks as if something were being held in the mouth, swallowing is slow, there is a tendency to choke, and large hard pieces of food may be rejected. During a cold the tonsils swell, and, when large masses of adenoids are present as well, respiration may be seriously impeded. The recurrent attacks of acute tonsillitis, to which children with enlarged tonsils are subject, are of considerable importance; they lower the general health and seriously interfere with development and education. Febrile attacks without obvious tonsillitis are also common, and enlargement of the cervical glands is almost inevitable. The large tonsils are also dangerous because they are an abode of pathogenic bacteria; for example, diphtheria bacilli may be found, in large numbers, in the crypts for many weeks after an attack of the disease.

PATHOLOGY.—Microscopic examination shows that the enlargement is due to an increase in the size, as well as in the number, of the lymph follicles of which the tonsil is composed. The crypts frequently contain pus and small yellow masses consisting of bacteria and cell detritus. There does not appear to be any relationship between the size of the tonsil and the amount of infective material it contains.

Tuberculous Tonsils.—The frequency of tuberculous deposits in large tonsils has been a matter of much investigation. Nébécourt and Texier examined twenty-two cases without finding satisfactory evidence of tubercle in any. Mathiasen and MacConkey obtained similar results in thirty-four cases. On the other hand, Hord and Wright found tubercle in the tonsils of nine out of twelve patients, and Carmichael in five out of thirteen; these authors were examining patients in whom enlarged cervical glands were the prominent symptom. From these results it may be concluded that tuberculous deposits are present in few cases of simple enlargement of the tonsils, but that where large masses of cervical glands coexist it is not uncommon to find tubercle in the tonsils. In all these cases the tonsil is

probably the primary site of infection, and the glandular enlargement is secondary. The tuberculous tonsil may be large and projecting, but is more often small and pale, very rarely ulcerated. It has no specific character by which the unaided eye can distinguish it from the non-tuberculous tonsil. It should be noted that the tuberculous tonsils found in adults are almost invariably secondary to disease in the lungs, and commonly exhibit ulcers or tubercles on their surface.

Prognosis.—Enlarged tonsils are met with less and less frequently after the age of fifteen, as a result of the tendency of the tonsillar tissue to atrophy. This involution may take many years, and does not always occur, for enlarged tonsils may still be present at the age of forty.

Treatment.—Gargles, paints, and internal medication, have had little effect on enlarged tonsils. If treatment is necessary, removal is the only satisfactory procedure. Tonsils which are moderately enlarged and give rise to no symptoms require no treatment. Removal is necessary wherever the enlargement is sufficient to cause alteration in the voice, when swallowing is affected, and when symptoms of ear disease are present. Even comparatively small tonsils should be removed when they give rise to stroke attacks, or when the patient is subject to recurrent lacunar tonsillitis and to peritonsillitis. The small buried tonsil, which is the seat of chronic inflammation, associated with enlarged glands in the neck, should also be removed, as well as those found in cases of articular rheumatism; and of course those believed to be tuberculous. The chief methods of removal are by the guillotine and by dissection or excision. In the hands of many operators the guillotine is not a very efficient instrument, and a piece of the tonsil, sometimes a large piece, is left behind, with an unsatisfactory result. Nevertheless, it is possible to obtain a thorough removal of the tonsil by means of the guillotine, and this method answers well for those cases in which operation is undertaken on account of the size of the tonsil. When performed on account of recurrent lacunar tonsillitis, quinsy, or tuberculous glands in the neck, it is important that the entire tonsil, including the capsule, should be removed, and the operator must adopt some method by which he is certain of attaining this end. For this purpose the dissection method is more reliable than the guillotine.

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CHRONIC RHINITIS.

CATARRHAL.

RHINITIS SICCÆ.

ATROPHIC.

Catarrhal (Hypertrophic).—Chronic catarrhal rhinitis is an inflammatory condition of the nasal mucous membrane characterized by swelling and excessive secretion of mucus.

ETIOLOGY.—This condition occurs with great frequency in children. The most common cause is an attack, or series of attacks, of acute rhinitis, the second and third attacks supervening before the patient has recovered from the first. The most potent underlying cause of this inability to "throw off a cold" is nasal obstruction, and in a child this is usually due to the presence of adenoids.

SYMPTOMS.—The nose is stuffy; the voice is thick and "nasal" in character. A slight but constant discharge of mucus from the nostrils causes a "dirty nose." Attempts to clear the nose by blowing are only partly successful, and have to be repeated frequently. The patient is particularly liable to fresh colds, and these are apt to be prolonged, and the general nutrition usually suffers.

PATHOLOGY.—The mucous membrane is swollen, particularly that covering the turbinates; its surface is covered by a layer of secretion, which may be clear or mucus-purulent, giving rise to considerable narrowing of the nasal fossæ. The anterior end of the inferior turbinate usually presents a smooth rounded swelling, which pits on pressure and can be made to shrink by the application of vaso-constrictant drugs, such as adrenalin or cocaine. In some cases the surface of the inferior turbinate is nodular, instead of being smooth; the appearance is that of closely set nodules separated by deep pits. This condition is known as "papillary" or "moniform" hypertrophy. The investigations of Kuhn have shown that the lobules consist of soft fibrous tissue, and that there is no ground for believing that there is any increase in the glandular, or in the cavernous layer. The condition cannot be correctly termed either a true hypertrophy or a true papilloma.

DIAGNOSIS.—The diagnosis is not difficult when the history and the local appearances are considered. The important point is to discover the underlying cause, particularly the presence or absence of adenoids. If adenoids are not present, a careful search must be made for other causes of nasal obstruction, such as enlargement of the turbinates, polypus, or congenital atresia of the choanæ. The possibility of syphilitic rhinitis, and of a rhinitis continuing after scarlet or other infective fever, must be borne in mind.

TREATMENT.—The treatment depends entirely on the cause found. Adenoids when present must be removed; in such cases the results of the operation are very satisfactory, and it is seldom that any further treatment is required. When, however, no adenoids are present, and there is no polypus or choanal obstruction, the advisability of dealing with a deflected septum or with a swollen turbinate must be considered. The writer believes that it is seldom necessary to operate on the deflected septum of a child, and that, although cases of great deflection are seen from time to time, in which it is clear that rectification should be attempted, yet that such are rare. With regard to enlarged turbinates, also, the greatest caution must be observed; free removal of a turbinate is never permissible, but if there is

persistent swelling, in spite of other measures, it is advisable to reduce the size of the swelling by means of the cautery. If the child is old enough, he can be taught to sniff an alkaline lotion up the nose after clearing it by blowing. In the case of younger children, an oily mixture containing menthol and eucalyptus should be instilled into the nose or applied by an atomiser. Improvement in the general health is also an aid to getting rid of the catarrh. Change of climate is often of great advantage. Damp and low-lying districts should be avoided; country air is much better than that in towns, and that of the seaside is best of all.

Rhinitis Sicca.—Rhinitis sicca is a form of chronic rhinitis which is characterised by dryness of the mucous membrane.

ÆTIOLOGY.—Although frequent in adults, it is comparatively uncommon in children. Anaemia is a predisposing cause; also the habitual inhalation of air laden with dust; in some cases no adequate cause can be discovered.

SYMPTOMS.—The nose is unusually dry and never requires blowing. From time to time small crusts of dried mucus form in the anterior part of the nose, especially on the septum, and cause a sense of irritation; this leads to picking the nose, and, by causing superficial excoriation of the mucous membrane, is a frequent source of epistaxis. Repeated removal of the crusts may lead to an ulcer, or even to a perforation. Although the nose is clear, complaint is often made of stiffness, which appears to be largely a subjective sensation. Nevertheless, at times, the swelling of the mucous membrane does occur, particularly on entering a hot stuffy room, and may cause real temporary obstruction. On examination, the nasal fossæ are found to be abnormally roomy; frequently the back wall of the nasopharynx can be seen through the nostrils. Fine crusts of dried mucus of a white or branny appearance are seen on the mucous membrane, and a few larger crusts of darker colour may also be visible. Evidence of recent hæmorrhage is frequently present on the septum.

TREATMENT.—Adenoids are not usually present, but if a large pad is found it should be removed. The chief indications are to place the child in a situation where it can breathe fresh air; to treat the anaemia; and to keep the nasal mucus moist. This is best attained by the daily application of an ointment containing small amounts of eucalyptus and oil of pine. The *unguentum eucalypti* diluted with twice its volume of soft paraffin answers the purpose very well.

Atrophic Rhinitis.—The most characteristic features of this form of rhinitis are the formation of large crusts of dried secretion, which emit an intensely disagreeable odour, and the atrophic condition of the nasal mucous membrane.

ÆTIOLOGY.—Atrophic rhinitis reaches its full development at the age of puberty and in the subsequent years, but the beginnings of it date back earlier, and may be seen in children at the age of four years and upwards. The worst features of the disease are never seen in a narrow nose such as most children possess, but when the nasal bones enlarge, as they do between the ages of ten and fifteen years, the ill-developed turbinates fail to fill the cavities adequately, and accumulation of secretion is permitted. Females suffer more often than males, a fact which may possibly be accounted for by the greater prevalence of anaemia amongst them. Several members of a family are often attacked, and not infrequently one or both parents will recollect having had the same symptoms. The incidence is much greater amongst those with the broad brachycephalic type of head than amongst

those with narrow or dolichocephalic heads. The starting-point of the disease, when it can be ascertained, is a severe attack of acute rhinitis, such as may follow scarlet fever or measles. In others there is a history of severe and long-continued colds. The present writer treated a small boy for membranous rhinitis; eighteen months later, at the age of five years, he had well-marked atrophic rhinitis. Lack attributed 5 per cent. of his cases to congenital syphilis. Other authors agree that syphilis is responsible for only a small proportion of the cases.

Symptoms.—The cardinal symptom is the offensive odor. The patient is quite unconscious of it, and it is left to others to call attention to it. The patient himself complains of hard lumps or crusts coming from the nose, of stuffiness, of loss of the sense of smell, of headaches and inability to blow the nose. In numerous cases the patient is not brought on account of his nose at all, but on account of the dryness and crusting in the pharynx or of the chronic laryngitis which always accompanies this disease, and it is only on investigation that the condition of the nose is discovered.

Examination.—The external nose is frequently small, the bridge broad and flat, and the nostrils rounded. The nasal fossae are always abnormally wide. The inferior turbinate is small, leaving a wide space between it and the septum; in a severe case it may be so small as to be hardly visible. The middle turbinate is also small as a rule, and inspection of the deeper parts, such as the naso-pharynx, the front of the sphenoid, and the Eustachian tube, becomes easy. Lying in the inferior meatus, a large crust is soon adhering to the mucous membrane; the free surface of the crust is dry, hard, and green; the attached surface is moist with a yellow mucopurulent secretion which emits a disgusting odor. Higher up in the nose crusts are seen, and constantly a mucopurulent secretion is visible between the middle turbinate and the septum. In adults, concomitant sinus suppuration is found in some 10 to 20 per cent. of cases; in children sinus suppuration is practically unknown, owing to the late development of these cavities. Crusts and dryness of the mucous membrane commonly extend into the naso-pharynx, and in many cases the larynx also is attacked. When all traces of the crusts and secretions have been removed, the tumor disappears, and is therefore not an essential part of the disease; it results from the changes which occur in the secretion when it is allowed to remain in the nose.

Pathological Changes.—Microscopic examination of the atrophic mucous membrane shows that squamous epithelium has replaced the normal columnar ciliated epithelium over considerable areas; there is round-cell infiltration of the tissue immediately beneath the epithelial layer; the glands are fewer in number and show degenerative changes, and the blood-spaces are empty. Although it has been stated that active absorption of bone occurs, recent observers do not confirm this. In short, the pathological condition is one of atrophy, consecutive to inflammation, unaccompanied by ulceration.

Bacteriology.—A bacteriological examination of the secretion will usually show the presence of a mixture of organisms:

1. A capsulated diplobacillus, known as the "*Bacillus mucosus* of Abel," closely resembling Friedländer's *Bacillus pneumoniae*.

2. A bacillus belonging to the diphtheroid group, in some cases indistinguishable from the Klebs-Löffler bacillus.

3. A number of the group of bacteria commonly found in cases of acute rhinitis, such as the pneumococcus, the influenza bacillus, the micrococcus catarrhalis, staphylococci, or streptococci.

It has not yet been proved that any one of these bacteria is the specific cause of atrophic rhinitis. The view taken here is that the damage to the nasal mucous membrane and the wide nasal fossa renders the nose unable to protect itself against infection.

DIAGNOSIS.—As a rule no difficulty arises; the severe cases must be distinguished from the syphilitic by the absence of ulceration and necrosis of bone. The milder cases in which there is little or no foetor bear a close resemblance to rhinitis sicca.

PROGNOSIS.—When once the condition is well established and the nasal fossa are wide, the prospect of a cure is remote; but the worst symptoms can be relieved, and in the course of years the nose will cause less and less trouble. In the milder and earlier cases the outlook is more favourable, and by careful treatment progress can be arrested. Impairment of the sense of smell is usually permanent.

TREATMENT.—The most important step is to free the nose from crusts and to prevent the secretion from accumulating. The large crusts are carefully pulled out; the nose is then syringed until it is quite free from secretion. This must be repeated night and morning, and is the essential point in the treatment. Merely sniffing the lotion is of very little use; it does not dislodge the crusts. When the nose is very wide, the insertion of a pledget of cotton-wool helps to loosen the crusts, and also prevents the secretion from drying; it thus forms a valuable aid during the first weeks of treatment. The wool can be moistened with paroline and worn at night. Some of the oil should be sprayed into the nose after each cleansing.

Powerful irritating antiseptics must be avoided, although the temporary inflammation set up by them may appear to do good. Still more unwise is it to do any operation which can make the nose any wider. Diet and drugs taken internally do not appear to affect the disease at all. The character of the air breathed by the patient is important. A hot, dust-laden atmosphere is the worst; the smoke of a big town is also objectionable. Pure air is certainly an advantage, and the writer has seen marked improvement, including a partial return of the sense of smell, follow three months' residence at the seaside.

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LUPUS.

INTRODUCTION.—Lesions in the nose due to tubercle bacilli are divided into two groups, in accordance with the clinical manifestations: (1) Lupus; (2) tuberculosis. There is no complete agreement as to the limits which separate these two groups, but the position taken here is that lupus is a primary manifestation of the disease without any evidence of pulmonary infection; whereas tuberculosis of the nose is invariably secondary to a lesion of the lungs. The latter is rarely seen save in adults, and will not be described.

Ætiology.—Lupus of the nose occurs chiefly amongst children and young adults. It may be primary in the nasal mucous membrane, or may spread inwards from the alæ. The nose is seldom the only point of infection, patches of lupus usually being found on some other part of the body. Infection is probably conveyed by the fingers.

SYMPTOMATOLOGY.—The symptoms are remarkably slight. There is no pain before ulceration occurs, and very little afterwards. Complaint may be made of crusting or of obstruction to breathing. In a case observed by the writer, epiphora was the first symptom. The anterior part of the septum is the area most frequently attacked, and from it the disease spreads to the floor and outer wall of the nose. The nasal duct is apt to become obstructed, and infection may travel up to the lacrimal sac. Sooner or later the external nose becomes red and swells, and when disintegration occurs the tip and alæ are destroyed, leading to unsightly deformity. As the disease advances, the cartilaginous septum is perforated, but the bony portion is spared, and massive necrosis does not occur. From the floor of the nose infection may spread through the hard palate and appear in the roof of the mouth, and in many cases the pharynx and larynx are affected as well. The appearance in the early stage is that of eczema; later, groups of closely-set firm red nodules are seen in patches, or coalesce to the formation of small tumours. Superficial ulceration of the nodules is frequent, and pale granulations may form which fill the nasal fossæ. The secretion from these is scanty, and dries into crusts; the tendency to bleed is slight. Progress is very slow; years may elapse before there is serious destruction of the tissue.



FIG. 21.—LEPUS OF THE NOSE OBSTRUCTIVE HAVE LACRIMAL DUCT.

DIAGNOSIS.—Diagnosis from syphilis is made by the presence of tubercles, by the absence of destruction of bone and the slow progress of the lesions. Nevertheless considerable difficulty may arise; Wassermann's reaction and the effect of treatment are then of great service.

PROGNOSIS.—The prognosis is favourable, provided the treatment is long-continued and a careful watch kept for early evidence of recrudescence. The scarring may render the external nose unsightly and cause troublesome obstruction of the interior.

TREATMENT.—(a) *Local.*—For the external nose the most satisfactory treatment is the application of the Flouren light or of X-rays. The interior of the nose is best treated by scraping away any large nodules or masses of granulations, and applying a powerful caustic to the smaller lesions. The galvanic-cantery is satisfactory for this purpose, and applications of silver nitrate are also effective. Plastermill gives large doses of sodium iodide, and causes the patient to inhale air containing ozone.

(b) *General*.—In addition to other measures for the general treatment of tuberculosis, it should be noted that favourable results have been obtained in these cases by tuberculin injections and the administration of small doses of arsenic.

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SYPHILIS OF THE NOSE.

Nasal syphilis in children is always due to congenital disease, and occurs in two forms:

- Syphilitic coryza*.
Gummatous lesions.

Syphilitic Coryza is one of the earliest manifestations of syphilis. The infants who suffer from it are attacked as a rule within a few days of birth, rarely after the third month.

SYMPTOMS.—The symptoms are similar to those of a rhinitis due to any other cause. The nose becomes more and more obstructed; the breathing is noisy and snuffy, and nasal respiration is so poor that feeding is immensely difficult. The discharge from the nose is clear at first, becoming more purulent in the later stages. The upper lip is reddened, and fissures may appear; crusts are apt to form at the anterior nares and increase the obstruction. It is impossible to view the interior of the nose for any distance, so that the condition of the mucous membrane is largely a matter of surmise. It is certainly swollen, and it is possible that some periorrhinitis occurs as well. Ulceration and necrosis are very rare.

DIAGNOSIS.—The long duration of the snuffles must always arouse suspicion of syphilis, but the diagnosis really depends on the appearance of a skin rash, mucous tubercles, or some other evidence of syphilis.

PROGNOSIS.—The mortality amongst these syphilitic infants is high, but death is seldom attributable to the rhinitis, although the difficulty in feeding which results may be a contributory cause. When recovery occurs, a deformity of the nose may become apparent as the child grows up. The bridge fails to develop properly, and remains depressed, so that a "saddle-back" nose is the result. The damage to the mucous membrane may also be permanent, and give rise to atrophic rhinitis in later years.

TREATMENT.—The general treatment is much more important than the local. The aim of the latter should be to clear the nasal passages for breathing. With this in view, LAURENS advises that the secretion should be aspirated from the nostrils by a rubber syringe having a suitable nozzle. This is followed by the instillation of a few drops of hydrogen peroxide (5 volumes); and when the resultant foeting has cleared the nasal cavities, a few drops of sterilized liquid vaseline are allowed to run into each nostril. The nose may also be cleansed by gently driving saline solution from a glass syringe into the nasal cavity, the child being held on its side to prevent choking.

Gummatous Lesions.—The gummatous lesions of inherited syphilis are similar in character to the tertiary manifestations of acquired syphilis. Such manifestations are exceptional before the age of four, and more frequent between the ages of eight and twelve.

SYMPTOMS.—The symptoms depend on the area of the nose which is attacked. A gummatous infiltration of the alae and external nose causes a dull red swelling of the tissues, which may bear a considerable resemblance to lupus. Ulceration supervenes more rapidly, however, and in an untreated case the external nose may be destroyed, leaving the nasal cavities widely open. Such an event must be extremely rare. Internally gummatous deposits cause nodular swellings on the septum and inferior turbinates, giving rise to nasal obstruction. Extensive superficial ulceration of the mucous membrane is also found, and is evidenced by a purulent discharge. In more severe examples the ulceration extends deeply into the tissues; necrosis of bone and cartilage occurs, pieces of the vomer and of the inferior turbinate come away, and a perforation of the hard palate may result. It is astonishing to note how far the destructive process may extend before advice is sought, and it is evident that the symptoms may be trivial until great damage has been done. There is remarkably little pain, with the exception of headache. The presence of a bony sequestrum is indicated by intense tenderness, whilst perforation of the palate gives rise to a characteristic alteration of the voice and a discharge of liquid through the nose when drinking. In the pharynx gummata occur on the posterior surface of the soft palate and on the laryngeal walls; these deliquesce and form ulcers; pieces of the soft palate are cut away, and the dense cicatricial tissue which results binds the remains of the soft palate to the walls of the pharynx. In this way the communication between the nose and the oesopharynx may be completely obliterated.

DIAGNOSIS.—When the external nose is affected, syphilis must be distinguished from lupus by the more rapid destruction of tissue and the absence of tubercles. When the disease is inside the nose, an extensive ulceration is unlikely to be due to any other cause than syphilis. The presence of large sequestra may be regarded as pathognomonic of syphilis, so that a careful search for various hints must always be made.

PROGNOSIS.—Impairment or loss of the sense of smell is the rule after extensive nasal syphilis. The destruction of bone is irreparable, and the nose remains wide and dry, with a great tendency for crusts to accumulate in the empty cavity.

TREATMENT.—The routine general treatment for syphilis should be adopted. Locally sequestra must be removed as soon as they become loose. The cavity should be cleaned regularly, and a paint containing 10 grains of nitrate of mercury saturated, with half an ounce of olive-oil to an ounce of liquid paraffin, should be applied to the mucous membrane.

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- GERARD: *Syphilis of the Nose, Throat, and Ear*. Boston: S. Karger, 1910.

ABSCESS OF THE SEPTUM.

INTRODUCTION.—A septal abscess is a collection of pus lying beneath the mucoperichondrium, and separating it from the cartilage.

ETIOLOGY.—It is met with more frequently in children than in adults, but the latter are not exempt. With few exceptions, the condition is bilateral. The only common cause is a blow on the nose; as a result of the injury a hematoma is formed, which becomes infected. In rare cases a septal abscess has been noted in the course of infectious fevers, and infection may also spread from a various tooth or carious being in the neighbourhood.

SYMPTOMATOLOGY.—There is usually a history of an injury to the nose, followed by nasal obstruction, which gradually becomes complete. The external nose is swollen, tender, and sometimes reddened. A rounded fluid swelling appears on each side of the septum just within the nasal orifice. The two swellings fill the nasal fossae and have a symmetrical appearance. The mucous membrane is acutely inflamed.

PATHOLOGY.—A mixture of pus and blood-clot is found lying between the cartilage and mucoperichondrium. The cartilage is nearly always perforated either by the initial injury or as a result of necrosis. The opening thus made allows of communication between the two sides. In a few cases the pus is confined to one side of the septum.

DIAGNOSIS.—The symmetrical swellings are unlikely to be mistaken for any other condition; should there be any doubt as to the nature of the fluid, a small amount can be withdrawn by means of an aspirating syringe.

PROGNOSIS.—A permanent perforation of the septum may remain after the abscess has been drained, and in some cases the external nose has fallen in. This may be a result of the original injury, which has hitherto been obscured by the swelling, or may result from necrosis of the cartilage and subsequent contraction of scar tissue.

TREATMENT.—A free incision must be made through the mucous membrane to release the pus, and the lips of the wound must be kept apart by means of a dressing until drainage is complete; otherwise they soon adhere and the pus accumulates once more.

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G. TRANTMAN: *Archiv für Laryngologie*, 1908, *Koel.* 240.

EPISTAXIS.

Bleeding from the nose of a child is such a common event, and as a rule of so little importance, that medical advice is seldom sought on account of it. Nevertheless the bleeding may signify the presence of some serious local or general disease, and by its severity and frequent recurrence may affect the general health.

ETIOLOGY.—Bleeding may be due to some purely local cause situated within the nose, or may result from some general condition of the blood or bloodvessels.

Local Causes.—1. Of the local causes, the most frequent is the presence of a group of dilated vessels in the mucous membrane covering the cartilage of the anterior part of the septum, a region known as Kiesselbach's area. Any cause of local congestion—e.g., blowing of the nose or severe cough—may excite hemorrhage, which by its severity or frequent repetition may be very troublesome.

2. Rhinitis sicca is another common cause of epistaxis. This may occur at any stage: during the separation of the small adherent crusts, or from an ulcer or the margins of a perforation. The amount is usually small, and always comes from the anterior portion of the septum.

3. Acute rhinitis, especially in infants and small children, is often accompanied by a small amount of bleeding.

4. Ulceration. Any ulcer inside the nose, whether due to lupus, syphilis, growth, or foreign body, may give rise to bleeding; but in such cases there is a blood-stained discharge rather than a free hemorrhage, and the amount is small.

5. Fibroma of the naso-pharynx frequently gives rise to severe hemorrhage from the nose, which is difficult to control and may prove fatal.

6. Bleeding polypus of the septum. This is an uncommon tumour based on the anterior part of the septum, and more rarely on one of the turbinates. The growth has the microscopic appearance of an angioma, and bleeding is usually its most conspicuous symptom.

7. Injury readily excites bleeding from the delicate vascular mucous membrane of the nose.

General Causes.—1. Diseases of the blood. In leucocythæmia and all the severe anæmias epistaxis is frequent, and is apt to be very persistent.

2. Diseases of the heart and bloodvessels. On three occasions the writer has seen children suffering from valvular diseases of the heart admitted to hospital solely on account of bleeding from the nose. The heart should always be examined when there is troublesome nose-bleeding without sufficient local cause. Apart from cardiac lesions, anything which obstructs the venous flow from the head is likely to cause epistaxis. In adults degenerate bloodvessels give rise to very intractable epistaxis, but that is a rare event in children.

3. Infectious fevers, such as scarlet fever, measles, influenza, are frequently ushered in by epistaxis, seldom severe.

4. Hemophilia, scurvy, and purpura, are among other causes.

Source of the blood.—In all the foregoing cases, with the exception of those in which there is an ulcer or a tumour, it should be recognized that the blood almost invariably comes from the anterior part of the septum; any other source is very uncommon. Brown Kelly has described cases in which the bleeding came from the ethmoidal veins.

Symptoms.—The blood runs out from the front of the nose, issuing from one nostril. If the patient tilts up the head or lies down, the blood passes backwards into the naso-pharynx and is swallowed. When the bleeding from one side of the nose is very free, some of the blood may come out of the other nostril, after passing round the back of the septum, so that a single bleeding-point causes apparent bleeding from both nostrils. When dependent on cardiac disease or vascular degeneration, the bleeding is apt to be profuse and has a tendency to recur, and the same applies in those cases in which there is alteration in the character of the blood.

TREATMENT.—Treatment is often unnecessary, but, if required, simple measures should be tried. The natural tendency of the patient is to stoop and to blow the nose. Both of these acts increase the congestion of the nose, and therefore encourage the bleeding. It is better that the patient should sit upright, the head being kept cool by means of flannels soaked in ice-cold water and applied to the face and neck. The feet should be immersed in a hot bath, and the arms raised above the head. These measures will suffice in many cases, but if not, the next step is to apply a haemostatic to the nasal mucous membrane. For this purpose a mixture of equal parts of alumina chloride, 1 in 1,000, and 10 per cent. cocaine hydrochloride, is the best. A small plug of cotton-wool soaked in the mixture is firmly pressed against the bleeding area. The pressure may be relaxed in two minutes, and it will then be found that the bleeding has ceased. Oozing may continue from the blood already accumulated in the nose, and create the false impression that the hemorrhage continues. At the end of half an hour the wool can be removed and replaced by another plug covered with argentine hamamelids. It is desirable that the nasal mucous membrane should be kept moist with this preparation for several days subsequently in order to avoid the formation of crusta. Should the bleeding tend to recur, the mucous membrane must be anesthetized and the bleeding vessel touched with a caustic at a dull red heat. In those rare cases in which the bleeding does not come from the anterior part of the septum, the bleeding-point must be sought for and pressure applied to it by means of plugs of wool or ribbon gauze moistened with the mixture mentioned. Plugging the posterior nares has been recommended and frequently practised on tuberculate patients. Before adopting this method of treatment, it should be borne in mind that it does not apply pressure to the bleeding vessel; that it is horribly uncomfortable to the patient; that there is a real risk of causing otitis media; and that, as far as the writer's experience goes, it is never necessary.

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FOREIGN BODIES IN THE NOSE.

INTRODUCTION.—Children frequently insert small objects into the nose when at play, although it is difficult to understand their fondness for doing so. Pieces of paper, pieces of string, beads, shells, bits of wool from blankets, and other small objects, may be found, but the most frequent of all is a beet button.

SYMPTOMS.—It is possible for a foreign body to remain lodged in the nose for a long period without exciting noticeable symptoms. When symptoms do arise, the most constant phenomenon is a discharge from one nostril. At first it consists of clear watery mucus; later it becomes purulent, and tinged with blood from time to time. The upper lip is sore and reddened. The discharge may become offensive. In addition, the nose is obstructed more or less completely on the affected side. With such symptoms the diagnosis of a foreign body should be readily made without any reliance on the history or the absence of one.

PATHOLOGY.—The foreign body is usually lodged in the anterior part of the nose, and may be found lying between the inferior turbinate and the septum, or at a somewhat higher level opposite the upper border of the inferior turbinate. It

process causes the mucous membrane to be red and swollen in all cases, whilst in many the body is imbedded in a mass of granulations which bleed readily, and from which the purulent and offensive discharge proceeds. A foreign body which has lain in the nose for several years becomes encrusted with a layer of earthy salts, chiefly calcium phosphate and calcium carbonate, and it is in this way that a rhinolith is formed. The persistent irritating discharge from the nostril may set up an eczematous condition, which may result in a considerable narrowing of the lumen.

DIAGNOSIS.—The diagnosis must be made from unilateral membranous rhinitis; the symptoms in the two conditions are very similar. In both there is a unilateral discharge of mucus or pus, accompanied by occasional bleeding and unilateral nasal obstruction. But the discharge is rarely offensive in membranous rhinitis, and frequently so when a foreign body is present. In order to differentiate between the two conditions, therefore, it is necessary to make a careful examination of the nose after the application of adrenalin and cocaine. If membranous rhinitis be present, a thick white membrane will be seen covering the mucous membrane, and a portion of it should be removed for bacteriological examination. On the other hand, after the secretion has been wiped away, the foreign body may come into view, and then its character and consistency can be ascertained by gentle probing.

TREATMENT.—Having made certain that a foreign body is present, the next step is to remove it. The method of doing this depends on the character of the object and its consistency. A soft body, such as a piece of string, can be seized in forceps and withdrawn; but this method is not suitable for a hard round object, such as a boot button or a bead. It is very difficult to grasp either of these objects firmly with the ordinary nasal forceps, and an attempt commonly results in the button being pushed farther in, bleeding is set up, and the child is frightened, so that extraction may become impossible without a general anæsthetic. The best plan is to introduce a little more cocaine, and then pass a blunt hook up through the middle meatus of the nose until it is well behind the button; the hook is then drawn downwards and forwards, and brings out the foreign body. If this method be adopted, it will be found that a general anæsthetic is rarely necessary. Occasionally the patient may be seen soon after the introduction of the foreign body, and before it has become fixed. A little snuff should then be introduced into the nostril, and in the violent sneezing which results it is probable that the foreign body will be expelled.

ADENOIDS.

SYNONYMS.—Adenoid vegetations; Pharyngeal tonsils.

INTRODUCTION.—The term "adenoids" is applied to hypertrophy of the lymphoid tissue on the roof and back wall of the nasopharynx. The first comprehensive account of the condition was given by Wilhelm Meyer of Copenhagen, under the title of "Adenoid Vegetations," in 1868.

ETIOLOGY.—*Age.*—Adenoids may appear during the first year of life; pads of a size sufficient to cause nasal obstruction have been removed from infants six months old. At the age of three years adenoids are exceedingly common, and remain so during the succeeding years of childhood. At the age of ten there is an

appreciable diminution in the number of cases, whilst from the age of fifteen onwards the condition becomes relatively uncommon.

In 400 cases operated upon in the Throat Department of St. Bartholomew's Hospital, the ages were as follows :

Age of Patient.	Number of Cases.
2	10
3	18
4	23
5	37
6	36
7	36
8	24
9	32
10	24
11	31
12	30
13	40
14	19
15	15
16	10
17	4
18	5
19	1
Over 19	5

The incidence amongst the general population of children is extraordinarily high. Chestle examined 1,000 children of poor parentage, and found adenoids in 134. Yearley examined 1,246 London school-children, and found that 37.7 per cent. had adenoids. These numbers include children in whom no symptoms had been observed by the parents or teacher. In East Suffolk 7,790 school-children were examined by Stewart, who found adenoids or enlarged tonsils in 1,205, or, roughly, 16 per cent. This lower figure is accounted for by the fact that he included only those patients in whom the tonsils and adenoids were causing symptoms. Sex has no influence.

Hereditv.—It is by no means rare to find that every member of a family suffers from adenoids, and in such a case one or other of the parents is almost certain to have suffered in a similar way in earlier life. The Hebrew race is alleged to be peculiarly susceptible.

Climate.—The experience of those who practice in warm, dry climates is that adenoids are much less frequent than in those countries where the weather is cold and damp.

Exciting Cause.—Although in many cases the onset of symptoms is so gradual that no date can be fixed, yet in others the symptoms suddenly become prominent after an acute rhinopharyngitis or a specific fever, such as measles. At present the trend of opinion is in favour of the hypothesis that the enlargement of the lymphoid follicles of the naso-pharynx is a response to infection. Inherited syphilis has been held responsible, but, although adenoids are often present in that disease, there is no satisfactory evidence of a causal connection. The question of a tuberculous infection is discussed under the heading of Pathology.

SYMPTOMATOLOGY.—Adenoids may be present and give rise to no symptoms. The most conspicuous symptom is obstruction to nasal respiration, the degree of

obstruction depending on the completeness with which the mass fills the nasopharynx. During the daytime the patient breathes heavily, especially when eating, the voice is thick, and the mouth is kept open. At night there is snoring, always more marked when the patient has a cold. The hearing is dull, and attacks of earache are frequent; colds are common, and tend to persist a long time. The facial appearance may be very striking. In severe cases the expression is vacant, the lower jaw is allowed to droop, and the upper lip is retracted, allowing the incisor teeth to appear. The nose is pinched, the nostrils narrow, and the alae fall in during inspiration. It should be noted that with this type of face the arch of the palate is high and the alveolar arch flattened, whilst the teeth are crowded and displaced. Some believe that this type of jaw is inherited, and that the presence of adenoids is a coincidence; others maintain that the shape of the jaw is the result of the prolonged mouth-breathing, and is therefore due to adenoids. It is certainly very difficult to establish free nasal respiration in such cases by merely removing the adenoids. The voice lacks resonance, and all those sounds for which a free nasal passage is essential are altered. Thus "king" becomes "kig," "sine" becomes "sile," and so on.

Ear.—Adenoids are by far the most common cause of affections of the middle ear during childhood, and the aural complications arising from them are of great frequency and importance. The combination of hardness of hearing and nasal obstruction produces in some children a lack of attention and heaviness of demeanour which is mistaken for stupidity (aprosopia).

Larynx.—The larynx is more subject to inflammatory attacks when adenoids are present; recurrent attacks of bronchitis are also frequent.

During sleep the natural tendency of the child is to return to nose breathing; the obstruction to respiration then becomes more prominent, and if the tonsils are enlarged the child may have serious difficulty in obtaining sufficient air for the maintenance of good health. The poor supply of oxygen impairs the general nutrition and gives rise to stunted growth and anaemia.

Suffocative attacks also occur, during which the breathing becomes more and more difficult, until the child wakes with a start, gets fresh air by opening its mouth, and then does off again. The strain of respiration influences the development of the chest wall, and results in falling in of part of it, so that a pigeon-breast or other deformity may arise.

The following conditions have a doubtful relationship with adenoids:

Artitis.—In patients suffering from true spasmodic asthma, removal of adenoids is seldom of any benefit to that affection; when the asthmatic attacks are associated with chronic bronchitis, removal often causes a marked diminution in the frequency of the seizures.



FIG. 21.—ADENOIDS: FACIAL APPEARANCE.

Epilepsy.—Sondniak, in a report on 1,000 operations for adenoids, mentions the cure of 7 cases and the partial relief of 18 others; this is in accordance with general experience.

Nocturnal *Enuresis* is not cured by removal of adenoids, though it is always advisable to get rid of this possible cause of irritation.

PATHOLOGY.—Lymphoid follicles are found in the naso-pharyngeal mucous membrane of every child. It is only when they become hypertrophied to a degree that is considered pathological that the term "adenoids" is employed. The mass which is formed lies on the back wall and roof of the naso-pharynx, grooved by deep longitudinal furrows which separate it into a number of ridges, usually five. The covering is of columnar ciliated epithelium, dipping down into the folds. The bulk of the swelling consists of lymphoid tissue, arranged in regular follicles, supported by a delicate fibrous-tissue framework. In older patients the growth becomes fibrous. The summits of two ridges may unite and enclose a space, which becomes a cyst or abscess. Search for evidence of tuberculosis has been made with varying results. Piffi found histological evidence of tubercle in 3 per cent.; Lantigen and Nicoll found it in 12 of the 55 cases they examined. On the other hand, Macfadyen and MacConkey, in a series of inoculation experiments with guinea-pigs, could obtain no evidence of tubercle in 44 specimens. Nobilcount and Timm's experiments led them to a similar conclusion. Adenoids may undoubtedly be tuberculous; the writer has seen three examples. There is, however, as is obvious from the foregoing statistics, no reason for believing that any large proportion of these vegetations are due to tuberculous infection.

DIAGNOSIS.—Diagnosis is rarely difficult. In many cases it is possible to see the postnasal space by means of a mirror. If the symptoms are present in a child under eight years of age, and the tonsils are much enlarged, adenoids may be inferred with a great degree of certainty; at a later age enlarged tonsils are often present without adenoids. In all doubtful cases a digital examination should be made. Congenital atresia of the choanae dates from birth, and may cause obstruction of one or both nostrils; it can be diagnosed by inserting the tip of the finger into the choana and feeling the occluding membrane. Chronic rhinitis causes very similar symptoms, but can be distinguished by a careful inspection of the nasal fossae from the front. Deviation of the septum is often thought to be a sufficient explanation of nasal obstruction. In the writer's experience severe symptoms always indicate the presence of adenoids as well, the removal of which will probably give adequate relief. Paralysis of the soft palate occasionally leads to a mistaken diagnosis of adenoids, owing to the loud snoring. In its early stages, a fibroma of the naso-pharynx causes symptoms exactly similar to those of adenoids, but the steady progress of the obstruction and the associated attacks of epistaxis should be a sufficient indication for a digital examination, when the smooth round hard tumour is easily differentiated from the soft pad of adenoids.

PROGNOSIS.—The prognosis is excellent. Colds are less severe; nasal respiration is re-established; hearing recovers; and the general nutrition is improved. Nevertheless, when the palate has a very high arch, mouth-breathing is apt to persist; deformities of the chest may also remain, in spite of exercises, although they tend to become less conspicuous.

RECURRENT.—If the patient be three years of age or less, there is a definite risk of recurrence, even after an efficient operation has been performed. From

the age of six upwards recurrence becomes very uncommon, and is then usually ascribed to incomplete removal, though unjustly perhaps. When three or more operations are required, it is almost certain that an incorrect diagnosis or an inefficient operator is to blame.

TREATMENT.—All large masses should be removed, and even small pads, if there is reason to believe that they are infecting the middle ear or giving rise to epileptic attacks. Small vegetations accidentally discovered may be left alone.

Breathing exercises have not proved a satisfactory substitute for operation, and it is clear that when the obstruction is considerable exercises with the mouth shut tend to exaggerate the deformity of the chest walls. The operation is best performed under a general anæsthetic. The use of chloroform is attended with more risk than that of the A.E.E. mixture. The cough reflex should not be abolished. The growths are removed in one piece by means of a curette, or in many pieces by Lovenburg forceps. After the removal the postnasal space is to be carefully examined for any small fragments that remain, and the finger is inserted into each nostril to ascertain that the passage is clear. The patient must remain in bed for two days, and indoors for the remainder of the week; no other treatment is necessary, and local applications are dangerous. When convalescence is established, a course of exercises designed to improve the chest movements is of great service.

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RETROPHARYNGEAL ABSCESS.

INTRODUCTION.—Retropharyngeal abscess is the term applied to collections of pus which lead to a bulging forward of the wall of the pharynx. When considering this subject, it is of advantage to divide the cases into two distinct groups:

1. Collections of pus in the retropharyngeal space proper.
2. Collections of pus which lie between the vertebral column and the prevertebral fascia.

The former group constitutes the ordinary retropharyngeal abscess of infants. The second group includes those abscesses which are secondary to tuberculous lesions of the spine. The distinction is important because the treatment is different in the two cases.

1. Retropharyngeal Abscess.—In these cases the pus lies in the space between the posterior or lateral wall of the pharynx and the prevertebral fascia.

ÆTIOLOGY.—It is essentially a disease of infancy and early childhood. About half the cases occur in children under the age of one year. The greatest incidence occurs at the age of six to twelve months, and there is diminished frequency in each succeeding year until the age of four, after which it is very uncommon. The

explanation offered for this age incidence is that the lymph glands in the retro-pharyngeal space disappear between the fourth and fifth years. The primary infection is of the adenoïd tissue of the wall of the pharynx, and spreads to the retropharyngeal tissue, leading to abscess formation. It may be noted that in infants retropharyngeal abscess appears to take the place of peritonsillar abscess in older patients. We therefore find an acute pharyngitis preceding the abscess as its most common cause. The pharyngitis may be primary or may follow measles or some other infection. Other causes are much less frequent. Pus from suppurating deep cervical glands may make its way into the retropharyngeal space, but more commonly gives rise to a swelling which pushes in the side-wall of the pharynx. Pallas collected twenty-six cases in which pus from the mastoid made its way by

various routes to the retropharyngeal tissue. Lastly, in rare cases a wound of the pharynx may be the cause.

SYMPTOMATOLOGY.—The two prominent symptoms are dyspnoea and dysphagia. A roaring noise is noticed first, which gradually becomes more and more intense until it develops into severe dyspnoea and suffocation is threatened. The breathing is at its worst when the child is lying on its back, and may be relieved to some extent by turning it to the prone position. The cry often has a peculiar muffled character. The difficulty in swallowing appears later than the dyspnoea, and gradually increases until a stage is reached in which it is impossible to feed the child. Severe dysphagia indicates a large abscess situated low down. On inquiry it is found that the urgent symptoms have been preceded for two or three weeks by a less noticeable illness in which there was evidence of a sore throat. A collection of tender swollen glands is found in



FIG. 22.—RETROPHARYNGEAL ABSCESS, PUSHING FORWARD ON THE ROOF OF THE TONGUE AND THE LARYNX.

From a boy aged two and a half years, admitted to hospital for urgent dyspnoea of two days' duration. (From the *Memories of St. Bartholomew's Hospital*.)

the neck on the affected side, and on examination of the interior a rounded swelling is seen bulging forward the back-wall of the pharynx. It is situated to one side or other of the middle line, and is never central. It is dark red in colour, and fluctuates on pressure. Should the frothy mucus obscure the view, a careful digital examination must be made. A pale yellow area on the swelling may indicate the spot at which the abscess is about to rupture. Farrillians points out that there is no rigidity of the neck when the abscess is purely retro-pharyngeal, and that this symptom, which is usually present, depends upon inflammation of the cervical glands affecting the muscles.

POST-ADENOÏD ABSCESS.—The same author also calls attention to a special variety of abscess in this neighbourhood which he calls "post-adenoïd abscess." The special feature of this uncommon form of abscess is that it is situated between

the layer of adenoids and the pharyngeal aponeurosis, and therefore higher up than an ordinary retropharyngeal abscess. It is in the middle line, and may extend to the roof of the naso-pharynx. Its presence gives rise to severe nasal obstruction, but does not cause dysphagia. A diagnosis is made by the finger encountering a firm fluctuating swelling in the situation of ordinary adenoids.

DIAGNOSIS.—Retropharyngeal abscess has been mistaken for laryngeal diphtheria on many occasions, and more than one house-surgeon has fallen into this error. The combination of dyspnoea and dysphagia in an infant should always lead to a careful examination of the back of the pharynx, both by inspection and palpation, and the diagnosis is then quite easy.

PROGNOSIS.—Evacuation of the abscess, whether spontaneous or by surgical means, is followed by rapid recovery. If it be allowed to remain untreated, there is grave danger of death from suffocation or exhaustion before the abscess bursts, and even after that event such rare accidents as hæmorrhage from the deep vessels of the neck or inhalation of the pus into the lungs have been reported.

TREATMENT.—The abscess should be opened at the earliest possible moment. A pair of sharp-pointed sinus forceps should be thrust into the abscess and the points widely separated. Care must be taken to hold the child in such an attitude that the pus runs into the mouth and not into the larynx; at the same time the opening must be carried down to the lowest part of the abscess so as to avoid the necessity of a second operation. Should death from asphyxia appear imminent, it may seem necessary to perform tracheotomy; but such an operation should be avoided if possible, and can usually be averted by hooking the back of the tongue forwards by means of the finger, and so allowing air to enter the larynx. When an abscess is due to suppuration in the cervical gland, around the carotid sheath, and also forms an external swelling in the neck, it is better to drain it by an external incision in front of, or behind, the sterno-mastoid, as seems best in the individual case.

2. Abscesses Secondary to Spinal Caries.—In this place it is only intended to call attention to the main features which distinguish this group from the preceding. As already stated, these abscesses are situated between the vertebrae and the pre-vertebral fascia.

ÆTIOLOGY.—The disease is by no means confined to infants, nor even to children, although they are the most numerous sufferers. The primary lesion is caries of the upper cervical vertebrae. The tuberculous pus which results therefrom collects behind the prevertebral fascia, and gradually forms a prominent swelling, which slowly moves in a downward direction.

SYMPTOMS AND DIAGNOSIS.—The symptoms are the same as those of the other variety—namely, increasing dyspnoea and dysphagia. Their onset is, however, much more gradual. The patient is not feverish, and there is no history of an acute infection. The existence of spinal caries may be known already, and if it be not, the stiff carriage of the head, the rigidity of the neck muscles, the pain elicited by pressure on the back, should call attention to it; the abscess itself is distinctly less tender on pressure.

TREATMENT.—These abscesses must always be opened through the neck. The incision is usually made behind the sterno-mastoid, and the pus is reached by

burrowing behind the caudal sheath. In this way it is possible to keep the wound absolutely aseptic, whereas if the opening is made through the pharyngeal wall it is impossible to attain that condition. The cervical cysts must of course receive treatment.

REFERENCES.

- A. FAYLLE: *La Presse Otolaryngologique Belge*, 1909, viii, 68.
D. C. L. PETERHANS: *Fruchtbaum*, 1902, ixix, 811.

TUMOURS OF THE NOSE.

Innocent Tumours.—**INTRODUCTION**.—Intranasal tumours are uncommon at any age, and particularly rare in children. Of innocent tumours the following varieties are met with:

- Papilloma.
- Glioma.
- Chondroma.
- Fibroma.

PAPILLOMA.—Small papillomata are seen occasionally inside the nose. They are situated on the anterior part of the septum, or more rarely on the anterior end of the inferior turbinate. Small specimens cause no symptoms, and are found during routine examination; the larger grow to the size of a small cherry, and give rise to a certain amount of nasal obstruction. The growth consists of a delicate connective tissue, covered with several layers of squamous epithelial cells. In rare cases large growths have been observed, causing nasal obstruction. These are situated deeper in the nose, and may give rise to free haemorrhage. The epithelium which constitutes their covering is transitional rather than squamous.

REFERENCE.

- A. BECKENHOF: *Archiv für Laryngologie*, 1914, xxii, 296.

GLIOMA.—Glioma of the nose is an exceedingly rare tumour. Two examples of it have been recorded by Payson Clark. In each case the tumour was present at birth as a pinkish-grey polypoid mass, which caused obstruction of the left nostril. In one case the swelling also caused deformity of the external nose. The tumours were composed of neuroglia; they were covered by mucous membrane, but were not encapsulated. They did not exhibit any tendency to recur after removal. In neither case was the exact point of attachment discovered, but there was no evidence of any direct connection with the interior of the cranial cavity. It appears probable that they arise as a result of some error of development.

REFERENCE.

- J. PAYSON CLARK: *American Journal of Medical Science*, 1905, cxviii, 505.

CHONDROMA.—Chondroma is another rare tumour of which few examples have been recorded. The patients are young, nearly all being under the age of twenty-

free. The tumours usually arise from the ethmoid, but may be attached to the septum or the lower part of the outer wall. They grow slowly, forming hard rounded swellings which cause nasal obstruction, and in some cases neuralgic pains. When the growths attain a greater size, they invade the orbits and displace the eyes; they expand the nose, producing the terrible deformity of the face known as "hog-face"; they encroach on the cranial cavity, and finally cause death by pressure on the brain or from exhaustion. In some cases the tumours grow with considerable rapidity, and it appears impossible, with our present knowledge, to draw a sharp distinction between chondro-sarcoma and chondroma. An extensive operation in the early stage of the growth offers the only chance of relief.

REFERENCE.

UPPESCHKE: *Archiv für Laryngologie*, 1908, xv, 253.



FIG. 22.—GROWTH OF NOSE, PARTLY COVERED BY MUCOUS MEMBRANE.

FIBROMA OF THE NASO-PHARYNX—INTRODUCTION.—Although less rare than those just mentioned, these tumours are by no means common. They exhibit a well-marked tendency to recur after removal, and on that account have been called "recurrent fibromata." When the difficulty of removal is borne in mind, it appears probable that each recurrence is evidence of an incomplete operation rather than of malignancy, for secondary growths in the lymph glands and general dissemination are unknown. Moreover, it has been asserted that spontaneous retrogression occurs after the age of twenty-five.

ÆTIOLOGY.—The age and sex distribution are remarkable. The disease is confined almost exclusively to males, and they become affected between the ages of ten and twenty.

SYMPTOMATOLOGY.—The chief symptoms are nasal obstruction and epistaxis. The nasal obstruction is of gradual onset, and at first is unilateral. As the tumour grows, the other nostril also becomes obstructed, but seldom as completely as the first affected. As a result of the nasal obstruction, the patient's voice is altered in quality, deafness and other middle-ear complications are common, and in some cases there is a remarkable sleepiness. The epistaxis is variable; in some cases there is only occasional trivial hæmorrhage, but in others profuse bleeding occurs, and reduces the patient to a state of grave anæmia. When allowed to grow unchecked, the tumour presses on the bony walls of the nose, expands, and finally perforates them. The growth finds its way into the maxillary antrum and other sinuses, and through the sphenomaxillary foramen into the cheek. It also invades the orbits, displacing the eyeballs, and by growing downwards may appear in the pharynx or perforate the hard palate.

PATHOLOGY.—The tumour may be a single rounded mass or lobulated. It grows from the pericostium by a broad base. The exact point of origin is difficult to ascertain. In some cases it is attached to the outer wall and roof of the clowm, just anterior to the naso-pharynx; in others it is alleged to grow from the roof of the naso-pharynx, although this was not true of any specimen examined by the writer. Microscopically it is found to consist of dense fibrous tissue. There is a covering of mucous membrane, and usually some large vascular spaces which are the source of the hæmorrhage.

DIAGNOSIS.—The condition is distinguished from adenoids by the fact that only one nostril is blocked up at first, by the steadily progressive character of the symptoms, and by the presence of an extremely hard rounded tumour in the naso-pharynx, which is almost immovable. Nasal polypi are never so hard, nor do they bleed, and as compared with a fibroma they are freely movable. The tumours grow much more rapidly, and are softer in texture.

PROGNOSIS.—The condition is serious. If allowed to remain, the tumour threatens life, whilst removal is difficult and not free from danger. As already mentioned, there is a belief that these tumours cease to grow after the age of twenty-five.

TREATMENT.—The removal of these growths is a matter of considerable difficulty, for the tumour is attached firmly in a somewhat inaccessible situation, and hæmorrhage may be alarming. It is possible to remove a small growth by means of a strong wire snare. In the case of a larger growth the tumour can be gripped by forceps in the postnasal space, whilst its base is separated from the bone by means of a periosteal elevator inserted through the nostril. When the growth is too large to be attacked in this way, access should be obtained by an external incision and resection of a sufficient amount of the outer bony wall of the nose. In the larger operation it is better to perform a preliminary tracheotomy and plug the laryngo-pharynx. The method of Nélaton, in which the soft palate is divided and part of the hard palate cut away, is not advisable; it does not give good exposure of the nasal part of the tumour, and may permanently injure the voice.

REFERENCE.

BRADY: *Journal of Laryngology*, 1905, xxi, 515.

Malignant Tumours—SARCOMA.—INTRODUCTION.—Malignant tumours growing in the noses of children are almost invariably sarcomata. This is in contrast to the condition found in adults, in whom there is a striking preponderance of carcinomata over sarcomata.

ÆTIOLOGY.—No causative factors are known. The belief at one time current, that nasal polypi and sinus suppuration were predisposing causes, has now been abandoned.

SYMPTOMATOLOGY.—The growth gives rise to obstruction of one nostril, which becomes complete in a few months. At the same time clear mucus runs from the nostril. Extension of the growth in an outward direction leads to obstruction of the nasal duct and filling of the antrum. Invasion of the orbit gives rise to proptosis and elevation of the eyeball. When pursuing an unchecked course, ulceration of the nasal portion of the growth supervenes, and the discharge becomes watery, sanguine, and foetid. Still later the tissues of the face are infiltrated, and the growth makes its way into the cranial cavity. The appearance presented in the earlier stages is that of a pink, smooth, rounded swelling filling the nasal fossa. Later gelatinous masses resembling polypi, and pale necrotic areas, are visible.

PATHOLOGY.—The tumours are frequently round-celled or small spindle-celled sarcomata. Melanotic sarcoma has been described. Their point of origin is the outer wall of the nose, in the neighbourhood of the middle turbinate. Exceptionally they grow from the septum or some other part of the nose.

DIAGNOSIS.—Diagnosis is made from innocent growths by the rapid progress of the symptoms, by evidence of early invasion of the antrum and orbit, and by macroscopical examination. For the latter investigation it is all-important that a large piece of the swelling should be removed; otherwise the macroscopical appearances may be easily misinterpreted.

PROGNOSIS.—The prognosis is bad. Early diagnosis is seldom made, and efficient removal is difficult. Rapid recurrence, ending in death is the rule.

TREATMENT.—Intranasal operations are unavailing for this form of growth. The upper jaw should be removed on the affected side, and as much of the lateral mass of the ethmoid as is possible should be taken away.

FIBRO-SARCOMA.—In addition to the growths of unquestionable malignancy just described, mention must be made of a group of tumours usually included under the title of "sarcoma," but exhibiting much less definite evidence of malignancy. In their clinical course, and in the symptoms to which they give rise, they resemble the fibromata of the naso-pharynx. The tumours, however, are softer and grow more rapidly, and have a greater tendency to local recurrence. There is no dissemination. Microscopically they are seen to contain large numbers of spindle cells in the fibrous tissue. For their treatment only local removal is necessary; a careful watch, however, must be kept for recurrence.

Nasal Polypi.—In children the most noteworthy fact with regard to polypi is the rarity of their occurrence. In a series of 850 cases recorded by Alexander, only 10 occurred in patients under ten years of age. Other authors publish similar statistics. The youngest patient seen by Leek was three years old, and the condition has not been recorded under the age of five months. The writer has met with polypi in patients under ten years of age on two occasions; in each case the polypus was single and unusually large. There was no evidence of sinus suppuration.

SYMPTOMS.—The symptoms are the same as in adults—viz., slowly progressive nasal obstruction on one or both sides.

DIAGNOSIS.—The diagnosis is established by finding a semi-transparent smooth swelling of soft consistency which has a narrow attachment to the middle turbinate or lateral mass of the ethmoid.

CONGENITAL DEFECTS OF THE LARYNX.

The congenital deformities of the larynx which are most frequently met with are—(1) Webs joining the vocal cords; (2) the condition which gives rise to congenital laryngeal stridor.

Webs.—The web is always situated in the anterior part of the larynx, and unites the two vocal cords. It is yellowish-white in colour, triangular in outline, the posterior free margin being slightly concave. The anterior part is thicker than the posterior. The size of the web is variable; it may extend as far back as the arytenoids, or may merely round off the acute angle between the cords at the anterior commissure. Its presence is due to a non-absorption of tissue during development. It is sometimes noted that the web is double, a second web being visible below the first.

The symptoms depend on the size of the web. A small web causes hoarseness, dating from birth, and no other symptoms. The larger webs render the voice high-pitched, feeble, and give rise to difficulty in respiration.

TREATMENT.—A small web which merely fills up the anterior commissure may be left untouched. The larger webs must be removed by an endolaryngeal operation. There is a strong tendency for union of the raw surfaces to occur in the anterior commissure. When a very large web causes serious obstruction, preliminary tracheotomy is necessary before any attempt at removal.

REFERENCE.

HANSERIK: *Zeitschrift für Laryngologie*, vol. i, part i, 1906.

Congenital Laryngeal Stridor—Introductory.—The symptom of laryngeal stridor, dating from birth or soon after, is well known, but its pathology has been a matter of much debate.

ÆTIOLOGY.—No predisposing causes are known. It does not depend upon the presence of adenoids.

SYMPTOMATOLOGY.—At birth or within a few weeks it is noticed that respiration is accompanied by a various stridulous noise. The noise increases steadily as the breath is drawn in, becoming a crowing sound at the height of inspiration. Expiration is silent. The voice is clear and cough natural. There is no cyanosis nor any sign of distress during quiet respiration. When the infant cries or becomes excited, the noise is much louder, and may only become audible on such occasions. Some indrawing of the chest wall is nearly always present.

PROGNOSIS.—During the first six months of life the symptom may become intensified, but with continued growth the stridor gradually diminishes, and usually disappears by the age of two years.

PATHOLOGY.—Lack and Sutherland made laryngoscopic examination of six cases. The epiglottis was folded on itself, and the aryteno-epiglottidean folds were lax and in close apposition, reducing the upper aperture of the larynx to a narrow slit. Post-mortem examinations by Conley and Kaplik showed the same condition, and all these observers agree that the stridor is produced by the vibrations of the lax aryteno-epiglottidean folds, which have not sufficient firmness to resist the sucking action of inspiration. They regard the condition as a primary deformity of the upper part of the larynx.

John Thomson and Logan Turner also attribute the noise to the vibration of the aryteno-epiglottidean folds, but are of the opinion that this is set up by spasm and imperfectly co-ordinated respiratory efforts which suck in the membranous upper aperture of the larynx. They have shown that a similar condition can be produced in the cadaver by violent artificial suction through the trachea.

Patonson examined five children by the direct method. He particularly noted that the stridor became more marked under deep anaesthesia. The arytenoids and lax mucous membrane on the upper margin of the cricoid were drawn into the larynx at each inspiration, and produced the noise by their vibration.

DIAGNOSIS from papilloma of the larynx must be made by the absence of hoarseness and freedom of expiration; from diphtheria and laryngitis stridulosa, by the history dating from soon after birth, and by the absence of distress and cyanosis.

PROGNOSIS.—The stridor commonly disappears during the second year. Broncho-pneumonia and other respiratory diseases run an unusually severe course.

TREATMENT.—There is no treatment.

REFERENCES.

- LOGAN TURNER: *Brit. Med. Journ.*, 1906, ii, 1480.
 LACK AND SUTHERLAND: *Lancet*, 1897, ii, 652.
 PATONSON: *Brit. Med. Journ.*, 1906, ii, 1447.

ACUTE LARYNGITIS.

- ACUTE CATARRHAL LARYNGITIS.
 SPASMODIC LARYNGITIS (LARYNGITIS STRIDULOSA).
 EROSIANT LARYNGITIS.
 MEMBRANOUS LARYNGITIS.
 LARYNGEAL STENOSIS.

Acute inflammation of the larynx in children exhibits certain features which serve to distinguish it from the corresponding condition in adults. These features depend upon (1) the small size of the larynx, which easily becomes obstructed; (2) the laxity of the mucous membrane, which is much more apt to swell when inflamed; (3) the readiness with which spasm is induced.

Acute Catarrhal Laryngitis.—**ETIOLOGY.**—Laryngitis is set up by the same causes that lead to a cold in the head, and any severe attack of rhinitis is likely to be accompanied by laryngitis. The inhalation of irritating vapours, a dusty atmosphere, and overuse of the voice, may set up a laryngitis apart from any infection. Laryngitis is also frequent in certain specific fevers, particularly measles. Lastly, a frequent phenomenon is an exacerbation of a chronic inflammation.

SYMPTOMATOLOGY.—A tickling sensation in the throat leads to frequent cough of a husky character, and the voice is rough. There is a sense of discomfort every time the saliva is swallowed, but at meal-times the swallowing of food gives a sense of relief rather than pain. In the more severe cases the voice is very hoarse, and may even be lost; the cough is loud and croaking. Examination of the larynx is rare on such occasions; but if it is made the mucous membrane is seen to be reddened and swollen, especially over the arytenoids. The vocal cords themselves show surprisingly little change. In the mild cases they are a little swollen, slightly pink, with an excess of mucus lying on and between them. In the more severe cases the swelling of the ventricular bands and arytenoids is greater, and in addition there may be considerable swelling of the subglottic mucous membrane, leading to continuous stridor. The general health is not disturbed, save for a slight rise of temperature up to 100° F.

DIAGNOSIS.—The husky voice, the croaking cough, make the detection of laryngitis easy. The detection of the cause is not so simple. It must be remembered that acute laryngitis may be an early symptom in measles, broncho-pneumonia in infants, and in diphtheria. Koplik's spots afford great assistance in excluding measles, and a bacteriological examination will determine the presence or absence of the diphtheria bacillus. In broncho-pneumonia the accompanying pulmonary symptoms should direct attention to the chest.

PROGNOSIS.—In all save severe obstructive cases, recovery is rapid, and the symptoms pass off in a few days. In babies, however, obstruction to respiration is apt to occur rapidly, and is always a serious danger.

TREATMENT.—When treatment is necessary, the patient must be confined to bed in a room with a temperature of 60° F. Considerable advantage is often obtained from the use of a steam-kettle. The neck should be wrapped up, and at intervals hot fomentations should be substituted for the dry wrapping. Internally small doses of syrup of Tolu, vinum ipecac., and tinct. campb. co., suitable for the age of the patient, relieve the cough and make it easier to clear the larynx.

Spasmodic Laryngitis (Laryngitis Stridulosa).—When children under the age of four are attacked by catarrhal laryngitis, laryngeal spasm may be added to the symptoms already mentioned; this is more apt to happen to the members of certain families. The presence of adenoids predisposes to the spasm.

SYMPTOMATOLOGY.—The patient suffers from hoarseness, croupy cough, feverishness, and other symptoms of no particular severity, during the daytime. At night, however, attacks of laryngeal spasm of the most alarming character occur. The child becomes restless, and wakes up struggling for breath, with well marked inspiratory stridor, so that it appears about to die from obstruction. After a time, however, the breathing becomes easier, and the child falls to sleep. A second or third attack may occur in the same night, and on several nights in succession. It appears probable that the spasm is set up by the collection of tenacious mucus in the larynx, and that as soon as this is coughed up the patient enjoys comparative comfort.

DIAGNOSIS.—The only difficulty is to determine the absence of membranous laryngitis. Chief reliance should be placed on the suddenness of the onset, the absence of cyanosis, and the rapid disappearance of the stridor under treatment.

TREATMENT.—The treatment is the same as for the catarrhal laryngitis, but in addition special measures must be taken when the spasm comes on. A hot bath, temperature 100° F., should be given at once. Should the spasm persist, vomiting must be induced; tickling the back of the pharynx or the administration of a drachm dose of *vinum ipecac.* generally suffices. The relief which follows the vomiting is remarkable. If relief can be obtained in no other way, intubation must be performed, and in an emergency a gum elastic catheter may be used for that purpose.

Edematous Laryngitis.—Edema of the larynx may result from (1) drinking scalding liquids or from taking food which is too hot; (2) from certain infectious processes severe enough to produce edema of the tissues. In the former case it is the epiglottis and the upper aperture of the larynx which are affected principally; in the latter, the subglottic area and the trachea are apt to be swollen as well as the interior of the larynx. In addition to evidence of laryngitis, there is steadily increasing dyspnoea, threatening death from asphyxia. A low tracheotomy, in the opinion of the writer, is the best means of obtaining relief.

Membranous Laryngitis.—Membranous laryngitis, with the exception of those cases due to diphtheria, is a rare event, but exceptionally it appears possible for a streptococcal or pneumococcal invasion of the larynx to lead to the formation of a false membrane.

For *Diphtheritic Laryngitis*, see Chapter XIX., p. 1027.

Laryngismus Stridulus.—By laryngismus stridulus is meant a spasmodic closure of the glottis unaccompanied by laryngitis.

ÆTIOLOGY.—The patients who are most likely to suffer are poorly-developed children between the ages of six months and two years, older children much more rarely. Males are attacked more frequently than females. The majority of cases occur in children who present evidence of rickets. Many patients with tetany are subject to laryngismus. Another important predisposing factor is whooping-cough; attacks of laryngismus may occur for weeks after the whooping-cough has disappeared. The seasonal incidence, as pointed out by Gee and others, is curious. Watson Williams records that, in 100 consecutive cases, 81 occurred during the first six months of the year, and only 19 from July to December.

SYMPTOMS.—As a rule only one symptom is present, and that is arrest of breathing. The child looks frightened, and appears to be holding his breath; holds his head back, and gradually becomes cyanosed, the cyanosis being succeeded by pallor. After a time, measured in seconds, the spasm relaxes and air enters with a crowing sound, and the child, though feeble, is able to resume playing until another attack comes on. Attacks occur both by night and day. Their length and severity vary greatly, and death may occur during the seizure. Between the spasms there are no symptoms. There is no fever, no stridor, and evidence of laryngitis is lacking. Bawling about, attempts to cough, and fits of anger, appear to bring on the laryngismus.

DIAGNOSIS.—The condition can be distinguished from laryngitis stridulosa by the absence of fever and laryngitis between the attacks. There is, moreover, no crowing cough; and the attacks recur over a much longer period.

PROGNOSIS.—The great majority of patients recover completely, but from time to time deaths are recorded.

TREATMENT.—*During the Seizure:* At the first warning that a seizure is imminent, strong smelling-salts should be held to the nose. Cold water (about 60° F.) should be poured over the head and shoulders, and if at the same time the lower part of the body is immersed in a warm bath, the beneficial effect is most pronounced. If breathing still remains in abeyance, the mouth must be opened and the epiglottis looked forward with the finger. *Between the Attacks:* A most careful watch must be kept over the child day and night, for the first attack is apt to be followed by others more severe. The cold sponging should be continued three daily. Small doses of potassium bromide and chloral are recommended to reduce the irritability of the nervous system, but do not prevent recurrence of the symptoms. The essential treatment is that of the general condition. Special attention must be given to the teeth, to the digestion, and to the provision of abundant fresh air; and all the measures directed to the care of infants must be carefully indicated.

CHRONIC LARYNGITIS.

CHRONIC CATARRHAL LARYNGITIS.
CHRONIC TUBERCUL.
LUPUS.
SYPHILIS.

Chronic Catarrhal Laryngitis.—**ETIOLOGY.**—Chronic catarrhal laryngitis is an uncommon affection in children, and is nearly always secondary to inflammation in the nose and pharynx. The chief causes are chronic rhinitis, especially rhinitis sicca, in which affection the larynx is never entirely healthy. Adenoids also undoubtedly predispose to the occurrence of chronic laryngitis, partly by forcing the patient to inhale air through the mouth instead of the nose, partly by allowing mucus and other secretion to collect in the pharynx.

SYMPTOMATOLOGY.—The voice is hoarse or may be almost lost. At times it becomes comparatively clear, but never quite free from roughness. This variation in degree leads to the complaint that the child "early loses its voice." Cough is frequent and harsh. Difficulty in respiration is exceptional, and indicates that something more than catarrhal laryngitis is present.

PATHOLOGY.—All the lining of the larynx is reddened. The vocal cords are red or show a brown discoloration. The margins are uneven, and the surfaces covered with dried secretion.

DIAGNOSIS.—The condition is distinguished from lupus and syphilis by the absence of infiltrated areas and of tissue destruction.

PROGNOSIS.—If the nose and pharynx can be restored to a normal state, the laryngeal condition will improve rapidly, and complete recovery ensue.

TREATMENT.—Any adenoids present must be removed, and rhinitis treated. Locally stimulating oils should be applied by means of inhalations. For this purpose steam plus eucalypti, turpentine, or creosote, are satisfactory.

Chonditis Tubercula (Singer's Nodules).—Although the most characteristic examples of chonditis tubercula are seen among those who use the voice professionally, it also occurs in children. Choir-boys and those who associate much with deaf persons are among the sufferers; but similar nodules are occasionally seen in

the cords of patients who give no history of excessive use. In such cases the conditions appear to be the result of a chronic laryngitis.

SYMPTOMATOLOGY.—The voice is never absolutely clear; a slight degree of roughness is always present, which merges into hoarseness on slight provocation.

PATHOLOGY.—Symmetrical white nodules are found on the inner margins of the vocal cords. They are round in shape, and about the size of a small pinhead. They appear to be embedded in the substance of the cord, and are invariably situated at the junction of the anterior and middle thirds.

Microscopically the nodule consists of an overgrowth of fibrous tissue with some thickening of the covering epithelium.

DIAGNOSIS.—A diagnosis can be made only by a laryngoscopic examination. The size, colour, situation, and symmetrical arrangement, prevents their being mistaken for any other condition.

TREATMENT.—The treatment is that of chronic laryngitis. Any cause of nasal obstruction must be removed; rhinitis must be treated, and with the removal of these sources of irritation the complete disappearance of the nodules is mostly a matter of resting the vocal cords. It is only in rare cases that any further treatment is desirable; the nodules should then be lightly touched with the actual cautery, an operation of considerable delicacy.

REFERENCE.

Günther: *Zeitschrift für Laryngologie*, 1906, x, 45.

Larynx—**INTRODUCTION.**—Tuberculosis in the larynx usually takes one of two forms. The one known as "laryngeal tuberculosis," secondary to disease of the lungs is not uncommon in adults, but rare in children. The other, known as "larynx," usually appears to attack the larynx by extension from the pharynx or nose, the lungs remaining unaffected. This form is that most commonly met with in children.

ÆTIOLOGY.—The same age incidence is observed as in lupus of other parts of the body, and it is seldom that the larynx is the only part of the body attacked. Most usually the nose and the pharynx are affected as well, and there are not infrequently patches on the skin of the face, buttocks, or elsewhere. It appears probable that in the majority of cases the infection reaches the larynx either from the nose or the pharynx.

SYMPTOMATOLOGY.—When the interior of the larynx is affected, long-continued hoarseness is the prominent symptom. In addition there may be obstruction to respiration and discomfort on swallowing. When, as sometimes happens, only the epiglottis is attacked, there may be no symptoms beyond slight discomfort on swallowing. As a rule there is no fever, and the patient's general condition is unaffected. The progress of the disease is very slow, tending to spread in a downward direction from the epiglottis to the arytenoids and the vocal cords. Spontaneous healing may be observed in certain patches, whilst others enlarge and ulcerate. The healing is always accompanied by cicatricial contraction, and when this involves the entire circumference of the larynx very serious laryngeal obstruction results, and forms one of the most formidable complications of the disease.

PATHOLOGY.—The part most frequently affected is the epiglottis; next the aryteno-epiglottidean folds; while the interior of the larynx is attacked least frequently. The lesion consists of small closely-set nodules with a smooth red surface, infiltrating the tissues and slowly destroying them, with but little tendency to ulceration.

DIAGNOSIS.—The condition is distinguished from tuberculosis by the lack of oedema and of deep ulceration, by the absence of lung signs, and by its extremely slow and comparatively benign course. From syphilis it is distinguished by the small amount of destruction, the slow progress, and the absence of other gummatous lesions.

PROGNOSIS.—The course of the disease is always slow; and although many patients respond satisfactorily to treatment, in others recrudescence or persistence of the infiltration make it difficult to obtain a cure.

TREATMENT.—*Local.*—Locally the most satisfactory results are obtained by the application of caustics, and of these puncture with the galvano-cautery is the best. The red-hot needle is thrust into the infiltrated tissue at several points, and acts partly by destroying the diseased tissue, and partly by setting up fibrosis in the immediate neighbourhood of each puncture.

General.—Small doses of arsenic have been of benefit on many occasions. The writer has also seen improvement follow the administration of small doses of potassium iodide. These cases are particularly suitable for the trial of tuberculin injections, and very favourable results have been reported from their use. As in other forms of tuberculosis, diet and climate are of the first importance.

SYPHILIS.—*Introduction.*—The only syphilitic lesions in the larynx which are of importance are those of the tertiary stage, and in a child they are almost invariably congenital.

ETIOLOGY.—As in the nose, the gummatous lesions are seen more frequently in the older children, about the age of seven to twelve years. It is very uncommon to find the larynx only attacked; the pharynx and the palate commonly sharing in the inflammation.

SYMPTOMS.—The symptoms in the earlier stages are slight. There is soreness of the throat and some discomfort during the act of swallowing, but even extensive ulceration causes surprisingly little pain. Thus, invasion of the epiglottis alone gives rise to no symptoms, but when the larynx itself is attacked there is hoarseness or loss of voice, and in the more severe ulcerative lesions severe dyspnoea, which may necessitate tracheotomy.

PATHOLOGY.—The gummata may appear as diffuse infiltrations or definite rounded growths. There is a strong tendency for the swellings to disintegrate, leaving deep ulcers which rapidly eat away the tissues. In this manner the epiglottis may be eaten away, one or both vocal cords may be destroyed, and the cartilages laid bare. In severe or neglected cases necrosis of the cartilages may occur. When healing is complete, the scarring is apt to give rise to great deformity; cicatricial webs form between the remains of the cords, the crico-arytenoid joints become fixed, and a severe intractable stenosis of the larynx results. Benson has called attention to a rare form of the disease; the arytenoids appear as two large rounded symmetrical swellings, having a smooth surface free from ulceration.

DIAGNOSIS.—Syphilis of the larynx is distinguished from catarrhal laryngitis by the presence of infiltration and deep ulceration; from lupus, by the greater amount of ulceration and destruction of the epiglottis, and by its comparatively rapid course. The presence of gummata in the palate or pharynx, and other evidences of congenital syphilis, often afford considerable assistance.

PROGNOSIS.—In neglected cases great destruction may occur before treatment is begun; the cicatricial contraction which follows the healing of the ulcers is liable to produce permanent hoarseness and intractable stenosis of the larynx.

TREATMENT.—In addition to the ordinary antisyphilitic remedies, steam inhalations are to be recommended. In a severe case with obstruction to respiration, tracheotomy may be necessary at any moment.

FOREIGN BODIES IN THE LARYNX.

Foreign bodies tend to enter the larynx by two different mechanisms:

1. A deep inspiration exerts forcible suction in the direction of the widely-opened glottis. Such inspirations precede and follow a laugh or a cough, and also occur during anesthesia and in the unconsciousness of epileptic or other seizures. Any pieces of food or other objects of small size present in the mouth, nose, or pharynx, tend to be carried by such an inspiration into the larynx.

2. The muscular contractions of swallowing may force portions of bone, etc., into the opening.

The character of the foreign bodies is extremely varied: pieces of bone, of meat, pins, nuts, pencil-cases, teeth, tonsils, and turbinates, are items of a list which could be almost indefinitely prolonged.

SYMPTOMS.—As soon as the interior of the larynx is touched by the foreign body, spasmodic closure of the vocal cords occurs, and is followed by violent coughing, which results in the rejection of the irritant unless it has become impacted or passed below the vocal cords. Should the foreign body remain in the larynx, the course of events depends on its character and situation. A large body will completely obstruct the airway; the patient becomes cyanosed, unconscious, and dies from asphyxia, unless help is immediately forthcoming. A small body continues to act as an irritant; attacks of glottic spasm and violent coughing fits succeed one another, during which the patient becomes cyanosed. Gradually the spasm and coughing become less violent, and eventually cease, so that the patient is restored to an apparently natural condition, except for a possible alteration in the voice. The voice may disappear entirely if the foreign body is wedged between the cords, or may be rendered weak and husky; but even when a piece of bone is lying between the cords it is possible for the voice to be strong and clear; indeed, it is a matter for astonishment to note how trivial the symptoms may be when once the larynx has become tolerant. When the mucous membrane is scratched, there may be bleeding, but seldom more than slight streaking of the sputum. A hard body, such as a piece of bone, may remain in the larynx for months, or even years, causing hardly any symptoms, but its presence may give rise at any time to perichondritis, suppuration, and other serious mischief. Soft vegetable or animal matter, such as a bean or piece of meat, rapidly decomposes, and, unless removed, is sure to set up acute inflammation in a short time.

Diagnosis.—A positive diagnosis can be made by inspecting the larynx, and in the majority, at all events, of the younger patients this is only possible under a general anæsthetic by means of Kilian's tube spatula. In the presence of laryngeal symptoms, such an examination is indicated (1) when there is a history of choking during a meal; (2) when the patient has recently been anæsthetized for an operation, such as removal of a tooth; (3) when laryngeal obstruction suddenly appears in an otherwise healthy child. Röntgen rays may reveal the foreign body, but the absence of a shadow by no means excludes its presence. If the patient be seen immediately after the entrance of the foreign body, and there is urgent dyspnoea, an attempt should be made to remove it with the finger or with forceps, if a pair happens to be available. Should the patient be unconscious, and this attempt is not immediately successful, tracheotomy must be performed without further delay with any instrument which is at hand. In the less urgent cases in which there is only temporary choking without persistent dyspnoea, the foreign body can be readily removed by means of forceps. The direct method under general anæsthesia must be used in the case of the young and intolerant patients, whilst the indirect method under cocaine anæsthesia is suitable for older patients. The actual extraction seldom presents any difficulty.

Foreign Bodies in the Bronchi.—Should the foreign body pass beyond the larynx, it may remain in the trachea or lodge in one of the bronchi. As a matter of experience, it enters the right bronchus about twice as frequently as the left, the reason for this being purely anatomical. The lumen of the right bronchus is about 10 per cent. larger than that of the left, and is very little less than that of the trachea. The carina, or division between the two bronchi, is frequently to the left of the middle line of the trachea. Moreover, the axis of the right bronchus is more in a line with that of the trachea than is the left, and the right bronchus therefore appears to be more nearly a direct continuation of the trachea. It is quite easy for an inexperienced bronchoscopist to pass his instrument into the right bronchus without realizing that it is no longer in the trachea.

The foreign bodies which reach the bronchi may be divided into two main groups—the soft and the hard.

The soft bodies include pieces of meat, fruit-seeds, leaves, nut-kernels, and such-like. They all tend to swell up when exposed to moisture, and therefore are likely to cause complete obstruction of the tube in which they happen to lie. Deficient air entry into the part of the lung affected, and collapse of the alveoli, commonly result from their presence. They usually contain bacteria in abundance, so that purulent bronchitis and pneumonia are set up within forty-eight hours. On the other hand, as a result of putrefaction and fermentation, the body may be so disintegrated that it is coughed up in the spittle specimen.

The hard bodies include such objects as pins, needles, beads, pebbles, collar-studs, pieces of metal from tracheotomy tubes, etc. They are incapable of swelling, and, unless they happen to fit exactly, are unlikely to lead to complete obstruction of a bronchus; their mobility allows them to be coughed up into the trachea repeatedly, and even to descend into the other bronchus. They are comparatively aseptic, and need not give rise to inflammation or suppuration for many days.

Teeth, although hard, are apt to carry many pyogenic organisms, and therefore to cause purulent bronchitis at an early stage. Pieces of bone usually have sharp edges, which lacerate the mucous membrane, and so become firmly wedged in position.

SYMPTOMATOLOGY.—The initial symptom in all cases is a severe cough. The patient suffers from bouts of coughing, which appear to be without cause, unless it is suspected that a foreign body has been inhaled. Dyspnoea and a feeling of oppression on the affected side is also present when the object is large enough to cause serious obstruction to air entry. In the case of some of the hard bodies no further symptoms may arise for a considerable time, but eventually serious attacks occur, accompanied by purulent sputum and evidence of bronchiectasis and fibrosis of the lung (vide Chapter VII, p. 385). In the case of the soft bodies dyspnoea is common; sputum containing pus, and possibly blood, appears at an early date, and is often offensive. Later evidence of pneumonia, and even gangrene of the lung, appear, and a fatal conclusion may be anticipated unless relief is obtained.

DIAGNOSIS.—Important assistance may be obtained from a Röntgen-ray examination of the chest, which should always be employed when a foreign body is suspected. It is important to recognize that foreign bodies which are neither metallic nor bony may be revealed in this way.

TREATMENT.—We owe the modern treatment of these cases to G. Killian, who was the first to show that it was possible and safe to pass a rigid tube down the trachea into the bronchus and its main branches. The patient is anesthetized; the bronchoscope is passed through the larynx and trachea into the affected bronchus. Should pus and sputum interfere with the view, they must be removed by the suction apparatus, and a preliminary dose of atropine may help to lessen the secretion. The interior of the air-tube is then examined until the foreign body comes into view. The tube is then manipulated so as to cause the foreign body to project into its lumen; a pair of forceps fitted with a suitable grip is introduced, and, after grasping the body firmly, is withdrawn together with the tube. Very great difficulty may be experienced in grasping and removing the foreign body, even when it is plainly visible. It is therefore necessary to have a number of grips at hand, so that one can be chosen which is suitable for the particular object, and if a duplicate of the body can be obtained, it is always advisable to test the grip on this beforehand. In the case of friable bodies, care must be taken not to crush them, although this may be unavoidable. In spite of all precautions it is sometimes found impossible to remove the foreign body. When the patient is very young, swelling of the vocal cords after the extraction may necessitate tracheotomy. If no bronchoscope is available, or if it is impossible to extract the foreign body, it is best to perform a low tracheotomy. The foreign body may then be expelled during an attack of coughing. This is a comparatively simple method of treatment which has been successful on many occasions.

NEW GROWTHS.

INTRODUCTION.—The appearance of a malignant growth in the larynx of a child is an event of the greatest rarity, and outside the experience of the present writer. Innocent new growths are relatively common, and occur in two varieties:

- Papilloma.
- Fibroma.

Papilloma of the Larynx.—**ETIOLOGY.**—Papillomata have been observed in children under the age of one year, but as a rule they make their appearance between

the ages of three and seven. The conditions which give rise to the growths are obscure, neither predisposing nor exciting causes being known. The multiplicity of the tumours and their recurrence after removal suggest that the growths are inflammatory in origin and infective in nature, but proof is lacking.

SYMPTOMS.—The presence of papillomata on the vocal cords is indicated by a gradually increasing hoarseness and loss of power in the voice. Attempts at speech may only produce a husky whisper, and the voice may be lost completely. The growths obstruct the lumen of the larynx, and lead to noisy respiration, which develops into dyspnoea when catarrh is present. Attacks of difficult breathing occur with increasing frequency, and are apt to become a prominent symptom when any intercurrent affection, such as measles or bronchitis, attacks the patient. It is probable that spasm accounts for these to some extent. During one of these attacks tracheotomy may become necessary to prevent death from suffocation. When the vocal cords are not affected, there is no hoarseness, and attention is only drawn to the growth by the occurrence of dyspnoea.

PATHOLOGY.—Papillomata in children are usually multiple, sessile, and of the compound type; if single, the growth is pedunculated. Each papilloma is red or pink, with a rough warty surface and of a firm consistency. Usually they are found set thickly on the vocal cords and ventricular bands; more rarely on the epiglottis, the aryteno-epiglottidean folds, and exceptionally below the vocal cords. In a severe instance the lumen may be so narrowed as barely to admit a probe.

DIAGNOSIS.—The condition must be suspected whenever long-continued hoarseness is accompanied by increasing dyspnoea in an otherwise healthy child. The diagnosis can only be established definitely by inspection.

PROGNOSIS.—Life is threatened by the difficulty in breathing, which may become urgent at any moment. The papillomata are said to show a tendency to disappear after a period of three or four years without any active local treatment. Unfortunately, in the writer's experience this is the exception rather than the rule, and the persistence with which they return after an apparently complete removal is one of the chief difficulties encountered in treatment. He has removed many growths from the larynx of a woman aged forty who asserted that the condition had been present as long as she could remember. Others have recorded similar cases. When the vocal cords are thickly covered, the voice is never likely to become perfectly clear, even after the most careful removal of the growths. There is no evidence that these growths lead to malignant diseases.

TREATMENT.—The treatment consists in the removal of the growths. The most satisfactory means of accomplishing this is by the direct method, using Killian's tube-spatula. After the patient has been anesthetized, a weak mixture of cocaine and adrenalin is applied to the interior of the larynx, and the growths picked off by means of forceps or a wire snare. In spite of the utmost care to avoid injuring healthy tissue, the removal is apt to leave the vocal cords with somewhat roughened edges. Two or three operations may be necessary in order to clear the larynx thoroughly, and the surgeon requires great patience and perseverance to obtain good results. Tracheotomy is frequently necessary. In it is the only means of giving relief during a severe attack of dyspnoea. It is also a necessary preliminary to the operation described above, when the mass of papillomata is large enough to cause serious interference with respiration. In addition to these indications for tracheotomy, that operation has been recommended as the

sole method of treatment. The larynx is set at rest, and it is claimed that this is sufficient to cause spontaneous disappearance of the growths; but no reliance can, in the opinion of the writer, be placed on this procedure. Thyrotomy has also been advocated and practised. When the larynx is opened, it is possible to remove the growths very thoroughly. Nevertheless, there has been recurrence even after so thorough a removal. The operation is severe; the voice is in jeopardy; cicatricial contraction may ensue; and the results are not sufficiently certain to justify the procedure.

Another method of treatment is by means of Lön's tube. This instrument is a cylinder with windows in its wall. When passed into the larynx, the papillomata project through the openings into the lumen of the cylinder, which is then withdrawn smartly, and brings with it the projecting pieces of growth. In this way a passage can be made rapidly through the larynx. Mosher has also devised an instrument whose principal feature is a spiral spring in which the growths are to be strangled. Claoué and other French writers have recommended the internal administration of calcined magnesia. About 75 grains is given daily for a fortnight, and then the dose reduced to 20 grains daily, which is continued for several months. It is claimed that the growths gradually disappear.

Fibroma of the Larynx.—INTRODUCTION.—Under the title of "fibroma" is included a group of small laryngeal tumours possessing somewhat different characters when viewed microscopically. The tumours are called fibroma, angio-fibroma, angroma, fibro-myxoma, or myxoma, according to the tissue which is found to predominate; but clinically they all behave in similar fashion.

SYMPTOMATOLOGY.—Even a very small fibroma situated on the edge of the vocal cord gives rise to changes in the voice, and roughness, huskiness, or loss of power, is usually the only symptom; for the tumour is rarely large enough to give rise to laryngeal obstruction. When the tumour is not situated on the vocal cord, it gives rise to no symptom until its size causes stridor, or its situation permits of its being caught between the vocal cords and so causing spasm.

PATHOLOGY.—These swellings are usually situated on the vocal cords at the junction of the anterior and middle thirds or in the neighbourhood of the anterior commissure. Rarely they are found on the aryteno-epiglottidean folds, on the ventricular bands, or in the subglottic space. They are smooth, rounded, and sessile. The colour varies from a pale pink to a deep red, according to the vascularity of the swelling; occasionally a larger stalked form is seen. Microscopically the swelling consists of fibrous tissue, fibrin, blood-clot, and collections of thin-walled vascular spaces. The proportions of these constituents vary greatly in different specimens.

DIAGNOSIS.—The diagnosis is readily made when the larynx is inspected, the smooth, rounded surface of the tumour, its sessile broad attachment and red colour, distinguishing it from other swellings.

PROGNOSIS.—There is no tendency to recurrence, and when the tumour is completely removed the cure is permanent.

TREATMENT.—Under an anæsthetic Killian's tube-spatula is passed into the larynx, and the swelling removed by means of a pair of forceps. If the tumour is very minute, it should be touched with the galvanic cautery.

CHAPTER VII

DISEASES OF THE RESPIRATORY SYSTEM

G. A. SUTHERLAND

THE PLEURA:

PLEURISY: SEROUS AND PURULENT.
PNEUMOTHORAX.
HÆMOTHORAX.
CYSTOTHORAX.

THE TRACHEA AND BRONCHI:

BRONCHITIS: ACUTE AND CHRONIC.
FIBRINOUS BRONCHITIS.
ASTHMA.
FUNGUS BALLS IN THE BRONCHI.

THE LUNGS:

ATELECTASIS: CONGENITAL AND ACQUIRED.
PASSIVE COLLAPSE.
EMPHYSEMA.
RHOCHO-PNEUMONIA.

THE LUNGS (continued):

LOCAL PNEUMONIA.
CHRONIC INTERSTITIAL PNEUMONIA.
CONGESTION AND ŒDEMA.
TUBERCULOSIS OF THE LUNGS AND
BRONCHIAL GLANDS.
CARCINOMA AND ABSCESS OF THE LUNG.
NEW GROWTHS OF THE LUNGS, PLEURA
AND MEDIASTINA.
HYDATID DISEASE.
ACTINOMYCOSIS.

THE MEDIASTINA:

MEDIASTINITIS.
ABSCESS.
ENLARGED LYMPHATIC GLANDS.
ENLARGED THYROID GLAND.

DISEASES OF THE PLEURÆ.

PLEURISY: SEROUS AND PURULENT.

INTRODUCTION.—The leading characteristic of pleurisy in infancy as contrasted with that disease in adult life is the tendency to purulent effusions. For this the pneumococcus is largely responsible, and it has been calculated that from 75 to 90 per cent. of empyemata are due to that organism. After the age of three years the tendency to purulent effusions becomes less marked. While there is nothing special to the pleurisy of childhood in its dry or serous form, there are many points in connection with the study of empyema in children which are of great importance. At the outset reference may be made to two opinions commonly held and often expressed. The first is that an empyema is easily diagnosed, and the second that the treatment only calls for an amount of surgical skill such as any newly qualified practitioner possesses. Both of these opinions are directly opposed to the experience of those who have had much to do with empyemata in children. It is quite true that an empyema may present no difficulty as regards the diagnosis and the treatment may be carried to a rapid and successful issue with the greatest ease. On the other hand, the diagnosis frequently presents very great difficulty, and many a young patient has died as the result of an undiagnosed empyema. The surgical treatment—for it is admitted that the only thoroughly satisfactory treatment is surgical—calls for a knowledge of the physical laws of pleuro-pulmonary

disease, for surgical skill and experience in the various types of empyema met with, and for great care in the after-treatment if the case is to run an uncomplicated course. There are few senior practitioners who have not received some of the worst shocks of their professional career in connection with errors of diagnosis and treatment of empyema.

ETIOLOGY.—The same varieties of pleurisy are met with in childhood as in adult life. We have to deal with cases of dry pleurisy, of pleurisy with serous effusion, and of pleurisy with purulent effusion. All of these conditions may be met with during the first three months of life, usually as the result of a form of infection from the skin or elsewhere.

Pleurisy is rare as a primary affection, but is common as part of a general infection of the system or as secondary to some local disease of the lung. The so-called "primary pleurisy," often traced to a chill or exposure to cold, is usually to be regarded as tuberculous in origin. A chill may be the exciting cause of an attack, but not the cause *causæ*. In all the general infective diseases, septicæmic or pyæmic, inflammation of the pleura may occur. In connection with children, we note especially, amongst general diseases, rheumatism, tuberculosis, scarlet fever, and measles. The pleura also shows a marked tendency to be involved in the general extension of some local disease, as, for instance, in connection with appendicitis, acute septic epiphysitis, umbilical infections, etc. Another large class of pleural affections is secondary to disease of the lungs. Amongst these pneumonia takes a prominent place in early life, and this not so much in the form of an acute pleurisy in the early stage as of a purulent inflammation in the later stages. Pulmonary tuberculosis is very commonly associated with tuberculous pleurisy. Amongst the rarer local affections leading to pleurisy may be mentioned the rupture of a caseous gland at the root of the lung into the pleura, or the bursting of a mediastinal abscess, or the extension of tuberculous disease from the spine, or a malignant growth in the lung or mediastinum. Injury may lead to pleurisy, as in the case of a fractured rib.

While pleurisy may occur during any part of the year, the chief seasonal incidence is in the colder months, and is directly dependent on the prevalence of pulmonary disease at that time. Age is a factor of importance not so much in determining the frequency of the disease as the purulent character of the effusion in infancy.

VARIETIES OF PLEURISY.—1. **Non-Purulent Pleurisy.**—There are in childhood the two usual forms—namely, (1) *dry pleurisy*, (2) *pleurisy with serous effusion*. Speaking generally, the symptoms, physical signs, and treatment, are the same as in adult life.

Many cases of *dry pleurisy* are characterized by an entire absence of symptoms, and are only discovered post mortem in the shape of recent inflammation or dense pleural thickening of old standing. These conditions are specially common in connection with tuberculous disease of the lungs. While at the onset of an attack of pneumonia there may be acute pain in the chest and audible friction, in children much more frequently than in adults, there may be an entire absence of pleuritic pains and signs during a pneumonic attack. On the other hand, the pain in pneumonia is frequently referred to one or other side of the abdomen, and is probably due to pleurisy about the lower lobe (see *Lober Pneumonia*). The pain of pleurisy is ascribed by some to involvement of the intercostal nerves in the inflammation, and hence it may be located by the patient at the peripheral ends of these nerves. By others—e.g., James Mackenzie—the pain is ascribed to a visceral reflex

from the pulmonary pleura leading to a disturbance in the corresponding spinal segment, which is expressed by pain referred to the areas of distribution of the nerves of that segment. Sometimes there is no actual pain present, but distinct tenderness over the chest at or near the seat of the lesion, as manifested on percussion, or there may be surface tenderness in the abdomen or back at some distance from the lesion. In some cases of pneumonia pleuritic pain may be referred to one or other shoulder. In rheumatic fever or in the subacute exacerbations of chronic rheumatism, dry pleurisy or pleurisy with effusion may occur. The affection may be bilateral or unilateral and is often associated with pericarditis. As a condition of dry pleurisy, often with coarse friction, and without symptoms, is frequently discovered in the course of a routine examination of the chest in cases of tuberculosis, rheumatism, or pneumonia, and the disease passes off without any active treatment, it is plain that many attacks of dry pleurisy remain undiagnosed because unsuspected. Other attacks will be recognized if pain is present as a symptom, but the importance of the discovery lies rather in the association of the pleuritic inflammation with some underlying disease, which ought to be treated, than in the pleurisy itself, which calls for little treatment save the relief of pain. Even in cases suggestive of pleurisy from the pain, repressed cough, and appearance of the patient, it is frequently impossible to detect any friction sounds as the child fixes the chest, breathes with the diaphragm chiefly, and will not be induced to draw a long breath. Here one can often make a reasonably safe diagnosis from the symptoms; and as the acuteness of the pain subsides, the breathing will become more natural, and friction sounds will probably be heard. The fine friction of early pleurisy is much less commonly heard in children than in adults, and frequently no adventitious sounds are audible until the coarse friction of a later stage appears.

The treatment of acute dry pleurisy is to be directed to the relief of pain by fixation of the chest in a flannel bandage, by counter-irritation (hot fomentations or a turpentine stupor), and in very acute cases by the application of two or three leeches around the painful part. In cases in which the pleural surface of the diaphragm is involved, the pain and distress of breathing may be so great that full doses of morphia are called for.

Pleurisy with serous effusion may occur in connection with any of the diseases already referred to. The special signs and symptoms and the diagnosis of fluid in the chest will be discussed later in connection with empyema. A passive effusion into the pleura may occur in connection with cardiac or cardio-renal disease, and presents the same features as in adult life. An active attack of pleurisy with effusion may develop acutely with high temperature, dry cough, dyspnoea, and marked prostration, or it may begin quietly without any of these symptoms, the patient continues to go about, and it is only increasing anæmia and lassitude or exertion which leads to an examination of the chest. One pleural cavity may then be found filled with fluid.

In the majority of cases both of these types are tuberculous in origin, although definite proof of the presence of the tubercle bacillus may not be found. In some cases there may be evidence of a tuberculous focus in the lungs. In other cases no such origin of tuberculosis in the lungs or elsewhere in the body may be discovered. The case may appear to be one of primary pleurisy with effusion; there may be no other disease present which one associates with pleurisy, but yet it is advisable to regard and treat the effusion as tuberculous in origin. So far as it

positive von Piquet reaction is of value, it will almost invariably be present in such a case; and if it is not elicited after several applications of the test have been made, then the diagnosis of tuberculosis must be reconsidered. From the clinical standpoint valuable evidence in support of the view that primary pleurisy with effusion is usually tuberculous is obtained by a comparison with tuberculous peritonitis. This affection may begin acutely with pain, pyrexia, and effusion, or quietly with effusion which is only discovered from the enlargement of the abdomen. No one at the present time would diagnose primary or idiopathic peritonitis in such cases, but in the absence of other manifest cause for the effusion all would diagnose and treat the case as tuberculous peritonitis. It has been said that the complete cure which often ensues in such cases of pleurisy with effusion is evidence against the disease being tuberculous. The fact, however, is that tuberculous pleurisy is an affection in which the prognosis is unusually favourable, in the absence of other tuberculous lesions, just as is the prognosis of tuberculous peritonitis. The real question of importance in connection with the prognosis of tuberculous pleurisy is as to the state of the lungs and the bronchial glands. If there is definite and active tuberculosis in either of them, then the prognosis as regards the patient's future must be grave, although the pleurisy may clear up entirely. If, on the other hand, there is no marked focus of disease in the lungs or glands, the prognosis of "primary" pleurisy, even when tuberculous, is by no means unfavourable.

While by means of elaborate laboratory methods it may be possible to detect tubercle bacilli in the pleural exudate, and by inoculation of the fluid into animals to produce tuberculosis, in a number of cases the results are negative, and for practical purposes it may be said that tubercle bacilli will not probably be found even in a tuberculous effusion.

As regards cytological examination of the exudate, Emanuel sums up the work of various observers as follows: (1) In tuberculous effusions there is a preponderance of lymphocytes; (2) in other inflammatory effusions the polynuclear cells are the more abundant; and (3) in mechanical transudations the cells are fewer than in either of the foregoing varieties, and consist of flat endothelial cells, often in groups.

The clinical course of an attack of tuberculous pleurisy will depend a good deal on whether the lung is healthy or tuberculous. In the latter case the pleurisy will probably become a chronic complication of the pulmonary disease. In the former we have first of all a febrile stage with active effusion. The fever ranges from 100° to 102° F., and lasts for from two to three weeks, a gradual desferescence by lysis taking place. The effusion lasts longer, and may increase to such an extent as to induce marked symptoms of pressure calling for relief by aspiration. In the case of young children with pleural effusion, it ought to be the practice to insert an exploring needle in order to determine whether the fluid is purulent or serous. The distinction can never be made with absolute certainty by any other means, and the risks of leaving a purulent effusion untreated are very great. After from three to six weeks a tuberculous effusion tends to subside, and the whole may be absorbed without leaving any permanent changes in the pleura. Or the fluid may be absorbed, but there may be left great pleural thickening, permanently interfering with the full expansion of the lung and the chest wall on the affected side. Or, in rare cases, a secondary infection of the pleural exudate may take place, leading to pus formation and empyema. This will usually be manifested by pyrexia and acute illness.

We have taken tuberculous pleurisy as a type of pleurisy with effusion because it is the most common and the most pronounced form met with in childhood. Other cases of serous effusion arising in connection with rheumatic fever or pneumonia are rather to be regarded as complications of these diseases, of importance it may be, but not possessing any features which have not been dealt with in connection with tuberculous pleurisy.

TREATMENT.—During an attack of tuberculous pleurisy the patient should be kept in bed during the whole of the pyrexial stage, and until absorption of the fluid is well advanced. There is always a liability, on the part of patients suffering from pleural effusion, to suffer cardiac failure if they are allowed to move about or even sit up in bed; so that the recumbent position should be maintained. The symptoms of an acute onset are to be relieved on the lines already indicated with reference to dry pleurisy. In the stage of active effusion with pyrexia there is probably no benefit to be obtained by aspirating the chest, as the fluid will most likely re-accumulate. Aspiration may be called for if signs of positive pressure by the fluid are present, a stage usually indicated by the disappearance of sternal resonance below the clavicle, and the pushing downwards of the stomach on the left side, or the liver on the right, through supra-diaphragmatic pressure. Even if these physical signs are not present, if symptoms such as breathlessness, cyanosis, faintness, or weak cardiac action, occur, these are to be regarded as indications calling for the immediate removal of fluid. The amount of fluid which is tolerated without discomfort and without danger varies greatly in different cases. In the absence of these urgent indications, which are not usually present, one may apply equal parts of the liniment and tincture of iodine, or *unguentum hydrargyri compositum*, or *linimentum hydrargyri*, to the affected side of the chest to allay inflammation and promote absorption. The internal administration of salicylate of soda, iodide of potash, or diuretics, is not of any especial value during the acute stage, and should be reserved for the time when absorption has begun, when they may possibly aid to some extent. The diet should be restricted in amount during the feverish stage, but, if the appetite is good, solids in the form of bread and butter, puddings, and eggs, may be allowed. After the fever has subsided we may find that the amount of fluid in the chest has begun to diminish, in which case it is advisable to continue on the same lines of treatment until it has disappeared entirely. The patient may then be allowed out of bed. On the other hand, we may find that the amount of fluid is not decreasing, and aspiration is called for. As a general rule it may be said that, if fluid has been present in the chest for two or three weeks, and the pyrexia has subsided, its removal is advisable. There is one condition which may make one hesitate as to the advisability of aspiration—namely, the presence of signs of active pulmonary tuberculosis on the affected side. It is held by some that the rest afforded to the lung by the presence of the pleural fluid and the accompanying pulmonary congestion is more favourable to the healing of a tuberculous lesion than the active pulmonary movements which are again re-established on the withdrawal of the fluid. Others hold that the freer pulmonary movements aid in curing the tuberculous lesion. It may be pointed out that, while an active increase in the signs of pulmonary tuberculosis not infrequently follows the removal of fluid from the chest, it is rare to find such activity progressing while the fluid is still present. So that, while the removal of the fluid in ordinary cases of tuberculous pleurisy is called for in order to secure a restoration of the pulmonary and pleural functions, it may be advisable to delay

aspiration in cases of active pulmonary tuberculosis so as to check, if possible, the progress of the latter trouble.

If removal of the fluid has been decided on, we have the choice of siphonage or aspiration. Siphonage by means of a trocar and rubber tubing filled with water has an advantage over aspiration in that the steady flow causes less disturbance of the pulmonary circulation. Whichever method is followed, the withdrawal of the fluid should be carried out steadily and slowly, and the occurrence of marked coughing or faintness or hemorrhage should at once bring the proceeding to a close. It is by no means necessary to empty the pleural cavity; the withdrawal of a few ounces will often lead to the rapid absorption of what remains. Incision of the pleura and drainage is rarely practised in this country for serous effusions, and does not seem to be called for.

Serous effusions, other than tuberculous, are to be treated on the same lines. As a rule, after an attack of two or three weeks, spontaneous absorption of the fluid takes place without aspiration. Rheumatic effusions are part of the general disease, and are to be treated with salicylate of soda. Effusions secondary to malignant growths are to be aspirated if the symptoms of pressure are present.

In the case of tuberculous pleural effusion, the after-treatment is of great importance. Many recover and show no further evidence of active tuberculosis. On the other hand, the fact that the lesion is tuberculous is sufficient reason for a prolonged course of dietetic and hygienic treatment, such as is employed in cases of chronic pulmonary tuberculosis. Special care must be directed to the removal of chronic thickening of the pleura, and to the prevention of the development of a chronic fibroid induration of the pleura and lung. Physical exercises directed to the expansion of the chest will be of service in this connection.

2. Purulent Pleurisy, or Empyema.—Empyema is almost invariably a secondary affection, and it is therefore desirable to ascertain as soon as possible what is the primary disease. In 75 per cent. of cases the disease is pneumonia.

The factor which makes a fluid effusion in the pleural cavity of a purulent character is the presence of a pus-producing organism. The chief difference between a serous and a purulent effusion is bacteriological. It is extremely rare to find an effusion which was serous at the onset becoming purulent later on. It must be kept in mind, however, that a serous effusion may be cloudy in appearance from an excess of cellular elements without being purulent.

It is generally recognized that in children under three years of age, if there are definite signs of pleural effusion, the fluid will in all probability be purulent. The frequency of the pneumococcus, either alone or with some other organism as the cause of empyema, has been estimated by different writers at from 75 to 90 per cent. of all cases. Certainly it is the organism which explains the frequency of empyema in early life. The question has been discussed as to whether a pneumococcal empyema is ever primary—i.e., arises without any pneumonic change in the lungs. Recognizing that a pneumococcal infection may be a general blood-disease, without any recognizable pulmonary lesion, we have to admit the possibility of an empyema arising as part of the general infection. As a matter of clinical experience, however, it is rare to meet with a case of pneumococcal empyema in which there have not been signs and symptoms strongly suggestive of pneumonia, or a history of some such signs and symptoms. In all probability the so-called "primary" pneumococcal empyema is associated with some lesion of the lung or with some lesion of the tonsils, naso-pharynx, or alimentary tract. The other

forms of empyema are also secondary in character. The *Staphylococcus pyogenus aureus* may be the cause, and this in children is often associated with osteomyelitis. Streptococci may be the active organisms, associated with scarlet fever, or diphtheria, or some focal infection such as otitis media or appendicitis. Friedländer's bacillus, the *Bacillus coli*, and the tubercle bacillus, are other organisms present in empyemata. As regards the tubercle bacillus, it is doubtful whether this is ever the sole cause of pus in the pleura, and in most cases some other organism is also present, commonly the pneumococcus. The important point about the discovery of tubercle bacilli in an empyema is that, probably, there is some active pulmonary or glandular tuberculosis present which renders the prognosis much more serious. It is usually held that the pneumococcus produces a comparatively mild form of empyema, and that the streptococcus is accompanied by a much more severe type of disease. As a general rule this is true, but, as Bythell has pointed out, we may meet with extremely virulent attacks associated with the pneumococcus, and very mild ones of streptococcal origin. The bearing of the bacteriological finding on the prognosis of an empyema has been summed up by Bythell as follows:

1. If the pus contains but a small number of poorly stained micro-organisms and gives feeble cultures, the prognosis is on the whole good.
2. Abundant and well-stained micro-organisms, giving vigorous cultures, do not necessarily indicate a bad prognosis, especially when there is a large amount of phagocytosis to be seen under the microscope. If, however, the phagocytosis is poor or absent in such cases, the prognosis is nearly always bad.
3. Vigorous cultures are not in themselves a sign of pathogenic activity.

SYMPTOMATOLOGY OF PLEURAL EFFUSIONS.—If one has been following up the development of pleural effusions in a patient who has been suffering from acute pleurisy or pneumonia, there is not as a rule much difficulty in making a correct diagnosis. Very frequently, however, advice is sought because of some indefinite signs of ill-health following an illness for which medical treatment was not considered necessary.

If there is a *history of acute pulmonary trouble recently*, one may be directed at once to examine the chest. Such a history will frequently take the form of "an attack of congestion of the lungs" or "an attack of influenza," from which the patient had never thoroughly recovered. It may be some weeks or it may be some months after an attack of pneumonia before medical advice is sought, but an indefinite illness after pneumonia is always strongly suggestive of empyema. Many cases also occur in which there is no history of any previous illness, and this is often in cases of tuberculous pleural effusion. The only symptoms complained of or observed by the patient may be breathlessness and tiredness on exertion. When the patient is too young to describe them, even these symptoms may have been entirely overlooked. *Cough* may be present, of a sharp, irritating, irritable character, which often suggests to the trained listener the presence of pleural irritation. On the other hand, cough may be entirely absent. The presence of *cyanosis* is to be noted in all cases of pleural effusion of some weeks' duration. This is visible both in the skin and the mucous membranes. In purulent effusions the *acrocyanosis* shows itself as a peculiar straw-coloured tinge of the skin of the face and the body generally, which is very suggestive of the underlying affection. *Edema of the chest wall* on the affected side is sometimes present in cases of empyema.

Clubbing of the fingers which has developed rapidly and without cyanosis is another condition sometimes said to be diagnostic of a purulent effusion. Although

such a statement is too sweeping, the fact remains that the combination of the peculiar yellowish complexion with shibbling of the fingers and toes is more characteristic of empyema than of any other affection. Albuminuria is often present in chronic empyema.

Breathlessness is a symptom which varies in degree with the amount of effusion present. In an ordinary effusion the breathlessness may be present only on exertion. In a rapidly advancing effusion the breathlessness is much more marked than in a slowly progressive one. If a very large effusion is present the breathlessness may be urgent and indicative of great danger to life. Wasting is not characteristic of simple serous effusion, but is frequently present in empyema. Goodhart lays stress on a puffy appearance of the face as indicative of a large effusion, simple or purulent. Venous engorgement of the face and neck points to a large effusion with cardiac embarrassment. In an ordinary case of pleural effusion in a child there is no superficial venous congestion. The temperature in both serous and purulent effusions may be definitely pyrexial, or hectic in type, or but slightly raised at night, or constantly normal. As a rule the temperature at the onset is raised and a high degree of fever may be reached, especially in purulent cases. In the severe forms of empyema a hectic temperature may be developed with sweating and rapid emaciation. In the milder forms, after a pyrexial stage, the fever disappears; the chest may contain a large amount of pus, but the symptoms are those of a chronic toxæmia, with local disturbance (mechanical) of the cardio-respiratory system. If an examination of the chest has been omitted, the general signs of empyema will often suggest chronic pulmonary tuberculosis as the diagnosis.

The physical signs in the chest are usually easily recognized in cases of free fluid. The problems associated with localized pleurisy, or empyema, or bilateral effusions, or a pleura previously affected with disease, are quite different, and present much greater difficulty in the diagnosis. In cases of free fluid there are certain physical changes in the lungs and surrounding tissues, which were first described by Garland and Douglas Powell, and which are especially well manifested in childhood. We may consider the chest as consisting of a bony framework, firm but flexible, and closed below by the movable diaphragm, and of two elastic bags, the lungs, which are everywhere unattached, except at the roots. When the lungs are equally expanded, the mediastinum, with the heart and other contents, is maintained in a central position, although not in any way immobile. The diaphragm is also a structure of great importance, because it is the chief factor in the expansion of the lungs by its descent during inspiration.

These structures are markedly affected by the presence of a moderate effusion. Let us assume the effusion is on the left side. The chest wall on that side has been to a considerable extent thrown out of action, so that as an inspiratory factor it may be left out of account. The left lung has not been expanded as usual, owing to the diminished movement on that side, and from its elastic reactivity it has been shrinking before the increasing fluid, and naturally shrinking towards its one fixed point, the root. The diaphragm on that side has also suffered in its muscular power, so that on relaxation (expiration) it tends to follow the shrinking lung above it, and to reach a higher level than normally. On the sound side of the chest the conditions are very different. There is a call here for fuller pulmonary expansion to counterbalance the effects of diminished respiratory power on the left side. The right side of the chest expands more, and the diaphragm con-

tracts more powerfully on inspiration, with the result that the right lung is more fully distended and its elastic tension is raised. This elastic tension, being opposed by a similar tension in the left lung, will tell on the mediastinum and its contents, and tend to draw the heart over to the right side. If this tension in the right lung is maintained for some time, as in the presence of an increasing effusion, it will tend to draw towards the right side, not only the mediastinal contents, but also the left lung, and, following the left lung, the diaphragm on that side will rise to a still higher level. The left side of the chest, unopposed by active muscular movements, will be driven in by the atmospheric pressure.

The evidences of these changes are manifested on an examination of the chest. The *deficient expansion* of the left side of the chest will be shown but slightly, if at all, during normal (diaphragmatic) breathing. On forced breathing, however, it will be much more evident. The chest wall on that side is not expanded, and as time goes on it tends to become flattened in the axillary region, from unopposed atmospheric pressure, with an outline more rectangular than cylindrical.

As regards the lung on the left side, the line of shrinkage will be approximately accurately shown by an arc of a circle drawn with the root of the lung as the centre and the tip of the apex of the lung as the periphery. The root of the lung is located anteriorly at the junction of the second costal cartilage with the sternum, and posteriorly one inch to the left of the tip of the fourth spinous process in the dorsal region. Exposed in other words, in the presence of pleural effusion, the lower border of the lung is highest in the axillary region, and from this point slopes downwards and inwards anteriorly towards the sternum, and downwards and inwards posteriorly towards the spine. The upper border of the lung is unaltered. On percussion over this lung area the characteristic note is one of impaired resonance, which varies according to the amount of retraction present, and which may be described in different cases as slight, or hoxy, or tympanitic. The note over this area is never flat or wooden. On auscultation the breath sounds are diminished, the expiration is prolonged, and a bronchial element is frequently present over the area, extending downwards from the root of the lung along the spine posteriorly, and along the sternum anteriorly. Bronchial breathing is also frequently present at the extreme apex, and the observation of Goodhart, that while bronchial breathing is present at the apex in a child the base of the lung should be examined for evidence of fluid, is well worth keeping in mind. The area immediately above the lower margin of the lung may show the presence of some crepitations (hypostatic congestion) or friction sounds, but as a rule there are no accompaniments in the retracted lung.

The position of the heart is of great importance. The heart is displaced towards the sound side, not pushed over by the fluid pressure, but drawn over by the unopposed elastic tension of the sound lung. When the effusion is on the left side, the cardiac dulness will be found extending to the right of the sternum, while the apex beat is also moving to the right. In young children a very valuable sign of this displacement is cardiac pulsation seen and felt below the ensiform cartilage, long before there is any evidence of engorgement of the right side of the heart. Visible and palpable cardiac pulsation in abnormal situations will often render clear, without the use of percussion, the altered position of the heart. The signs of cardiac displacement in children are more valuable than in the case of adults because they are manifested early in the disease and are more easily determined.

As regards the area of fluid effusion, the upper border will be found to follow

exactly the lower border of the retracting lung. It will therefore be highest in the axillary region, and from there will slope downwards to meet the cardiac dulness anteriorly, and downwards towards the spine posteriorly. The lower border is very variable, and is by no means to be regarded as identical with that of the normal lung. Even in the presence of a considerable effusion the elevation of the diaphragm is often considerable. One must not therefore assume that the presence of stomach resonance as high as the fourth or third rib in the axilla excludes the possibility of free fluid in the pleura; it is, in fact, strongly suggestive of such a condition. Posteriorly, also, one may find bowel or stomach resonance rising as high as the eighth rib, and this combined with a flat note above is also strong evidence of free fluid in the pleura. This elevation of the diaphragm in childhood is important in connection with exploratory punctures and surgical treatment. For if fluid is diagnosed, and a puncture is made where the pleura is situated anatomically, the needle may readily pass through the two layers of pleura (in apposition), the diaphragm and peritoneum. It has also happened that a surgeon, opening at a supposed "site of election" for pleural effusion, has opened the peritoneal cavity and missed the fluid, which was lying at a higher level.

The characteristic signs of fluid, as determined by percussion and auscultation, may be similar to those met with in adult life. The flatness on percussion, with hard like resistance, is present even with a small amount of fluid, but care should be taken that too firm percussion is not employed, as the note then may have a tympanic character from the underlying stomach or lung. The breath sounds over the fluid area may be faint or inaudible, with a corresponding diminution or absence of vocal resonance. Vocal fremitus is of little value as a test in childhood. On the other hand, a very frequent sign is the presence of bronchial or tubular breathing over the effusion. On quiet breathing it may be distant and feeble, but on deep breathing it may be quite as marked as in pneumonia, and accompanied by bronchophony or pectoriloquy. These signs frequently lead the unwary to diagnose pneumonia, but they are to be recognized as common accompaniments of an extensive pleural effusion. The explanation probably is that, when the breath sounds are absent over a pleural effusion, there is some air-containing lung tissue between the fluid and the bronchi; and when bronchial or tubular breathing is heard, either there is solid lung between the fluid and the bronchi or the lung is so collapsed as to form a solid conducting medium between fluid and bronchi. Although these signs are also met with in adults, they are much more common in infancy and childhood. Whatever the character of the breath sounds on the affected side may be, they will usually be found to contrast markedly with those of the healthy side. The sound lung has an increased amount of work to do, and the breath sounds are exaggerated *puerile*—i.e., an added harshness is present, but not a bronchial or blowing element. *Egophony* is a sign which is not so common in connection with pleural effusion in infancy, but is met with in later childhood.

So far we have been considering the signs of pleural effusion on the left side. The signs when the right side is affected are similar, except that the line of displacement is from right to left instead of from left to right. The stomach resonance is replaced by the hepatic dulness, which tends to be elevated although the liver has not the same range of movement as the stomach. The cardiac displacement in children is not so much to the left as upwards, so that one will find the apex in the fourth or third space.

The signs above-mentioned refer to the first stage of pleural effusion, the stage

in which the fluid is not directly pressing on any of the surrounding structures, but is occupying the space formed by the receding lung. The second stage is reached when the elasticity of the lung is exhausted, and the increase of the fluid leads to direct pressure on the lung, the heart, the diaphragm, and the chest wall. As regards the first two of these—the lungs and heart—the result will be an increased displacement in the same direction. As regards the diaphragm, the result will be that that muscle is pressed downwards, as shown by the lowering of the stomach resonance on the left side and of the hepatic dulness on the right. The chest wall, instead of being driven inwards by atmospheric pressure, will tend to bulge. Fortunately these extreme conditions are but rarely seen now, as the symptoms tend to become more marked with the increase of fluid, and relief is sought and obtained before this dangerous stage is reached.



FIG. 24.—INDURATED INTERLOBAR EMPYEMA FROM A BOY AGED FIFTEEN YEARS.

Two parts of pus were present in the right pleural cavity. (From the Museum of St. Bartholomew's Hospital.)

VARIETIES OF EMPYEMA.—While the most common form of empyema is one in which the fluid lies free in the pleura, in other cases it is bound down by adhesions, so that we have to deal with a localized abscess, and the physical signs already described do not apply. The abscess may be localized in any part of the pleura, but the situations which cause most difficulty in the diagnosis are between two lobes of the lung, or between the diaphragm and the lung, or at the apex of the lung. When the pus is pent up in the hollow along the spine, its recognition by local signs may be very difficult or impossible. This form of empyema often bursts through the parietal pleura and burrows between the intercostal muscles.

In the same pleural sac there may be two or more empyemata shut off from each other. This is a point which

ought to be settled by digital exploration at the time of operation, because it is very difficult to determine in any other way. In very rare cases there may be two separate collections of fluid—one serous and the other purulent.

A condition of double empyema is sometimes met with, either as the result of a double pneumonia, or of the spread of infection from one pleura to the other. The second one will as a rule be easily recognized if attention is not devoted too exclusively to the condition of the chest on the side first affected. It is always advisable, when a child is under an anæsthetic at the operation, to examine carefully the sound side for signs of second empyema, and to use the exploring needle if necessary. Double empyemata are especially common in young subjects under the age of two years.

COURSE AND TERMINATION.—Even in the present day one not infrequently has the opportunity of seeing what the course of an untreated empyema tends to be as the difficulty of diagnosis is great in some cases, while in others the symptoms have been so moderate that advice is only sought when some later complications arise. It is possible that an untreated empyema may terminate in absorption of the pus and complete or partial restoration of the parts to normal. We have seen an empyema, from which half an ounce of thick pus had been withdrawn, left to itself; and although there was no doubt that much more pus was present, within six months there was a complete restoration of the parts to normal, so that no difference could be made out on the two sides of the chest. Such a termination is so rare that it is not to be considered as more than a lucky chance. An untreated empyema may lead to secondary infection of the whole system, with hectic temperature, sweating, and death from toxæmia. Again, the infection carried by the blood may lead to local lesions, such as purulent meningitis, cerebral abscess, endocarditis, subcutaneous abscesses, and purulent arthritis. Or the infection may be carried by the lymphatics, causing purulent pericarditis, or peritonitis, or an empyema of the other pleura. An empyema may burst into the lung, and the pus may reach the air-passages and thence be expectorated, often in considerable quantities. This is sometimes termed the "natural" cure of empyema, and complete recovery has sometimes followed. As a rule, however, there is only a partial evacuation, a sinus follows, the expectoration of pus goes on, and there is a marked tendency to the formation of abscesses in the lung. An empyema may penetrate the parietal pleura, and the pus pushes its way amongst the surrounding tissues to appear as a subcutaneous abscess on the chest wall. It is curious to find how frequently under this condition the pus will point externally in the region of the nipple of the affected side. The fluctuating swelling which indicates a pointing empyema does not necessarily show that the empyema lies immediately underneath. Frequently the rupture through the pleura has taken place at some distant point—e.g., about the spine—and the pus tracks along between the intercostal muscles before penetrating to the subcutaneous tissues. Again, the empyema may rupture through the diaphragm, starting a subphrenic abscess, or general peritonitis, or a psoas abscess, or it may pass deeply into the lumbar muscles, forming a lumbar abscess. Chronic abscesses in or about the lower part of the trunk should suggest the possibility of some latent empyema, and the chest should always be thoroughly examined in such cases.

DIAGNOSIS.—The physical signs do not enable one to distinguish between a serous and a purulent effusion in the pleura, and the matter can be settled only by the withdrawal of some fluid by an exploring needle. This test should never be omitted in early life, when the signs of fluid are present, because empyema is so common, and delay in carrying out the appropriate treatment may be disastrous. Enlargement of the superficial intercostal glands is much more common in the serous than in the purulent cases, but its absence is a sign of no importance (Miller). Although free air in the pleura (pneumothorax) may give rise to the displacements of various organs, seen in the case of liquid effusion, the physical signs on percussion are quite different in the former affection. Pyopneumothorax is a rare condition, and is usually secondary to pneumothorax.

Empyema is so commonly associated with lobar pneumonia that a rise of temperature soon after the crisis should always make one suspect and examine for this complication (metapneumonic empyema). Sometimes pus is formed at an

early stage of the pneumonic attack or shortly before the crisis (parapneumonic empyema), with the result that a critical fall of temperature does not take place. In many cases of empyema, and especially the pneumococcal cases, the temperature falls after a pyrexial stage of a week or ten days, so that the presence of a normal or subnormal temperature must not be regarded as excluding empyema when the physical signs of that disease are found. The detection of pus in the pleura when the pneumonic lung is still unresolved and solid may present considerable difficulty. Of the physical signs, most reliance is to be placed on the increasing dullness, amounting to flatness, which is never found on percussion over a pneumonic lung. In the presence of such flatness, even although the breathing may be bronchial or tubular, and many crepitations may be audible at the same area, one should not hesitate to explore. A similar flat note, not due to fluid, may be present in connection with a markedly thickened pleura, or a new growth, or a lobe of lung in a condition of solid tuberculous caseation.

The use of the exploring needle may give a negative result, even when fluid is present, from various causes. The needle may be too small to allow of pus passing through it, or the layer of fluid may be thin and the needle has been pushed through it into solid lung. A large needle should always be used in the case of children, and it should be introduced slowly after it has penetrated the skin, gentle suction by means of the syringe being maintained all the time. Again, the needle may have entered an adhesion, or a piece of thickened membrane, or a mass of solid pus. Repeated exploration should be carried out in the presence of definite physical signs, and if the pus cannot be localized in this way it is sometimes advisable to give a general anæsthetic and explore more extensively.

Aid in the diagnosis may be afforded by examination under the X-rays. In the case of free fluid in the pleura, one can see by means of the fluoroscope the limitation of movement of the diaphragm on the affected side, the displacement of various organs, and a shadow marking the situation of the fluid. In cases of localized empyema there may be a definite shadow in some part of the lung or along the spine, pointing to an empyema as suggested by the general symptoms, but not easily localized by ordinary physical examination.

Examination of the fluid removed throws some light on the nature of the empyema. Typical pneumococcal pus is thick, viscid, of a greenish-yellow colour, and without any tendency to divide into layers on standing. Typical streptococcal pus is thin and yellowish, and separates into two layers, the upper clear and abundant. This must be further confirmed in all cases by bacteriological examination. As regards the blood-count, empyemata are always associated with a marked leucocytosis, amounting from 30,000 to 60,000 white cells, chiefly of the polymuclear variety. In serous effusions the white cells are chiefly lymphocytes.

Various diseases of the lungs have to be differentiated in the diagnosis from empyema. Amongst these may be mentioned chronic interstitial pneumonia, unresolved lobar pneumonia, pulmonary tuberculosis with caseation of a whole lobe, tumour of the lung, abscess of the lung near the pleura, and massive collapse. Of non-pulmonary diseases, mention may be made of purulent pericarditis and subphrenic abscess. From all of these diseases the diagnosis of an empyema when the fluid lies free in the pleura, ought to be successfully made after a careful physical examination completed by exploratory puncture. When the fluid is loculated, the differential diagnosis may be extremely difficult. In the diagnosis of empyema much weight is to be attached to the flatness or dullness of the per-

cussion note over the area of fluid, to the displacement of various organs, and to exploratory puncture. On the other hand, the character of the breath sounds, the presence or absence of accompaniments, and the state of the temperature chart, will only mislead in many cases if too much reliance is placed on them.

PROGNOSIS.—The prognosis depends on the nature of the infection, the age of the child, the duration of the attack, the state of the patient when treatment is carried out, the presence or absence of serious complications, and the treatment adopted.

As already stated, a pneumococcal empyema is usually milder, and is more rapidly cured, than a streptococcal. During the first year of life the prognosis of empyemata is more serious than later on, and during the first six months of life it is always grave. When an empyema has lasted for some weeks, it always has a debilitating effect on the patient's general health. If the patient has been previously pulled down by some acute illness, his condition may render operative treatment a serious undertaking. Many complications may occur which render the prognosis very grave. These may be of a local character, such as tuberculous disease of the lungs, purulent pericarditis, or meningitis; or of a general nature, such as pyæmia, septicæmia, etc.

At the same time the general prognosis of empyema, considering that the affection in so many cases is pneumococcal in origin, may be said to be very good, provided that efficient treatment is carried out without undue delay.

TREATMENT.—The only satisfactory treatment of empyema is the evacuation of the pus by surgical measures. Expectant treatment may be followed by rupture through the lung or through the chest wall, with a more or less complete recovery, but is much more likely to terminate by the death of the patient. Treatment by aspiration has been tried, but has failed to give more than a small percentage of recoveries. Holt says that, out of 129 cases of empyema treated by aspiration, 25 were cured, 13 died, and 101 had to be subjected to operative treatment.

It is never possible to tell beforehand whether the pus is of such consistency as to be removable by aspiration, nor is it possible to tell for some time after aspiration whether the pus is re-accumulating or not. Nevertheless aspiration has its use as a temporary measure in certain cases. When the amount of pus is large, and the symptoms of distressed breathing or cyanosis are present, aspiration should be carried out at once, in order to avoid the risk of death from cardiac or respiratory failure. Also it is always advisable in the case of young children to aspirate the pleura the day before operation, so as to reduce as much as possible the shock which occurs as the result of the sudden withdrawal of a large quantity of fluid. If the amount of fluid is small, a matter often rather difficult to determine, this preliminary aspiration is not called for.

All are agreed that the proper treatment of an empyema is to open the abscess and drain it externally. Opinion is still divided as to whether incision of the pleura alone, or supplemented by resection of a rib, is preferable. In favour of simple incision, it is stated that this operation is usually successful, that the space between the ribs is sufficient for drainage and for any necessary examination of the pleura at the time of operation, and that the tube can usually be removed at the end of three to six weeks. In favour of resection, it is stated that by no other means can free access to the pleural cavity and free exit for the contents be secured, that it is the only treatment suitable for every variety of empyema, that in chronic cases the

tube may be discarded in three or four days and in acute cases in from seven to ten days, and that a complete cure is secured more easily and more rapidly than by any other method. At the same time the larger opening carries with it the risk of subsequent infection, unless strict asepsis be maintained. We cannot help thinking that the more thorough method of resection is not only to be preferred as suitable to all cases, but also because it gives the best results, while simple incision may be employed in urgent cases in which aspiration cannot be successfully employed as a temporary measure.

The time to operate is as soon as possible after pus has been discovered by the exploring needle. In chronic cases the risk of delay is that the abscess may burst through the lung or parietal pleura. In acute cases the danger of extension of the infection to the other pleura or the pericardium or more distant parts is always present. In some cases pus may not have been obtained, but the local and general signs of empyema may be so marked that the physician is justified in asking the surgeon to open the pleura. We have already referred to those cases in which the exploring needle may have passed through a thin layer of fluid, or into a thick mass of pus, with a negative result. Some prefer a local to a general anesthetic. It is quite true there are definite risks in this operation when a general anesthetic is used, but if the risks are recognized they are as a rule easily avoided. They are associated with the interference with normal respiration, but if the degree of anesthesia is not pushed, and if a sudden evacuation of the fluid is avoided, the dangers are not greater than in, for example, a case of adenoid hypertrophy in the naso-pharynx. On the other hand, a local anesthetic is most unsatisfactory in the case of young children; fright and shock are both most marked, and a hurried and unsatisfactory operation usually follows. The site of operation has been much discussed, but the best place to operate in pneumococcal cases is where pus has been obtained with the exploring needle. This will prevent the surgeon from opening the pleura at a place where the pus is not present. The question of ordinary drainage does not come in, because after an opening is made the fluid is evacuated by the pressure of the re-expanding lung, and not by gravitation. If the surgeon is guided by the anatomical relations of the pleura, and attempts to make an opening at the lowest part laterally and posteriorly, he will probably enter the peritoneal cavity or fail to find any pleural cavity, owing to the high displacement of the diaphragm which is so often present. In empyemata of a septic or virulent character, it is probably wiser to have two openings, one being at the most dependent part of the pleura, and a drainage-tube connecting the two.

The details of the operation must be left to the individual surgeon, but some points may be referred to. When masses of thick pus are present, these are best removed with the finger, and not left to break down later. A thorough exploration of the pleural cavity should be made with the finger, in order to break down any adhesions and feel for a second empyema. The breaking down of adhesions frees the lung, and allows of the re-expansion which is so necessary for a complete cure. Some have objected to the breaking down of adhesions because of the tendency for infection to spread to healthy pleura, but such does not seem to be the case. What is found often is that there are pockets of pus about the pleura limited by adhesions, and it is clear that these cannot be effectually drained unless all adhesions are first of all broken down. The examination for a second empyema and its evacuation may mean all the difference between a successful and an un-

successful operation. At a later stage, although one may suspect the presence of a second empyema, it is extremely difficult to make an exact diagnosis, or locate it, until the necropsy. The thorough removal of all pus by means of irrigation of the pleura with hot sterile water at the time of operation is distinctly advisable, and tends to shorten the period of drainage. It is regarded by some as a dangerous proceeding, but if a free exit is maintained, and the intrathoracic organs are not subjected to fluid pressure, no harm is likely to follow. Curiously enough, the fatal results from pleural irrigation seem to have occurred on occasions subsequent to the first operation, and probably intrapleural pressure was the cause. On opening an empyema, if the patient is breathing quietly, the pus escapes slowly with each expiration. If the patient coughs, the pleura is rapidly evacuated, and this sudden emptying may lead to cyanosis or a tendency to syncope. The sudden emptying should be prevented by placing something over the opening. Before the drainage-tube is inserted, the lung should have expanded up to the opening, and this can best be tested by making the patient cough. If the lung does not re-expand, it is either tied down by adhesions or partly solid, and the subsequent treatment will be more prolonged. The length of the drainage-tube should be such that the inner end just reaches the pleural cavity, and the tube should be a large one. A large drainage-tube passing deeply into the pleural cavity will probably lead to the formation of a sinus and delayed healing. If a free exit is provided, the pleura will be kept empty by the intrapulmonary pressure.

The treatment after operation is most important. In but too many cases it is held that the surgeon's duty ends with the operation, and that anyone can carry out the subsequent treatment. What too often happens is that after the second or third day the temperature rises, the patient is manifestly ill, and the purulent discharge increases in amount and often changes in character. This is due to a secondary infection of the wound and pleura owing to failure in maintaining the strict asepsis which was probably pursued all through the operation. If this strict asepsis were maintained from the time of operation until the pleural opening was closed, the results of operation for empyema would be very much better than they are. The duration of drainage varies greatly in different hands. Taking an ordinary pneumococcal case of empyema, we have found that in the acute stage an average period of from seven to ten days is quite sufficient, and in the chronic stage from three to four days. It is quite possible that in these subacute pneumococcal cases the surgeon will yet secure an even more speedy cure by opening and exploring the pleura, washing out, and then closing the wound without any drainage at all. In cases of streptococcal and staphylococcal infections, and where there is a caseating gland or other tuberculous focus directly associated with the empyema, the drainage will have to be more prolonged than in pneumococcal cases. The amount and nature of the discharge are the chief guides as to the duration of drainage. As soon as the discharge becomes serous the tube may be discarded. The mere presence of a tube in the pleura may be a source of irritation, causing coughing and some pyrexia, as shown by the disappearance of these symptoms when the tube is removed.

The re-expansion of the lung on the affected side must be encouraged in every way. This undoubtedly goes on while the patient is still in bed with the tube in the chest, but it proceeds much more rapidly when the opening is healed and the patient is out of bed. The deeper breathing of a child running about tends to the fuller expansion of the lungs and the chest wall. While these natural movements

are to be preferred, aid is also obtained by giving a child a trumpet to blow, or a special apparatus consisting of two bottles, one containing a coloured fluid, and both connected with rubber tubing. It is an amusement for the child to blow the fluid from one bottle to the other. These artificial aids, however, must not be used to excess, as they tend to induce emphysema rather than the proper expansion of the lungs. In the pneumococcal cases with a short period of drainage one cannot but be struck by the rapid recovery of normal movements on the affected side, without any special respiratory exercises. On the other hand, if drainage has been prolonged it may be necessary to carry out chest-expanding exercises for some time.

In cases of *double empyema*—i.e., when both pleurae are involved at the same time—the question arises as to whether both should be opened and drained at the same time. Experience has shown that it is quite possible for respiration to be carried on with a tube in each pleural cavity, and this is probably due to the fact that the fluid in one or other case is localized. Still, it would seem advisable after opening and draining one cavity to aspirate the other, and when the first one is healed, or nearly so, to proceed to the more radical treatment of the other. A condition of *foetid empyema*, whether the infection has come from within or from without, is best treated by making two openings, and establishing thorough drainage by means of large tubes. The whole cavity should be well irrigated. In the case of a *pointing empyema*, it is necessary not only to evacuate the pus externally, but to trace its source to the original empyema and to drain directly over it. In the case of an empyema bursting through the lung, it is not advisable to trust to the occurrence of a "natural cure," but the empyema should be located and drained.

Cases of *tuberculous empyema* are very unsatisfactory as regards treatment. The case may have appeared to be an ordinary empyema, and it is only later to secure a cure that causes suspicion of the underlying cause. A history of symptoms pointing to pulmonary or glandular disease for some time before the empyema developed may help one. Definite signs of pulmonary disease, such as riles, coughing, wasting, and fever, may be present, and are of value. As in such cases an empyema is only a complication of the tuberculous lesion, the latter must be treated as efficiently as possible.

The treatment of an old-standing empyema with a chronic sinus gives very unsatisfactory results. Decortication of the lung has been tried, and has not proved effectual. Estlander's operation by removal of pieces of many ribs, to allow of complete retraction of the lung and collapse of the chest wall, produces such distortion of the trunk as the child grows that it can hardly be recommended. So far as the chronic sinus and discharge are concerned, relief may be obtained by means of an autogenous vaccine in some cases, and an effort should always be made to isolate the special organism and make a vaccine. If the discharge can be cured, the collapsibility of the chest wall in childhood will usually permit of the closing of the cavity, even although the lung is so bound down as to be incapable of expansion.

PNEUMOTHORAX.

Pneumothorax is a rare condition in childhood, and the majority of the cases in which it occurs as a complication of pulmonary disease are not recognized until after death. If no disease of the lung is present, it is due to some form of *trau-*

matism—*e.g.*, an injury to the chest, with or without fracture of a rib, paracentesis thoracis, where the lung has been punctured by the exploring needle, or tracheotomy. The association with tracheotomy seems to arise from the passage of air into the cellular tissues of the mediastinum, and then the lung, followed by a rupture of the visceral pleura.

The common form of pneumothorax in adult life—that associated with chronic pulmonary tuberculosis—may occur in the later years of childhood and under similar conditions. A girl of eleven years was admitted to hospital with definite signs of active phthisis pulmonum in the upper part of the right lung. Some days later she was seized with pain in the right chest, and the breathing became very laboured. The typical signs of pneumothorax were present on the right side, and the heart was much displaced to the left. She was kept under the influence of morphia, and, as the dyspnoea continued, the right pleural cavity was punctured, and a considerable amount of air escaped, evidently under pressure. For some time signs of air and fluid were present in the right pleura, but they gradually cleared off, and the heart swung back to its normal position. The pulmonary disease continued to advance, and the patient was discharged.

The more characteristic form of pneumothorax in early life is that associated with softening and breaking down of the lung, in the acute forms of pneumonia, most commonly of a tuberculous nature. This accident would be much more common were it not for the fact that firm pleural adhesions and thickening are usually present in cases of tuberculous pneumonia. As a rule there are no acute symptoms to mark the occurrence of pneumothorax, and it is easily overlooked on physical examination, owing to the variety of other pulmonary signs present. Pain is seldom complained of. Dyspnoea is probably already marked owing to the pulmonary disease, and is only increased in those rare cases in which the pneumothorax fills the whole or a large part of the pleural cavity.

A child of two and a half years, suffering from pneumonia of the right lung, was found during the course of the illness to have an area of tympanitic resonance over the lower third of the right lung anteriorly and laterally, with cavernous breathing and bell sounds, but no riles. There was no cardiac displacement. The condition persisted until death a week later, when pus and air were found in the pleura at this region. The pyopneumothorax was shut off by adhesions, and the lung was riddled with small abscesses. Part of the visceral pleura was ulcerated through. The lungs and bronchial glands were tuberculous.

Alice E. Sanderson has recorded a case of pneumothorax complicating broncho-pneumonia in a child of two years. Signs of pneumothorax developed in the right axilla. The right side of the thorax was bulging, and there was a tympanitic note over the lower part of the lung, with shifting dullness. Cavernous breathing and the bell sound were present. The heart was displaced to the left, and the liver was pushed downwards. At the necropsy pneumothorax of the right side was found, the lung being small, collapsed, and airless. The pneumonia was of pneumococcal origin, and small areas of the lung tissue were beginning to break down, but there was no evidence anywhere of tubercle. Some greenish-yellow pus was in the pleural cavity.

Pneumothorax may occur in connection with general diseases, such as enteric and scarlet fever and hooping-cough, but there is always present some local disease of the lung. The pulmonary affections leading to destruction of tissue may at any time be complicated by pneumothorax when the area of softening

involves the pleura. Amongst these the commonest are tuberculosis, pneumonia, abscess of the lung, and infarct of the lung. In the case of young children the onset is not usually accompanied by any marked symptoms. The pneumothorax is much more frequently of limited extent and surrounded by pleural adhesions, so that marked displacement of surrounding organs does not occur. Pyopneumothorax follows as a rule, and a serous effusion is rare. The diagnosis rests on the same physical signs as in the case of the same disease in adult life. The prognosis depends primarily on the underlying disease, but as a rule pneumothorax occurs in the course of an otherwise fatal illness. There is little to be done in the way of treatment in cases associated with tuberculosis or abscess formation; in young children, because of the acute underlying disease. In a case of bronchopneumonia, a limited pneumothorax may heal spontaneously, or it may be amenable to surgical treatment when pus accumulates. Traumatic pneumothorax often terminates in spontaneous recovery, when the chest is fixed with a supporting bandage and the patient is kept at rest under the influence of opium. If the accumulation of air in the pleural cavity is causing marked pressure and dyspnea, it should be relieved by puncturing or aspirating the pleura.

HEMOTHORAX.

While blood may be effused into the pleura in connection with any of the hemorrhagic diseases of infancy and childhood, it is not usually present in such quantity as to be recognizable or of any special importance. Hemorrhagic pleurisy is rare, but it has been occasionally found in connection with tuberculosis and rheumatic fever. If at the second exploration a serous effusion is found to be hemorrhagic, this is probably due to bleeding following the first puncture. The malignant growths of the lung and pleura in childhood do not usually lead to hemothorax.

CHYLOTHORAX.

The condition of chylothorax is very rarely found in early life. It is due to a rupture of the thoracic duct and the passage of the contents into the pleural cavity. This may follow direct traumatism, or, as in a case recorded by Merton, it may be due to pressure of enlarged bronchial glands on the thoracic duct. The term "chyliform fluid" has been applied to any exudation in the pleura with a milky appearance, and this has been found to be associated with the presence of fat, leucin, milk-albumin, or cholesterol, singly or in combination. The underlying disease in the majority of cases has been pulmonary tuberculosis.

DISEASES OF THE TRACHEA AND BRONCHI.

BRONCHITIS.

Acute Bronchitis.—Etiology.—Bronchitis, both in the acute and the chronic form, is a very common affection in early life. There are various factors which contribute to render young children prone to this disease. In many cases it is an extension of the catarrhal process known as a "common cold," which, beginning

in the nasal passages, spreads downwards to the bronchi. The various throat affections, acute and chronic, are often accompanied by bronchitis, partly as a result of the direct spread of infection, and partly from the mouth-breathing which is so often present. Most of the specific fevers of early life are marked by a tendency to catarrh of the bronchial tubes, which is almost invariably present in measles and hooping-cough. It is not yet known whether the bronchitis in these cases is of a specific nature—i.e., directly due to the organism causing the disease; or whether, as in the case of the ordinary forms, it is due to the organisms which hang about the mouth and nose—e.g., streptococci, staphylococci, and pneumococci. Probably, in the case of influenza, both the general disease and the bronchitis are due to Pfeiffer's bacillus. The liability to bronchitis is much greater in winter than in summer, and this must be ascribed to cold winds, damp weather, fogs, and the other accompaniments of the cold season. Delicate children often manifest their delicacy by an undue susceptibility to bronchitis. Certain other affections predispose their subjects to bronchitis. Amongst these rickets takes a foremost place, and one of the commonest indications of a rachitic state is a tendency to catarrh of the bronchial tubes. A child which is described by the mother as "always cutting its teeth with bronchitis" will usually be found to be rickety. The delicacy which accompanies tuberculosis or a predisposition to that disease will often be manifested by a tendency to bronchial catarrh. Defective circulation in the lungs will also predispose a patient to bronchitis from slight causes. This is seen in cases of heart disease, of fibroid lung, and in various forms of dropsy. Any cause which leads to defective aeration of the blood will act in a similar way. The pulmonary circulation becomes engorged, the bronchi become oedematous and swollen, and bronchitis quickly follows. It is probable that this factor plays a part in rendering fat babies more liable to bronchitis. No doubt rickets plays a part in many of these cases, but there are others in which this does not apply. It is evident that in a very fat child the capillary circulation through masses of oily tissue is considerably hampered, and that the circulation of the blood through the lungs is thereby weakened. The upbringing of children in hot stuffy rooms favours a condition of delicacy which often manifests itself as bronchitis. Amongst the poor, the badly-ventilated dwelling-rooms with vitiated atmosphere render the children liable to chills and bronchitis when they go out of doors. Such chills are also frequently induced by improper clothing—either too much, causing sweating, or too little. Inadequate clothing about the abdomen or the lower extremities during cold weather is a common cause of attacks of bronchial catarrh.

A distinction must be drawn between primary and secondary cases of bronchitis. In the primary cases are included those in which an apparently healthy child is attacked with bronchitis as the result of a chill or exposure to an inclement atmosphere. Amongst the secondary class are included those in which the bronchitis is the result of a spread of infection from adjoining regions (e.g., the nose and throat); those in which it is part of a general infection (e.g., influenza); and those in which some disease of a debilitating character is present (such as morbus cordis, rickets, etc.). Taking all the causes of bronchitis together, we shall probably find that in childhood measles and hooping-cough are the most frequent. Further, this form of bronchitis is as a rule specially prolonged and specially severe.

SYMPTOMATOLOGY.—In dealing with the symptoms of bronchitis, it is important to bear in mind the two chief factors which produce discomfort—namely, *obstruction* and *spasm* in the bronchial tubes. The patient may be distressed and anxious

and sleepless, but these symptoms are not caused by pain or high fever, but by embarrassment of the respiration through the bronchial tubes. Bronchitis is, in short, one of the forms of obstructed breathing. The lumen of the bronchi is diminished by the swelling and softening of the wall, and by the catarrhal exudation which is poured into it. If sputum is superadded, the lumen is still further diminished, and this condition may be expected when paroxysms of dyspnea occur. During expiration the collapse of the lung is apt still further to diminish the lumen of the bronchi, the walls of which have to a large extent lost their rigidity through softening, and have become much more compressible.

The symptoms vary according to the extent to which obstruction to the breathing is present. Even in a mild case the normal abdominal (diaphragmatic) breathing will be reinforced by costal breathing. In more marked cases there will be definitely exaggerated movement of the chest during inspiration and expiration, both of which are prolonged. With increasing obstruction comes an increase in the signs of dyspnea, the anxious expression, the tendency to cyanosis, the movements of the alæ nasi and larynx, and the over-expansion of the chest. These are the more serious symptoms, all of them traceable to bronchial obstruction, which may develop in this affection, but which, fortunately, are seldom reached except in the case of very debilitated infants whose breathing powers are easily exhausted. Probably in all cases of bronchitis the trachea is involved, so that the disease would more properly be termed "tracheo-bronchitis." Owing to the larger size of the trachea, the obstructive signs of tracheitis are not so obstructive as those of bronchitis.

In an attack of acute bronchitis in a child the temperature is usually raised, but not to the same extent as in pneumonia. An ordinary range of pyrexia is from 100° to 102° F., and if the patient is under treatment this will usually subside in a few days. In young infants the pyrexia may be greater owing to the marked instability of their heat centres. The pulse-rate is increased to 100 or upwards, but is not usually disturbed to an extent which is disproportionate to the state of the temperature. The breathing is not markedly quickened; in the early stages, when the respiratory obstruction is not marked, it may reach a rate of 30 to 35 per minute, due largely to the fever; but the nature of the obstruction when marked will tend to induce rather slow and deep respiration. Pain may be present, referred to the sternal region, and is dependent on the associated tracheitis. When the patient is at rest and breathing quietly, the pain is not usually severe, but it is always aggravated by any strain, such as that of coughing. Cough is usually frequent and troublesome. In the early or "dry" stage of the attack it is irritative and hacking, and is caused by the inflamed state of the bronchi, while in the later or "moist" stage it is loose and rattling, and is caused by an effort to clear the tubes of mucus and catarrhal products.

The physical signs of bronchitis are the same at all ages. The chest note on percussion is not usually altered, although as the result of prolonged coughing a certain degree of hyper-resonance may be present owing to the production of emphysema. On auscultation, during the early stage, there may be various dry sounds present, described as "snoring," "whistling," etc., which partly control the nature of the respiration. The breath sounds are harsh during both inspiration and expiration, and the latter is specially prolonged, a condition of breathing known as "exaggerated puerile." After one or two days the dry stage passes off, to be replaced by the moist stage, in which secretion from the bronchi becomes more

abundant, as shown by the presence of numerous bubbling rales heard on auscultation. As a rule these are distributed over both lungs, and in an ordinary attack are produced chiefly in the larger tubes. The phlegm is easily coughed up, but, except in the case of older children, is not available for examination, as it is swallowed. The severity of the attack is largely to be measured by the extent to which the catarrhal inflammation penetrates to the smaller tubes, and the nature of the rales heard are an indication as to this. In the smaller divisions of the bronchi—the bronchioli—the rales are of a finer quality and more like the crepitations of pneumonia. When the smaller tubes are involved, the dyspnoea becomes greater, and there may be retraction of the subcostal region during inspiration, indicating a great degree of obstruction. This is, in fact, the borderland between bronchitis and capillary bronchitis or broncho-pneumonia, and in some cases it is impossible to say when one ends and the other begins. The disease takes on a much more serious aspect, the circulation through the lungs is much more seriously interfered with, and clinically the condition is to be regarded and treated as broncho-pneumonia (*q.v.*). At the same time, an attack of bronchitis only may prove sufficiently grave in weak children, in infants exhausted by previous disease or malnutrition, and more especially in those with defective expansion of the chest walls. The rachitic infant with big abdomen and small chest, and with the lower part of the chest sinking in at every inspiration, is a specially bad subject. The lungs are never properly expanded, the bronchial tubes are rapidly filled with secretion, the cough is feeble and useless, and the accessory muscles of respiration are too weak to be of any service. Air cannot be got into the lungs, and mucus cannot be got out; the circulation through the lungs becomes blocked; cyanosis and cardiac failure follow. From the blocking of the bronchial tubes areas of lung tissue are cut off and collapse. These conditions may render an acute attack of bronchitis a most serious illness, but, as before stated, they are not to be looked for except in the case of debilitated children.

An ordinary attack of acute bronchitis in a healthy child will run a favourable course in about a week to ten days, provided that the child is properly nursed. In weak children it will last longer, and may run into a chronic condition or into one of broncho-pneumonia. During an attack slight patches of pulmonary collapse may appear, due to blocking of a bronchial tube, but as a rule the collapse is evanescent if the coughing power is good. In the case, however, of a weak infant the expulsive powers are often so much diminished that it is impossible to cough up the plugs of inspissated mucus, and these areas of collapse persist, as shown by the impaired resonance and diminished breath sounds over them. As these areas increase in number and size, cyanosis and dyspnoea tend to become more marked. As a rule there are no special complications with an attack of acute primary bronchitis, but in the secondary forms the presence of concurrent disease may render the bronchitic condition much more serious.

DIAGNOSIS.—As a rule the definite clinical features and the short course of an attack render the diagnosis easy. In the case of the tracheo-bronchitis of influenza the symptoms may persist for some weeks, and the cough assume a paroxysmal and explosive character very suggestive of whooping-cough. In debilitated children the diagnosis between bronchitis and broncho-pneumonia may be difficult, and the latter may usually be assumed to be present when the disease takes the form of capillary bronchitis—*i.e.*, involvement of the bronchiolides. Cases of generalized tuberculosis may be characterized by signs of bronchial catarrh and no

other localizing symptoms. Here the progressive weakness of the child, the dyspnoea and cyanosis, and the pyrexia out of proportion to the physical signs, should suggest tuberculosis rather than bronchitis.

TREATMENT.—The treatment of an attack of acute bronchitis has changed very considerably in recent years. Some fifteen years ago the patient would have been shut up in a hot, stuffy room, his chest would have been poulticed day and night, and a steam-kettle in constant use would have rendered the atmosphere almost unbearable. Not infrequently, by the help of a good constitution, the patient struggled through, but as a rule the illness was more prolonged and the sufferer was more weakened than was at all necessary.

The days of hot, close rooms in the treatment of any pulmonary affection are over—for the present, at least. A well-ventilated and large room is selected, as high up in the house as possible so as to get the purest air, and the invalid's bed is placed in the middle of the room. Any screens used are only for the purpose of warding off draughts, not to keep away fresh air. The temperature of the room is maintained as steadily as possible at from 60° to 62° F. Some advise a higher temperature—65° to 70° F.—but there is no risk about the former, and it is a much more stimulating atmosphere. A steady temperature can only be maintained with accuracy by means of an electric or gas stove. The chief drawback to the use of a gas stove is the risk of an escape of gas about the tap, but this ought to be detected at once by an intelligent nurse. When a coal fire is used night and day, there is a considerable amount of smoke and coal dust always flying about the room, which not only vitiates the atmosphere, but directly irritates the bronchial tubes. As Still has pointed out, a smoky room may be enough to provoke an attack of bronchitis in a susceptible child, and much more will it tend to aggravate an already existing condition of bronchitis. The ventilation of the room must be attended to, and the best means is through open windows, day and night. At the same time, in foggy or very wet weather an atmosphere would thus be introduced which is by no means beneficial to the patient, and ventilation is then better carried out through the door.

The bedclothes should be warm, but not too heavy; and while the skin heat should always be maintained by a sufficiency of bed and body clothing and by hot bottles, there should be no overheating of the body, leading to sweating. A long flannel nightdress is desirable, with sleeves fastening at the wrist, so as to protect the arms. It is difficult to get young children to keep their arms under the bedclothes when they are awake. In summer weather a flannel undershirt is usually worn, and in winter thicker flannel or a cotton-wool jacket. The chest and abdomen are not constricted in any way, as free play is required for the abdominal and thoracic muscles of respiration.

During the acute stage a fluid diet is indicated for two reasons—first, because of the impairment of the digestive functions, and, secondly, because of the loss of appetite. The food should also be moderate in amount, and given at regular intervals every two or three hours by day. Beef-tea, mutton or veal soup, milk and barley-water, are sufficient at this stage. In addition, sips or drinks of hot barley-water or rice-water or lemonade may be given freely, as hot fluids are probably the most powerful expectorants we possess.

Certain local measures are useful in securing relief to the breathing during the acute stage. These should be carried out with as little disturbance of the patient as possible, for rest is most essential, and all treatment during the acute stage should

be carried out with the patient lying in bed. At the onset of bronchitis, relief will often be afforded by the use of the steam-kettle. The indications for this are two in number: first, the dry stage of bronchitis, in which secretion is defective; and, secondly, the occurrence of spasm in the bronchial tubes. In the first case the use of a hot, moist atmosphere will induce secretion, and in the second case it will relieve the spasm of the bronchial tubes, which is so easily excited in young children. A steam-kettle may be used for this purpose, or in the case of older children direct inhalation from a special inhaler may be carried out. Plain hot water is most commonly used, but in some cases a more powerful antispasmodic effect will be produced by the addition of compound tincture of benzoin, or creosote, or oil of eucalyptus, $\frac{1}{2}$ to 1 drachm being added to the pint of water. Continuous steaming is not advisable, because of the depressing effect on the patient, but the intermittent use of steam for from fifteen minutes to half an hour, at intervals regulated by the indications present, will accomplish all that is desired. After free secretion in the bronchial tubes has been established, no further benefit will be derived from steaming, and harm may be done by producing excessive secretion.

Under the same conditions—namely, spasm and defective secretion—poultices or hot fomentations may give relief. Here, again, the intermittent use of these applications will do good, but their constant use is both very disturbing to the patient and embarrassing to the respiration. Fomentations are to be preferred as being lighter, more easily made, and more likely to be applied hot. Amongst the poorer classes it is common to see scars of former scalds on the back or front of the chest from the application of poultices which were too hot. But the opposite mistake of applying them too cold is much more common. In ordinary practice fomentations are more likely to be properly made and properly applied, and may be kept on for an hour on the front and back of the chest. If a more speedy effect is desired, the addition of one or two drachms of turpentine to the fomentation will secure this, the application being removed when the skin is thoroughly reddened or painful. Encasing the chest in any adhesive substance, medicated or otherwise, is not advisable in the case of children, as it limits free movement during respiration and prevents a proper examination being made as to the condition of the lungs. When the attack is subsiding, a liniment containing turpentine and belladonna may be rubbed into the chest twice a day with good effect.

As regards the use of medicines, it is very desirable to have a clear idea what the object aimed at is, and to be assured that the medicines employed will attain that object. The disease in the vast majority of cases is due to microbial infection, and runs a limited course, under favourable conditions, of from a week to ten days. The ideal treatment therefore would be to attack the microbe by a serum or vaccine, but such treatment is not at present available. As we cannot attack the disease at its source, the next best thing is to increase as far as possible the powers of resistance in the patient, and this is secured by rest, warmth, fresh air, and careful feeding. All that remains in the way of medicinal treatment is the relief of symptoms. The use of expectorant medicines in bronchitis is hallored by universal and prolonged custom. At the same time it is questionable whether the flooding of the system with drugs of the nature of ipecacuanha, antimony, carbonate of ammonia, and squill, is directly beneficial either to the patient generally or to the bronchial tubes. While the effect of these remedies on secretion from the bronchi is, to say the least, very problematical, what is certain is that they irritate the stomach and impair the appetite. While it is probable that a mixture contain-

ing small doses of *Lipicarmtha*, citrate of potash, and syrup of Tolu, will do no harm and will meet with the approval of the senior members of the family, it is not advisable to employ large or frequent doses of those so-called expectorants. Far less is it desirable to encourage the use of stereotyped "cough mixtures" for bronchitis, which may contain in equal proportion drugs to increase secretion and drugs to diminish secretion, and are recommended for use at all stages of the disease. As already stated, an abundance of hot fluids to drink, given in small quantities at frequent intervals, is the best means of encouraging free secretion, and also of producing a watery secretion which will not clog the bronchial tubes. This effect may be increased, if necessary, in the dry stage by the employment of iodide of potassium, which is well tolerated by children, and of citrate of potash. In the moist stage, if secretion has become embarrassing by its excessive amount, relief will be afforded by the use of tincture of belladonna in an acid mixture. In the acute inflammatory stage a dose of calomel or grey powder may be given with advantage every other night. Flatulent distension of the abdomen and constipation are to be avoided, as they tend to interfere with the free movement of the diaphragm, and thus increase the respiratory distress.

Coughing is not usually so severe as to interfere with sleep, but, if local measures fail to relieve it, a dose of paregoric may be given at bedtime. Opium, however, should not be pushed to such an extent as to check the expiratory coughing which is necessary for the clearing of the bronchial tubes. The use of emetics has been dropped within recent years; they were not required in moderate attacks, and in severe cases they were depressing. Stimulation may be necessary, and probably atropine and strychnine, given by the mouth or hypodermically, are the most useful drugs. Alcohol is believed by many to increase congestion and secretion of the bronchial tubes, which is often undesirable in severe cases, so that it should be used only in small doses. It should not be forgotten that the stimulating effect of a hot-puck is often very marked, and that after its use relief to the breathing and quiet sleep quickly follow. The treatment of severe cases of bronchitis is the same as that of broncho-pneumonia, and will be considered more fully in that connection (p. 358).

After the acute symptoms have subsided, the chest usually clears rapidly, but it takes some weeks before the bronchial tubes are again in a healthy condition. In winter the patient may be allowed to move about the house, but should not be out of doors too soon. In summer fresh air and exercise out of doors will probably expedite the healing process. If the bronchial catarrh persists, a change to the seaside or to some inland health resort will often prove beneficial. When the appetite is restored, a nourishing diet should be given. In bronchial affections fatty foods seem to be specially advantageous, and cream and cod-liver-oil may be ordered. Iron will probably impair the digestion, and is of no special value. The persistence of bronchial catarrh after an acute attack should always lead one to suspect the presence of pulmonary or glandular tuberculosis, but it may be dependent on some persistent trouble in the upper respiratory passages.

Chronic Bronchitis.—This is a common affection in early life. It is frequently the result of an attack of acute bronchitis, which has left the bronchi in a weakened condition and rendered them susceptible to fresh attacks of bronchitis from what is often only a trifling disturbance. In some cases it will be found to be due to a lowered condition of the health generally, but in others it is dependent on some local or general disease or disorder, which must be discovered and treated. It

other words, it is advisable to regard chronic bronchitis as a secondary form of disease, and to look elsewhere for the primary source of trouble. Even when the bronchi are themselves free from active trouble, as judged by physical signs, the disease is kept up by a certain degree of tracheitis.

Ætiology.—One of the most common causative factors is obstruction in the upper respiratory passages from the presence of enlarged tonsils or adenoid hypertrophy. The habit of mouth-breathing which results leads to the direct introduction of cold air into the trachea, and congestion follows. When nasal breathing is unobstructed, the warm air which enters the trachea is filtered and is practically free from organisms. In the case of mouth-breathers, numerous organisms are carried directly into the trachea from the outside, and others are picked up as the air passes the tonsils and pharynx, which are in a diseased state. The two factors, chill and infection, are therefore constantly present in cases of naso-pharyngeal obstruction. The tendency to bronchial catarrh which follows will be most marked during the cold season, while even in summer there is often a loose rattling cough at intervals. It is necessary in all cases of chronic bronchitis to examine for chronic rhinitis, adenoid hypertrophy, and enlarged tonsils, because until the mouth-breathing which accompanies these disorders has been cured it is futile to hope to secure permanent relief from the bronchial catarrh.

Amongst constitutional diseases which lead up to chronic bronchitis are rickets and tuberculosis, and here again the general disease must be treated. After some of the specific fevers, more especially measles and whooping-cough, a chronic form of bronchitis may follow, which often and very naturally raises a suspicion of tuberculosis. Some of these cases are undoubtedly tuberculous, as shown by the accompanying signs. At the same time, the persistence of the bronchial catarrh may be only the result of the specific infection acting on a somewhat debilitated system.

The home surroundings are often of such a character as to predispose directly to bronchitis. In the homes of the poor, the children frequently breathe a close, hot atmosphere, all ventilation being cut off, and the whole family breathing again and again the same air in a highly contaminated state. In the homes of the well-to-do, children are kept in stuffy, overheated dayrooms and bedrooms, and are often overloaded with clothes, so that they are strongly predisposed to recurrent chills. With each attack of bronchitis the precautions may be increased, until the child becomes a regular hothouse plant, unable to withstand any change of temperature. The obvious cure for this is to regulate the temperature of the dwelling-rooms, to fill them with fresh, pure air, and to have a sufficiency of clothing for warmth, but not for overheating. An excessive amount of food, especially of the carbohydrate variety, will also tend to induce a chronic bronchial catarrh. Overfeeding of this kind acts in two ways: In the first place the blood is surcharged with food materials, the pulmonary circulation is overtaxed in getting rid of these, and venous congestion and bronchial catarrh follow. In the second place there is an accumulation of fat throughout the body, which mechanically impedes the circulation and tends to induce pulmonary venous congestion. Fat babies who suffer from chronic bronchitis should have their diet rigidly restricted.

SYMPTOMATOLOGY.—The leading symptom for which advice is usually sought in cases of chronic bronchitis is cough. The child is never free from a cough, which comes on irregularly during the day or night, and is usually worst in the

moaning. It may be dry and irritative or loose and rattling, according to the amount of secretion present in the bronchial tubes. Sometimes the cough is loose and ineffective, apparently sufficient only to displace the mucous secretion, but not to bring it into the pharynx. Such a cough is very suggestive of nasal obstruction and tracheitis. Shortness of breath is not as a rule present, except on exertion, when the breathing soon becomes laboured and coughing is induced. There is no pain or fever, the child can eat and sleep well, and as a rule does not suffer as regards the general health, except in prolonged cases. On auscultation one can hear the ordinary sounds of bronchial catarrh, dry or moist, with those of the rarer variety predominating, the catarrh not usually extending beyond the larger bronchi. In prolonged cases, if the cough has been severe, there may be a condition of chronic emphysema. The chief danger to be feared in protracted cases is the development of peribronchitis, at first an active condition of cellular exudation, which is replaced later by fibrous tissue. This form of inflammation spreading through the lungs may lead to hardening and contraction of the fibrous tissue and dilatation of the bronchial tubes, a condition known as "pulmonary fibrosis" or "cirrhosis of the lung."

TREATMENT.—The main point about the treatment is that the underlying cause of the trouble should be ascertained and removed. Many of the conditions leading up to chronic bronchitis are remediable, and with their removal the bronchitis will quickly pass off. Others, such as heart disease, are not curable to the same extent, but must be treated in order to secure relief from the bronchitic trouble. Some children will be benefited by a residence for some months in a warm, dry climate, and for others a bracing mountain air will be advantageous. Cases of moist catarrh are most suitable for the former class, and those of dry catarrh for the latter. Under all the conditions, cod-liver-oil with malt or hyperphosphites is beneficial, and a stimulating liniment may be applied to the chest.

FIBRINOUS BRONCHITIS.

SYNONYM.—Membranous or plastic bronchitis.

The expectoration of casts of the bronchi may occur in various diseases. Of these the commonest is diphtheria, in which the characteristic membrane may extend to the bronchi, and under favourable conditions may be coughed up. Again, in acute lobar pneumonia the fibrinous exudation may fill the bronchi in the area affected, and may be expelled in the form of casts. A case of this nature has been recorded by D. J. M. Miller (1907). The patient was a girl of four years, who during an attack of pneumonia was seized with great difficulty in breathing, and after a violent paroxysm of coughing, accompanied by cyanosis, expectorated about $\frac{1}{2}$ ounce of bright red blood and two large irregularly cylindrical pinkish-white masses. These masses were solid casts, possessing at irregular intervals twig-like projections of smaller calibres, and composed almost entirely of red blood-corpuscles. Such conditions are to be distinguished from real fibrinous bronchitis.

ACUTE FORM.—True fibrinous bronchitis may occur in an acute or chronic form. The acute form is ushered in with fever, severe coughing, dyspnoea, and the signs of acute bronchitis. It is probably due to bacterial infection of unknown character. The dyspnoea and cyanosis may become very urgent, quite out of proportion to the physical signs present. The distress in breathing and the signs

of obstruction are much greater than in ordinary acute bronchitis. The expectoration of one or more casts will throw light on the diagnosis, which at best can only be a matter of conjecture until casts appear. The casts are often in the form of balls or plugs, which on being floated out are found to consist of hollow branching tubes, varying in size according to the part of the bronchial tube they have come from. They are composed of fibrin, epithelial cells, and leucocytes. With the expulsion of the casts marked relief to the breathing follows, but fresh ones are apt to form and to lead to a most distressing form of death from asphyxiation. Fortunately, the disease is a very rare one.

CANCEROUS FORM.—The chronic form of fibrinous bronchitis is characterized by recurrent attacks of a similar nature to those of the acute form, but without constitutional symptoms. The coughing and dyspnoea may come on suddenly and severely, but relief usually follows the expectoration of the casts. An interval of months or years may elapse before another seizure occurs. The casts are composed of mucus chiefly, and may contain Curschmann's spirals, Charcot-Leyden crystals, and eosinophile cells. The subjects of this affection are usually neurotic; a history of asthma is often present in the patient or family, and the whole condition suggests some secretory nervousness, comparable to that which, when affecting the large bowel, is known as "mucous colitis." The signs are those of chronic bronchitis and asthma, which is the usual diagnosis until casts are expectorated. In some cases, from blocking of the bronchial tubes, large areas of collapse of the lung may be present, with absent breath sounds. These usually clear up rapidly when the cast is coughed up. The rapid onset and the nature of the symptoms may sometimes suggest the presence of a foreign body in a bronchus—e.g., a pea or a button—which should be carefully inquired into. The prognosis in chronic fibrinous bronchitis is good, but, as already stated, relapses are apt to occur.

TREATMENT.—In the acute form attention should be directed to the loosening of the casts in the bronchial tubes. Inhalation of steam, impregnated with alkalis (lime-water, citrate of soda), may be employed. Further benefit in the same direction may follow the use of an emetic, which tends to induce free bronchial secretion, in addition to the straining, which may dislodge a cast. Potassium iodide may be given internally along with hypodermic injections of pilocarpine. As it is possible that many cases of so-called "fibrinous bronchitis" in the past were really cases of diphtheria, and as the symptoms are very urgent, it would seem reasonable to try at once a full dose of diphtheria antitoxin, 4,000 to 8,000 units, which will do no harm and may do much good.

In the chronic form the same treatment may be carried out during an attack, while in the intervals the measures adopted for the relief of chronic asthma will prove the most useful.

ASTHMA.

INTRODUCTION.—True spasmodic asthma is not a common disease in early life, but it may occur at any age. Ashby met with a case at the age of eight weeks, in which the disease was distinctly familial, the father and an uncle of the patient having suffered from the same complaint. I have met with one case which appears to have been true asthma, and which terminated fatally from exhaustion at the age of eleven weeks. As a rule the tendency to this affection does not manifest itself until after the age of two years. The term "asthma"

is met with much more frequently in connection with children than the disease itself, and the diagnosis is often made in the family circle. Certain conditions have to be distinguished from true asthma.

In children, spasm of the bronchial tubes is easily produced by irritation from within, from without, and from reflex action. Especially does this apply during the first three or four years of life. In connection with bronchitis and bronchiopneumonia there is considerable irritation of the bronchial tubes from the contained secretions, and spasm may follow with cough and dyspnoea. Hence readily follows a diagnosis of bronchitis and asthma. In such cases it is advisable to inquire as to whether the bronchitis or asthma came first. In genuine bronchitis the spasmodic symptoms follow the bronchial catarrh, while in genuine asthma the bronchitic symptoms follow the spasmodic attack. A spasmodic element in bronchitis, simulating asthma, is to be recognized, and may require special treatment, as already described (see Bronchitis), but is not to be regarded as true asthma. Irritation from outside the bronchial tubes is illustrated by the pressure of enlarged glands at the root of the lungs, which may produce cough, dyspnoea, and cyanosis. Attention to the nature of the symptoms and the accompanying physical signs will enable one to distinguish between this and true asthma (see p. 303).

Reflex irritation may also induce spasm of the bronchial tubes. Perhaps the most common example of this is the spasm that results from the presence of adenoid growths in the naso-pharynx. Undoubtedly, the removal of these will in some cases cure the tendency to spasm of the bronchi. It has accordingly been claimed that the removal of adenoid growths will cure asthma, but this does not follow unless every case of spasm of the bronchial tubes is to be described as due to asthma. The recognition of this liability of the bronchial tubes to spasm from many diseases and from many sources is of the greatest importance if we are to have clear ideas as to the nature and treatment of true asthma in early life.

The condition known as "thyroid asthma" is essentially one of latency, but is unfortunately named, as the symptoms of dyspnoea and cyanosis which it presents are referable entirely to the thyroid gland, and must not be confused with those of spasmodic asthma. (See Chapter X., p. 580.)

Ætiology.—The ætiological factors of asthma in children, so far as they are known, would appear to be the same as in adult life. In the immediate family history there will be found gout or asthma or nervous disturbances of some kind, chiefly of the functional variety. In the induction of attacks, as well as in the cure of the disease, local surroundings, particularly the atmospheric conditions, play a great part. The home surroundings, if of an exciting kind, will by disturbance of the nerve centres develop a tendency to asthma in the child, which might have remained latent under a more placid upbringing. The disease is not confined to any one class of society, although the well-to-do provide a larger proportion of cases, and their habits of life tend to the propagation of the disease. The exciting cause of an attack may be unascertainable. In other cases it is trifling, and would be negligible in a child not predisposed to asthma. A slight attack of indigestion, a heavy meal before going to bed, a trifling cold, an exciting party, a railway journey, a change of locality—these are some of the factors which may excite an attack. Sometimes there would appear to be a pent-up storm in the nervous system, which, after showing itself by certain signs of instability, at last finds vent in a well marked attack of asthma, which may continue for a week or more. As Leonard Guthrie expresses it, "Most nerve storms brew before they break." Asthma may

be regarded as one of the manifestations of a nervous system which is unstable in some of its parts, although apparently normal in others. The same child may at one time suffer from asthma, at another from eczema, and at another from urticaria or erythema exudativum multiforme, but never as a rule from more than one at the same time.

SYMPTOMATOLOGY.—True spasmodic asthma, viewed as a clinical entity, is characterized by attacks of dyspnoea coming on suddenly, with over-expansion of the chest, imperfect action of the respiratory muscles, and obstruction to the passage of air through the bronchi, most marked during expiration.

The young subjects of asthma are usually bright, intelligent children of the temperament which used to be described as "sanguine," but at the same time of spare build. They are liable to catch cold easily, and any of the infectious fevers they may be exposed to; they are often dainty feeders, with a liking for the delicacies rather than the substantial of the table; and in working off the superabundant cerebral energy they are apt to overtax the bodily strength. A typical attack will usually be preceded by over-exertion of some sort, mental or physical.

An attack is often developed suddenly during the night, in the very early hours of the morning. The patient is awakened by a feeling of tightness about the chest, the breathing is laboured and difficult, coughing comes on, and a painful sense of want of breath soon alarms the sufferer. The dyspnoea may be intense, and the chief trouble is expiratory, the chest being in the position of full inspiration, and strenuous efforts being made by the expiratory muscles to empty it of air. The attack may last for an hour or more, and then the spasm passes off and the patient drops off to sleep again. In severe cases there may be marked orthopnoea and cyanosis. Constitutional symptoms, such as fever, rapid pulse, etc., are usually absent, and the whole of the manifestations of an attack may be traced to obstructed breathing in the bronchi, to obstructed pulmonary circulation, and to the accompanying emphysema.

On examination of the chest during an attack one finds it over-distended, in the position of full inspiration. The note over all is hyper-resonant, and the cardiac dulness may be obliterated. The breath sounds, both on inspiration and expiration, are prolonged, especially the latter, and are accompanied by cooing, whistling, and musical sounds. In a later stage coarse moist sounds from the out-pouring of secretion into the bronchi may be audible. The chest symptoms are usually bilateral, but in rare cases it may be found that only one side is affected.

Typical attacks of asthma tend to recur, and may subside so completely in the intervals, which may last for weeks or months, as to leave no permanent traces on the lungs or the chest or the general health. In other cases they recur more frequently and last longer. In the case of repeated attacks, from the over-expansion of the chest and the difficult expiration, emphysema is apt to develop, the normal elasticity of the lungs to be impaired, and a barrel-shaped chest to be formed. From repeated attacks of severe coughing and cyanosis the facial appearance may be altered in a suggestive way. There is a tinge of cyanosis about the face, and some dilatation of the vessels of the cheeks and of the conjunctivae. The bronchial tubes become weakened in time by repeated attacks and a condition of chronic bronchitis is established. It is important, therefore, to recognize that we may meet with a chronic asthmatic subject who has bronchitis, as well as a chronic bronchitic subject who has attacks of bronchial spasm, often called "asthma." The distinction between the two is to be made chiefly from the history. In asthma we

obtain a history of sudden attacks of dyspnoea occurring without fever and passing off rapidly. In bronchitis we learn of feverish attacks of bronchial catarrh, lasting for a week or longer. The recognition of these two different conditions is important also from the point of view of treatment, for in the one case asthma is to be treated as the primary disease, and in the other case bronchitis.

PATHOLOGY.—The pathology of asthma would appear to be the same at whatever period of life it occurs. Many adults suffer from asthma which first developed in childhood. Opinion is still divided as to whether the disease is due to a primary spasm of the bronchial tubes or to a vasomotor disturbance with swelling and oedema of the mucous membrane of the bronchi. Probably both factors may be present, and in some cases the muscular spasm may predominate, while in others the vasomotor element may be most pronounced. The vagus nerve is probably the means of conducting some disturbance from the higher nervous centres to the bronchi, which manifests itself as an attack of asthma. Whether the seat of disturbance is in the lower levels or in the higher is at present undetermined. What is definitely known is that a nervous element is always present, and that a certain predisposition exists in those affected which is not a feature of normal childhood. This predisposition to asthma is often accompanied by certain other peculiarities which must be regarded as directly associated. Amongst these may be mentioned paroxysmal sneezing, rhinorrhoea, and hay fever. It will also be found that the more frequent the attacks of asthma, the greater is the tendency for it to become habitual. That is to say, the nerve centre involved will in time become more excitable and more easily discharged, until finally the condition of stable equilibrium is entirely lost and the nerve centre has acquired the habit of asthmatic breathing.

PROGNOSIS.—The prognosis as regards life is good provided that the patient can be protected from the development of chronic bronchitis and emphysema to a marked extent. The prospect of a complete cure is by no means so good. It is quite possible to take a hopeful view, and say that the young patient will "grow out of it." This may happen, more especially in those cases in which the family tendency to the disease is not marked, and in which a spasmodic tendency has been manifested in the child in other ways. Very often, as the child grows, the tendency to asthma dies down, but only to be replaced by some other neurosis, such as *irritaria*, periodic vomiting, or erythema. But in cases of true asthma it is unwise to give anything but a very guarded prognosis as to the future prospects, for they will depend on many factors which we cannot control, and on many features of the disease which we do not understand. Undoubtedly, the frequency and severity of the attacks will be greatly influenced by the course of treatment pursued.

TREATMENT.—Perhaps the most important part of the treatment is to see that the asthmatic child is not regarded as delicate and requiring coddling, owing to a tendency to bronchitis and a weak chest. The weak spot is in the nervous system, and not in the lungs. We have to consider first the treatment of an acute attack, and secondly that of the underlying disorder.

In the case of a first attack the practitioner will probably find that he has nothing to guide him as to the special peculiarities of the child, and the diagnosis will often have to be arrived at by excluding the other possible causes of such urgent dyspnoea. There will probably be a considerable degree of air-hunger from mechanical causes, and therefore the room should be filled with fresh air—and

emptied of anxious relatives. The inhalation of oxygen may also tend to relieve this special form of distress. For the relief of spasm of the bronchial tubes, hot poultices or fomentations or turpentine stupe may be applied to the back and front of the chest. A turpentine stupe is to be preferred, because of its speedy action and the fact that its application hampers the freedom of the chest movements less than poultices. An even more effective measure in skilled hands is a hot wet pack, in which the child should be enveloped until the skin generally is red and moist. Another method of allaying spasm is by means of an emetic, which in addition, by emptying the stomach, will probably remove some irritating food, a common exciting cause of an attack. Sulphate of copper is usually effective, and 1 or 2 grains may be given in 2 drachms of water. If this fails, or if a more drastic action seems called for, from 1 to 5 minims of the injectio apomorphinae (R.F.) may be given subcutaneously. Sips of very hot water may be given freely. After the stomach has been thoroughly emptied, a sedative draught containing iodide of potash (grs. x.), bromide of potash (grs. x.), and chloral hydrate (grs. v.) will increase the desired effect. It is impossible to say beforehand what means of allaying the spasm will prove most successful in any given case. The use of so-called steam inhalations, as in bronchitis, may sometimes do good, but is often of no benefit. One antispasmodic is very reliable—namely, a full dose of brandy, ½ ounce in 1 ounce of hot water for a child of five years. This must be reserved for occasional use only. It is distinctly preferable to morphia, which may or may not relieve the attack, but has many drawbacks. In cases of extreme cyanosis it may be advisable to bleed or leech the patient. After the acuteness of the attack has subsided, the child should be kept on a spare diet for a few days and the bowels freely opened.

An asthmatic child, if the tendency to asthma is to be kept in check, requires his whole mode of life and surroundings carefully supervised. The nervous constitution which underlies the affection must be carefully protected from overstrain. Lessons should go on, but they must be regulated as to duration and character, and the competitive element of school life must be eliminated. The home life should be quiet and unexciting, and children's parties, theatres, etc., must be forbidden. Plenty of open-air life and exercise is required. The place of residence may be all-important. For some sufferers seaside air is bad, for others high mountain air, and for others the lowland valley air. Some will be free from asthma only in towns, and others only in the country. These individual peculiarities must be considered, and, if possible, a residence should be established where the atmospheric surroundings are suitable. In hospital practice it is difficult to study the clinical features of asthma in the wards, because as a rule all symptoms disappear after the patient's admission, probably from the healthier atmospheric conditions and the quiet mode of life. The dietary suitable for one child may be quite the opposite for another. As a rule a simple non-stimulating diet is best, with a predominance of the fats and carbohydrates, and a moderate allowance of proteids, the stimulating meat foods being specially limited. It is always advisable to give small meals at frequent intervals, so as to prevent the gastric disturbance which tends to bring on an attack of asthma. Specially indigestible foods are to be avoided, while in individual cases it may be found necessary to interdict certain articles of diet which, although harmless in the case of ordinary children, have directly induced an attack.

Certain accompanying conditions may call for attention. The chief of these is

bronchitis, which may act as an exciting cause of further attacks. Special attention should be directed to restoring the bronchial tubes to a healthy condition. Further, the whole of the upper respiratory passages should be examined, and any disease in the nose or naso-pharynx should be treated. While the cure of asthma is not to be expected from the removal of adenoid growths, great improvement will often follow the restoration of normal nasal breathing. This is probably due to the removal of a source of peripheral irritation, and also to the improvement in the patient's general health. Cauterization of the nasal septum or of some particular spot on it has not justified itself as a cure for asthma in early life. Any other sources of peripheral irritation that may tend to induce attacks should be looked for and removed. Amongst these may be mentioned fecal masses in the bowel, worms, pinworms, and errors of refraction.

The further medicinal treatment to be considered is that of relapsing or chronic asthma. There is considerable difficulty in telling beforehand what medicine will suit any particular case. The idiosyncrasy of the patient is manifested in regard to medicines as well as to other things. Most of the effusions will benefit from a course of iodide of potassium, arsenic, and carbonate of ammonium. This may be given for a fortnight continuously, and repeated at intervals of a month or six weeks. Other antispasmodic drugs which have been found useful are belladonna, hyoscyamine, lobelia, and atropine. When the attacks are nocturnal only, it is advisable to give the patient a full dose of the medicine before he goes to bed. The direct inhalation of the fumes of burning powder containing nitre, stramonium, etc., will undoubtedly give relief to the spasmodic breathing in many cases, but it is doubtful whether they should be recommended in the case of children. The action of these fumes on the bronchi would appear to be distinctly injurious, increasing the tendency to bronchitis, and even as regards the asthma the effect is short-lived and never curative. The more these fumes are used, the more the necessity for further relief, and the worst cases of chronic bronchitis and asthma are usually found to be associated with the frequent and prolonged use of sedative inhalations.

As already stated, the recurrence of attacks seems to induce a habit in the nervous centres which brings about more or less continuous asthmatic breathing. In such cases, if bronchitis is not marked, it has sometimes been found beneficial to put the patient to bed and attempt to break this habit by full doses of chloral and bromide of ammonium. Ten grains of the former and twenty of the latter may be given three times a day to a child of ten years, and continued until the patient is in a state of continuous drowsiness, and this condition may be maintained by the bromide alone for some days. By this means the habit may be broken, and a more or less prolonged interval from attacks may follow. Medicines directed to the toning up of the nervous system, such as *nux vomica*, and of the pulmonary system, such as cod-liver-oil, will be found useful in all cases of asthma.

FOREIGN BODIES IN THE BRONCHI.

INTRAUTERINE.—Young children, and even infants, are very liable to get foreign bodies into the air-passages, owing to their tendency to put all sorts of things into their mouths. The ordinary caution which comes with older years has not yet been developed, and when running or playing about with some foreign substance in the mouth it very easily slips backwards and through the glottis. Larger substances will probably be retained in the larynx or trachea, but smaller ones, such as peas,

beads, seeds of fruit, ticks, buttons, etc., may find their way *into* one or other bronchus. Owing to the anatomical situation, the opening into the right bronchus affords a more easy entrance than that of the left, and consequently the right lung is more frequently the seat of disturbance than the left.

When the occurrence takes place in the presence of some adult, and when the symptoms are immediate and severe, the diagnosis can be easily made. But when the occurrence is unobserved, and the immediate symptoms are not alarming, the condition may be entirely overlooked. Intermittent fits of coughing may lead to a visit to the doctor, and from the indefiniteness of the signs in the lungs a diagnosis of hooping-cough, or pressure from enlarged glands or some other irritative lesion of the lungs, may be made. Or it may be that the foreign body has already produced local changes in the lung of the nature of broncho-pneumonia, which is regarded as the original disease, and the primary cause is overlooked.

SYMPTOMATOLOGY.—The sudden onset of violent coughing, possibly leading to cyanosis, and without any pre-existing disease, always suggests the probability of a foreign body in the air-passages. This is Nature's reaction to an irritant, and her effort to remove the foreign substance. The severe attack of coughing is accompanied by great distress on the part of the child, evidently a sense of impending suffocation, and dyspnoea. After a time the coughing diminishes, but tends to recur in paroxysms at longer or shorter intervals. Pain may be complained of at some part of the back or front of the chest. Prompt relief will be afforded if the foreign body is expelled from the bronchus, but if it remains the symptoms will vary according to the nature of the foreign substance and its position in the bronchus. A sharp body fixed in the tube will cause more irritation than a smooth body with a certain range of movement. The symptoms of distress will be greater when a main bronchus is occluded than when only one of the subdivisions is blocked. Complete obstruction will cause greater distress than when a certain amount of air can pass through.

The physical signs will also vary considerably, according to the nature of the foreign body and the degree of obstruction. In the early stages the important sign in the case of complete obstruction is that the breath sounds are entirely lost over a lobe or the whole lung, according to the size of bronchus affected, while the note of percussion may be unimpaired. There may be a few rhonchi or rales from excessive coughing, but as a rule in the early stages the lungs show nothing farther on physical examination. In the case of a foreign body which allows the passage of air either through it or past it, there may be no physical signs until the air-passages are completely blocked.

The progress of the condition is very variable. Acute attacks of dyspnoea and coughing may recur. On the other hand, there may be a prolonged period in which the symptoms are few, although the bronchus is becoming ulcerated. Again, the child may continue acutely ill. Fever sets in. The bronchus soon becomes ulcerated, and secondary infection occurs, leading to pneumonia, usually of the broncho-pneumatic type. This disease spreads through the affected lung, and may extend to the opposite side. Around the seat of obstruction local inflammation may lead on to suppuration, and an abscess is formed which infiltrates the pulmonary tissues, or ruptures through the pleura, causing empyema, or may extend into the mediastinum. The lung tissue beyond the obstruction may show signs of one large cavity or a number of cavities containing pus, and in rare cases gangrene of the lung sets in.

The DIAGNOSIS may present very great difficulty in those cases in which there is no history of a foreign body and no persistence of signs of active irritation of the bronchus. If a paroxysmal cough continues, and there is no evident cause for this on physical examination of the chest, and no reason to suspect whooping-cough, the presence of a foreign body may be suspected. Two means are at our disposal in trying to secure an exact diagnosis. The one is radiography and the other is the bronchoscope. Radiography has only a limited application, because only certain substances will produce a shadow, and very often these substances are not present in the foreign bodies which find their way into the lungs of children. The bronchoscope in skilled hands is of the very greatest value in diagnosing the presence of a foreign body. In most cases there will have been sufficient evidence in the physical signs to indicate which lung is affected.

The chief points suggestive of the presence of a foreign body in a bronchus have been summed up by Newton Pitt as follows: (1) The history of swallowing something immediately before the onset of an acute attack of severe dyspnoea and cough. (2) A whistling or wheezing sound, which the patient can localize to one side, and which varies in quality if the foreign body is not fixed. (3) Great distress, with not infrequently a constricting pain behind the sternum, made worse by movement. (4) Dyspnoea with varying exacerbations, increased by coughing and exertion. (5) Absent or, rarely, noisy breath sounds, with some slight diminution of vocal fremitus, limited to one side of the chest. (6) At first resonance on the affected side, soon consolidation with dullness, and later the signs of cavity and the simulation of phthisis. (7) The X-rays show defective movement of the diaphragm and of the thorax, and alteration in the density of the lung, and not infrequently the outline of the foreign body itself. (8) Delay in the commencement of inspiration, as compared with that on the other side. (9) Defective movement, with consequent diminution in the size of the affected side. (10) Violent paroxysmal persistent cough. (11) Early pyrexia, which later becomes hectic, associated with chills. (12) After a short time an expectoration, more or less profuse, purulent, often bloody, and later often fetid. (13) In some cases tussis and vomiting, which may be very troublesome. (14) Pneumonia often develops in forty-eight hours; this may be septic and followed by gangrene.

The PROGNOSIS in the case of a foreign body in the bronchus is always serious. Spontaneous recovery may follow the expulsion of the substance by coughing. When this occurs at an early stage, recovery may be complete. When the foreign substance is coughed up or removed artificially at a later stage, the prognosis will depend on how far inflammatory and infective changes have progressed in the lung beyond the seat of obstruction. As a rule in children these go on rapidly, and even with the removal of the foreign body pulmonary disease of a serious character remains. When a foreign substance remains fixed in a bronchus, the secondary changes which follow almost invariably lead to a fatal issue. Toleration may be established for a time, but this only means postponing the inevitable issue.

TREATMENT.—The great advance in the successful treatment of foreign bodies in the bronchi is due to the invention of the bronchoscope. All other methods previously employed have been uncertain in their action, and successful only in very few cases. Inversion of the patient and the induction of violent coughing may be tried with a view to the forcible expulsion of the foreign body. The severe cough which the irritation of a foreign substance induces may be sufficient to cause

its expulsion. Many foreign bodies swell from the absorption of moisture in a bronchus, and thus become even more firmly impacted, so that it is not until suppurating ulceration has occurred that they become sufficiently loose to be expelled by coughing. Tracheotomy and exploration of the bronchi through the wound may lead to the discovery and removal of a foreign body by forceps, but it usually ends in failure to find the body. On the other hand, by means of the bronchoscope a foreign body even in the subdivisions of a bronchus may be exactly located and removed with ease. The instrument calls for a considerable amount of practice and skill in its use. In the case of children a general anæsthetic will be necessary. The results when immediate treatment has been carried out are very good, but in cases of some standing the removal of the foreign body may require to be followed up by treatment of the pulmonary inflammation which has arisen secondarily. (See also Chapter VI., p. 300.)

DISEASES OF THE LUNGS.

ATELECTASIS: CONGENITAL AND ACQUIRED.

INTRODUCTION.—The term "atelectasis" is applied to a condition of the lung in which the air vesicles, in whole or in part, are not expanded. In the congenital or natal form the air vesicles remain in the foetal condition. In the acquired or post-natal form the air vesicles, which have at one time been expanded, collapse as the result of disease or disturbed function in the lungs. In both cases the condition is one of early life, and after the age of six years most of the causes have ceased to act.

1. Congenital Atelectasis.—**ETIOLOGY.**—In the case of a healthy infant the lungs are rapidly expanded immediately after birth by the establishment of respiration, and more especially by the powerful inspiratory and expiratory movements that accompany crying. Hence it has rightly been considered advisable to make a newly-born infant cry lustily, by mechanical stimulation if necessary, in order to make sure that the breathing powers are normal. We may safely assume, on hearing a child cry strongly, that the breathing powers are all right and that the lungs are fully expanded.

In certain cases after birth an infant does not breathe properly. The respirations are irregular and shallow, there is a tendency to cyanosis, the cry is feeble or absent, muscular movements are defective, and the infant generally shows signs of extreme debility.

The condition of feebleness in the infant is the leading factor in the production of atelectasis. The cause may be in the state of the mother's health and nutrition, in the fact of prematurity, or in the imperfect development of the infant. Or, again, the cause may lie in the process of birth, which may have been prolonged or difficult, or complicated in such a way as to leave the infant exhausted or almost lifeless at the end. It is plain therefore that, while the non-expansion of the lungs may be one of the conditions present, and one which attracts attention by its symptoms, the whole system of the child needs attention, and respiration will probably only be established if the other vital powers rally or are strengthened. Too much stress must not be laid on atelectasis pulmonum as a separate condition.

and severe mechanical measures must not be adopted, which in the process of inflating the lungs would still further exhaust a feeble infant.

The respiratory act being a complicated one, the defective inflation of the lungs may be dependent on various factors acting singly or in combination. The cerebral centres, cortical and medullary, are essential for normal respiration, and they may be acting feebly. Specially will this be the case when during parturition there has been great venous congestion, or possibly hemorrhage, inside the cranium, and the newly-born infant is in a semi-stuporous condition. The normal stimuli to respiration which come from the skin and elsewhere do not produce much effect on the enfeebled nervous centres. In cases of simple venous congestion about the brain following a difficult labour, the disturbance of function will soon pass off and normal respiration be established, provided that the infant is healthy. Again, the costal muscles are necessary for respiration, and if feeble and flaccid they may not be able to inflate the lungs. In cases of great abdominal distension from fluid or air, the action of the diaphragm may be so interfered with that it cannot play its proper part in respiration. Another factor that plays a part in increasing the volume of the lungs after birth is the pulmonary circulation, and the new circulation through the lungs is also, in all probability, a stimulus to the respiratory centres. It has often been noted in fatal cases of atelectasis that the fetal condition of the heart has persisted, the foramen ovale and ductus arteriosus being still patent, and it has been assumed that this is a superadded defect in the infant. On the other hand, it may be that the persisted fetal condition of the heart has led to an imperfect pulmonary circulation, and has thus contributed directly to the non-expansion of the lungs. While in some cases it may be possible to determine the presence of some of these special factors, in others one can only ascribe the atelectasis to the extremely enfeebled condition of the infant.

SYMPTOMATOLOGY.—The signs of very low vitality are usually well marked. The infant is probably small and ill-nourished. The muscles are soft, and the ordinary muscular movements of the limbs are absent. The cry is feeble or entirely absent. The respiratory movements are extremely limited and the breathing irregular, with considerable intervals in which it is unperceptible. The lungs sink in instead of expanding during inspiration. The skin surface may show a general pallor, with touches of cyanosis about the face and extremities. Intermittent attacks of cyanosis are common. The power of sucking is absent or feeble, and even the power of swallowing may be much diminished. The temperature is often subnormal, the fontanelle depressed, and the skin cold. Stimulation may lead to a slight improvement in all these symptoms, but it is usually temporary only, and the infant soon lapses again into its former condition.

Physical examination of the chest does not give much additional information. If the child survives even for a few hours, there is usually some expansion of the lungs—patchy, it may be, but sufficient to give a resonant note on percussion. If dullness is present, it is usually most marked at the bases posteriorly. The tracheal sounds are of a feeble vesicular type, rarely bronchial. The pulse and heart sounds are extremely weak.

PATHOLOGY.—If the infant has never breathed at all, the lungs will be found in a state of complete atelectasis. They are dark red or purplish in colour, feel tough and leathery, are non-compliant, and sink in water. They can be easily and fully expanded by air pumped in through the trachea.

If the infant has breathed, but imperfectly, for a few hours, parts of the lung will be found inflated, and even emphysematous. These inflated parts show up against the collapsed portions, being raised above the general surface and of a rose-red colour. The expanded areas are often found along the anterior borders of the lungs, and may have a patchy distribution through the lungs generally. The other viscera—liver, spleen, and bowels—are usually in a condition of passive congestion.

Diagnosis.—The diagnosis is to be made from the symptoms rather than from the physical signs in the lungs. When an infant has never breathed properly after birth, and remains in a more or less apnoeic condition, one can assume with certainty that the lungs are not expanded. Before ascribing all the symptoms to general debility, it is advisable to examine carefully for any indications of a cerebral cause, intra-cranial pressure, or abdominal distension which may have interfered with normal respiration. At birth there may have been considerable disturbance of the respiration by the passage of foreign substances (liquor amnii or mucus) into the air-passages, but this condition usually manifests itself by coughing, extreme cyanosis, etc.

Prognosis.—As the condition of atelectasis is usually only one of the manifestations of extreme debility in an infant, the prognosis will depend on how far the general vital weakness is amenable to treatment. In cases of simple exhaustion following a difficult labour the prognosis is good. In the case of premature or wasted infants the prospect of recovery is not good. There may be a certain success in the efforts to establish respiration, but the infant tends to relapse; cyanosis becomes marked, and death follows. In other cases death takes place rather suddenly after partial restoration of the respiratory powers.

The longer the patient lives, the better is the prospect of recovery. Death usually occurs, within the first seven days, and if the child survives that period, and can take nourishment, one may be hopeful. In the case of premature infants one usually finds that for some time the respiratory movements are extremely shallow and irregular, and yet they may become normal in time. When congenital cardiac lesions are present or a persistence of the fetal cardiac condition, the prognosis is less good. The persistence of a subnormal temperature, of cyanosis, and of inability to swallow, and the supervention of convulsions, must be regarded as ominous symptoms in cases of atelectasis.

Treatment.—In the case of a healthy infant born apnoeic and possibly cyanosed, active measures must be taken to induce respiration. Slapping with a cold, wet towel may be employed; or the child may be put into a bath of hot water (temperature 110° F.) for a few moments, and then into a cold one. Failing these means, artificial respiration should be carried out by means of Sylvester's or Schultz's method. In both of these methods there is a risk of doing harm to the infant unless great care is taken. Schultz's method is specially likely to lead to injury. It is an advantage to the infant to have the lungs expanded if the vital powers are exhausted or some injury done to the body by the efforts at restoration.

In the case of a delicate and feeble infant, in whom the atelectasis is only one symptom of the impaired vital powers, violent measures should not be adopted. The chief requisites are warmth and stimulation. The child should be loosely swaddled in flannels and cotton-wool, surrounded with hot bottles, and at the same time allowed a full supply of cool fresh air to breathe. A hot, stuffy room is the

word place for the infant to be in, while pure air at a temperature of 60° F. certainly will tend to induce full respirations. Stimulation is best carried out by means of hot brandy and water. Five drops of brandy in 2 drachms of water may be given every hour until six doses are taken. If the child is going to live, some improvement will probably have taken place by this time, and a hot bath may be given, followed by gentle friction of the whole body. The further treatment is the same as that adopted for premature and delicate infants, and it will be found that more benefit will be likely to accrue from treatment directed to the general condition than from local measures directed to the expansion of the lungs.

2. Collapse of the Lung—Acquired Atelectasis.—**ÆTIOLOGY.**—When consolidation has once been fully established, the fatal condition of the lungs can never be returned to, but there remains for some years the possible danger of collapse of some part of the lung tissue. Various factors tend to bring about this condition, which is not found in the case of healthy children with healthy lungs. An important predisposing cause is general debility from any cause, whether it be from constitutional delicacy, or wasting disease such as diarrhoea, or exhausting disease such as rickets. It is in the subjects of such conditions that collapse of the lung may be expected to occur.

Collapse of the lung is invariably secondary to some local interference with the normal action of the lungs. The most common cause is acute disease of the lungs, and here bronchitis and broncho-pneumonia take the chief place. The secretions of the bronchi accumulate in the tubes and are not expelled. The air is not able to pass freely to the alveoli during inspiration, although during expiration the alveoli may be partly emptied. This is due to a valve-like action of the plugs of mucus lying in tubes of a gradually narrowing diameter as they approach the alveoli. Finally the smaller tubes become completely blocked, so that no air can pass in or out, and what air remains in the alveoli is absorbed by the bloodvessels. As this condition of collapse is limited to early life for the most part, it is clear that some factors must be present which disappear with the growth of the child. Amongst these, the inability to cough in a forcible manner is one. In early childhood the reflex act of coughing is not easily excited, and an accumulation of mucus in the trachea or bronchi excites only a slight cough, which is not sufficient to clear the tubes. The valuable function of coughing would therefore seem to be acquired by some children with difficulty, and the lining membrane of the bronchi would seem to be less irritable than it becomes later on. It is plain, therefore, that children who have not learnt, or who have not been taught, to cough properly are especially liable to collapse of the lungs during an attack of bronchitis. Another characteristic is the weakness of the muscles of expiration in infancy as compared with later years. The act of coughing implies a full inspiration, a firm closure of the glottis, and a strong and sudden expiratory effort. Now, in infancy the muscles involved in all of these acts are not nearly so powerful as they become later on, while the difficulties to be overcome in the way of bronchial obstruction are practically as great. In the presence of acute pulmonary disease these difficulties will be increased as the child's strength fails, and it is usually in the later stages of the illness that pulmonary collapse manifests itself.

There are other causes lying outside the lung which tend to produce collapse. Any mechanical interference with the expansion of the lung will lead to more or less collapse in the part affected. In the case of a pleural effusion, the lung adjoining shows an amount of general collapse proportional to the amount of fluid

present. Similarly, in the case of pericardial effusion or great cardiac hypertrophy the left lung is partially collapsed. The softened chest walls in rickets not only fail to expand properly, but show an actual falling in during inspiration. The area of this falling in is also shown in the lungs by a corresponding area of collapse. In the case of chronic obstruction in the upper respiratory passages—e.g., the nasopharynx—there is an imperfect entrance of air into the chest, the chest walls fail to expand properly, and at the same time the lungs tend to collapse.

SYMPTOMATOLOGY.—The effect of collapse of the lung is to reduce the effective area of respiration, and to diminish to the same extent the breathing-power of the patient. As Nature has supplied more than enough pulmonary tissue for ordinary and even extraordinary breathing purposes, the collapse may not immediately manifest itself. More especially is this the case when the collapse takes place gradually, as in the case of naso-pharyngeal obstruction or pressure from a pleural effusion. Any shortness of breath or hurried breathing consequent on this may be manifested only on exertion. Even a rachitic child may continue to breathe without obvious distress with a markedly softened and sunk-in chest, and it is only on any forced effort of respiration that the collapsed condition of the lung manifests itself. In all of these cases the gravity of the collapse may not be revealed until the occurrence of some acute affection of the lung, inflammation, catarrh, or oedema, when the condition of the patient may rapidly become alarming. In such a case hurried respirations, dyspnoea, and cyanosis, are apt to develop with a rapidity and severity quite out of proportion to the signs of pulmonary disease, and are an indication that the previous collapse had seriously diminished the area of available lung tissue.

In cases of bronchitis and broncho-pneumonia, collapse of the lung is almost invariably present to a greater or less extent. In some cases it will be found that large areas, almost lobar in extent, are involved, while in others small areas of a lobular distribution are scattered over the lungs. The anterior borders of the lung and the posterior parts lying in the hollows along the spine are specially apt to be involved. The exact areas will usually be those associated with the bronchial tubes which have been blocked with mucus, although in some cases collapse takes place in areas to which the air entry seems to be unimpeded.

The symptoms of collapse are very difficult to distinguish from those due to the accompanying disease. Hurried respiration, an increase of dyspnoea, orthopnoea, and cyanosis, may be due to collapse; but, on the other hand, these symptoms may be due to an increase of the bronchitis, of bronchial spasm, or of inflammation. One may assume that collapse has occurred when the respiratory symptoms become suddenly aggravated, without any alteration in the temperature, and without any marked alteration of the physical signs of the primary disease. In the case of delicate children who have presented signs of slight bronchitis, the sudden increase of pulmonary distress is often due to extensive collapse, which may prove rapidly fatal. The possible onset of collapse is a very real danger in the case of all young children under the age of two years who are suffering from obstructive disease of the upper or lower respiratory passages, or from any weakness in the muscular or nervous organs of respiration, or from any mechanical impediment to the expansion of the lungs.

The physical signs of collapse are not more characteristic than the symptoms. If the affected area is sufficiently large to be recognized clinically, there will be found impaired resonance, bronchial breathing or diminished breathing, and in-

creased vocal resonance. But, as will be seen at once, these are also the signs of a pneumonic patch, such as one meets with in broncho-pneumonia. Probably the best evidence in favour of a collapsed area, as contrasted with a broncho-pneumonic area, is that the former develops suddenly, without rise of temperature, and tends to disappear suddenly if the air-passage is cleared. Collapse of considerable extent is frequently found in the lung lying along the spine posteriorly, and when the physical signs of consolidation are found there the condition is more likely to be one of collapse than of pneumonia. Small areas of collapse give no definite signs, and any impairment of resonance that might be expected is often obscured by a condition of emphysema which develops around the collapsed portions.

The clinical course of a case of collapse will vary with the nature of the primary disease, and also with the extent and duration of the collapse. In acute pulmonary diseases numerous small areas of collapse may develop, and rapidly disappear with the fuller expansion of the lungs. Others persist and present all the physical signs of solid lung. But in acute disease of the lungs, while collapse may be an important factor in determining the issue of the case, it will be found a difficult matter to decide clinically as to what extent the collapse is responsible for the symptoms.

PATHOLOGY.—On examining a collapsed lung in the post-mortem room, one finds certain areas of the surface of a deep purple colour, rather depressed below the adjoining parts, and of a soft, firm feel. On section of the lung it will be seen that these areas have a lobular distribution corresponding to the finer divisions of the bronchi. The surrounding tissue is usually emphysematous. A piece of collapsed lung will sink in water. Sometimes larger areas, amounting to half or the whole of a lobe, are involved, due to the extension of the collapse associated with the weakened breathing preceding death. The naked-eye appearance of a collapsed portion of lung is very similar to that of a pneumonic patch, and the microscope may be necessary to distinguish between the two. A patch of *areal* collapse will show the collapsed alveoli surrounded by congested blood-vessels, while in pneumonia there will be exudation of blood-cells into the alveoli.

In cases of long-standing collapse, such as occurs in connection with empyema or other forms of effusion into the pleura, marked changes take place in the lung. The lung shrinks and becomes much denser and firmer from the formation of fibrous tissue. Its power of expansion is lost, and it has the appearance of a fleshy mass, which has suggested the name commonly applied to the condition—*i.e.*, "ossification of the lung." The condition is very similar to that which has been already referred to as chronic fibroid pneumonia.

DIAGNOSIS.—As the symptomatology is so indefinite, the diagnosis is often difficult. The sudden onset of hurried and laboured breathing in a feeble infant who has had no marked signs of pulmonary disease, save perhaps a slight cough, should always suggest the possibility of collapse. This is strengthened if the temperature is not raised, if cyanosis and laboured action of the heart are present, if there is sinking in of the lower ribs or infra-costal region, and if the physical signs of collapse of the lungs are present. In the case of acute disease of the lungs the occurrence of collapse is very apt to be masked by the signs and symptoms of the primary disease. If areas of collapse can be made out by the physical signs, the chief distinguishing feature between them and pneumonic patches are the suddenness with which the former come and go. By exciting the act of coughing one may find that a dull area has suddenly become resonant, in which case it has probably

been collapsed and not pneumonic. Sudden dyspnoea occurring from spasm of the bronchi in the course of an attack of bronchitis is to be distinguished from that due to collapse by the auscultatory signs. In spasm of the bronchi the breath sounds become extremely harsh and prolonged, the expiration being especially laboured and long. The sudden onset of emphysema may lead to acute dyspnoea, and this condition is to be distinguished from collapse by the marked increase of resonance over the lungs generally.

PROGNOSIS.—In the case of weak infants suddenly attacked by collapse of the lung, the occurrence may be taken as an indication of an extremely enfeebled state of health. The accompanying difficulty in respiration is often sufficient to exhaust the remaining strength of the patient. In the case of acute pulmonary disease the importance of collapse depends on the extent and duration of the complication. Collapse of extensive portions of the lungs in the course of an attack of bronchitis or broncho-pneumonia may greatly aggravate the symptoms, or even lead to a fatal termination.

TREATMENT.—Slight and passing attacks of collapse occurring in the course of acute pulmonary disease call for no special treatment. It is only in the severe form that attention must be paid to relieving the condition, if possible. The objects of treatment are to free the breathing and to stimulate the respiratory powers. The breathing may be freed by changing frequently the position of the patient in bed, by propping him up, and by seeing that the body and bedclothes are light and loose. The respiratory passages may be cleared of their contents by the use of expectorants (hot water, ammonia, iodide of potash) or an emetic (sulphate of copper, gr. i. in a teaspoonful of water), if the patient is strong enough. Stimulation may be carried out mechanically, but with great caution, for strenuous efforts to relieve collapse of the lungs may lead to collapse of the patient. Hot baths, stimulating liniments to the chest, or a turpentine fomentation, are useful. Small quantities of beef-tee or meat extract may be given frequently. Brandy and strychnine are valuable. Plenty of fresh air, or oxygen inhalations when cyanosis is present, will act as direct stimulants. All epate medicines used in the treatment of the primary disease should be stopped, as collapse of the lung tends to induce a stuporous condition. Any interference with the free action of the diaphragm, such as is caused by flatulent distension of the abdomen, should be relieved as quickly as possible.

MASSIVE COLLAPSE OF THE LUNG.

This term has been applied by Pasteur to a condition of active deflation of the lung, affecting one or more of its lobes, which occurs in the absence of any obstruction in the air-passages, and is probably always due to disturbance of the muscular mechanism of respiration. The upper lobe or lobes may be affected when the costal muscles are thrown out of action, but the lower lobe or lobes are more frequently affected as the result of paralysis of the diaphragm. In the case of children the most common cause is paralysis of the diaphragm following diphtheria, but he has also found that massive collapse may follow *ferm* reflex inhibition of the diaphragm after an intra-abdominal operation.

The clinical signs of massive collapse of a lower lobe are extremely like those of pneumonia, with which condition it has probably often been confused. There

may be an access of dyspnoea and cyanosis, with slight cough and greenish expectoration. There is very marked dullness over the area affected, with diminution or absence of breath sounds and possibly distant tubular breathing. The unaffected lung becomes over-distended, and the apex of the heart tends to shift towards the affected side. The temperature is not raised or only very slightly, which is an important point in distinguishing this condition from pneumonic consolidation.

The mechanism of its production would appear to be that, the distending force in the neighbourhood having become inactive, the lung tissue contracts by virtue of its own resiliency. At a necropsy the part affected is found to be completely emptied of air, and usually sinks in water. The treatment of the condition must be directed to that of the underlying cause, and the prognosis will depend on whether that cause is temporary or permanent.

EMPHYSEMA.

INTRODUCTION.—In childhood emphysema is common enough as a symptom, but does not exist as a separate disease such as is met with in later life. The over-distension of the air vesicles which characterizes the condition is often acute and fleeting and passes unrecognized. When the cause is prolonged, as in chronic diseases, emphysema of a chronic type may be established, and the resulting changes in the lungs and the thorax may closely simulate those of adult life. These changes may become permanent, may seriously interfere with the growth of the child, and may ultimately be found in adult life in a well-marked form of secondary emphysema. The ordinary type of over-distension of the air sacs is known as vesicular emphysema, and under great strain a rupture of one or more vesicles may occur, producing what is known as interstitial or interlobular emphysema. Here the air passes into the connective tissue supporting the lung, and burrows between the alveoli and under the pleura. Considerable accumulations of air may thus be formed and cause great pulmonary embarrassment. When the rupture occurs near the root of the lung, the air tends to pass into the mediastinum, and thence into the subcutaneous tissues of the neck and face and body generally. The tendency to interstitial emphysema from strain is much greater during the first five years of life than at any later period. Another form of emphysema is termed compensatory, inasmuch as it follows the inaction or consolidation of some other part of the lung in which the air vesicles are closed. In all cases of collapse and of pneumonia the solid areas are surrounded by over-distended vesicles, which are, as it were, doing double duty. In the case of a fibroid lung on one side, it will be found that the other lung shows increased bulk from compensatory emphysema. For this condition S. West prefers the term "complementary hypertrophy" rather than "emphysema," because it is accompanied with increased, and not diminished, functional activity of the lung.

ÆTIOLOGY.—Excessive respiratory movements are the cause of emphysema. In childhood the weakness of the pulmonary tissues and the great elasticity of the chest wall probably help to explain the greater frequency of emphysema at that period of life. The strain on the lung may be brought about either during inspiration or expiration, although no doubt the latter is the more important. The result of severe inspiratory efforts is over-expansion of the upper part of the front of the

chest, which persists as long as the dyspnoea lasts, and emphysema occurs in the upper and anterior parts of the lungs. Powerful expiratory movements, especially with any obstruction in the respiratory passages, quickly produces emphysema. In obstructive lesions of the larynx, or trachea, or bronchi, such powerful expiratory efforts are common. But the most effective cause is coughing, in which, the glottis being closed, the contraction of the diaphragm and chest wall drives the air with great force into the unsupported parts of the lung. Hence in hooping-cough emphysema to a greater or less extent is always present. Suffocative breathing from any cause has the same effect. In an attempt to resuscitate an apnoic infant at birth, the process of insufflating the lungs by blowing air through the mouth may produce emphysema.

All the acute pulmonary affections, such as bronchitis, broncho-pneumonia, and tuberculous pneumonia, which are associated with dyspnoea are also accompanied by emphysema. In croup, laryngismus stridulus, acute laryngitis, and hooping-cough, emphysema is common. The severity of the cough in hooping-cough may produce not only vesicular emphysema, but also interstitial emphysema. Amongst chronic affections, asthma is the most common cause, and here chronic emphysema is a constant accompaniment. In cases of rickets, also, with marked thoracic deformity emphysema is common in those parts of the lung which are unsupported.

SYMPTOMATOLOGY.—As a rule the symptoms of vesicular emphysema are obscured by those of the underlying disease in the case of acute pulmonary affections. The physical signs may be of an indefinite character, but if a sufficiently extensive area of the lung is involved it will be shown by an increase of resonance, with prolonged and faint respiratory sounds on auscultation. Marked dyspnoea or severe coughing usually precedes an attack of acute emphysema. There will then be found, not only an increase of resonance, but also an extension of the normal resonant area of the lung, so that the cardiac and hepatic areas may be completely overlapped. In the case of hooping-cough, emphysema is usually present, and the over-distension of the lung may be a cause of dyspnoea, of interference with the pulmonary circulation, of distension of the right side of the heart, and of cyanosis.

In chronic cases of emphysema, such as those associated with asthma, the lungs are over-expanded, the antero-posterior diameter of the chest is increased, the percussion note is everywhere hyper-resonant, and the breath sounds are feeble and prolonged. Chronic bronchitis and dyspnoea on exertion are usually associated symptoms.

Interstitial emphysema may greatly increase the distress accompanying acute pulmonary affections, but is not manifested by any characteristic signs as long as the extravasated air is confined to the pulmonary tissues. If it reaches the mediastinum, it will soon pass into the neck, and be recognized by the diffuse swelling and gaseous crackling on palpation. This complication is a serious one, but is not necessarily fatal.

PATHOLOGY.—Emphysematous patches are usually present in the lungs of young persons dying of acute pulmonary disease, although during life there may have been no means of determining their presence. On the surface of the lung they are easily recognized by their pale or pinkish-red colour, as contrasted with the much deeper colour of the surrounding tissues. They project from the rest of the lung surface in a manner which contrasts more especially with collapsed parts,

which are depressed. Emphysematous areas have a translucent appearance, due to the over-distended vesicles, which may be in groups of a shining pearly aspect not unlike that of tubercles. The chief areas affected are the upper parts of the lungs and the anterior borders, but in cases of patchy consolidation all the lung tissue which is not solid may be found to be emphysematous. In extensive emphysema the volume of the lung appears to be increased, and the anterior surface of the heart may be completely overlapped. In cases of interstitial emphysema the trabeculae supporting the alveoli are distended with air, while the alveoli and air vesicles are compressed and empty. The extravasated air also tends to accumulate in the subpleural tissues.

TREATMENT.—Emphysema is a symptom which does not call for any treatment apart from that of the underlying disease. Vesicular emphysema varies in degree during the course of the illness, and usually passes off entirely without leaving any permanent changes with the subsidence of the original malady. In the interstitial form, due to excess of coughing, epistates should be pushed as far as possible in order to check further mischief and allow of the healing of the ruptured vesicles. When the distension of the subcutaneous tissues of the neck and trunk is very pronounced, one may puncture them with a small trocar and evacuate the air.

PNEUMONIA.

INTRODUCTION.—The pneumonias of early life form a large and very important class of affections. There are considerable difficulties in the way of securing a satisfactory classification. The chief acute varieties usually described are lobar pneumonia, broncho-pneumonia, and tuberculous broncho-pneumonia. Attempts at classification have been made from various aspects—namely: (1) The distribution of the local disease; (2) the bacteriological conditions; (3) the primary and secondary forms.

1. *The Distribution of the Local Disease.*—Lobar pneumonia, as the name indicates, has a lobar distribution, and broncho-pneumonia has a lobular. Clinically, however, it is found that a pneumococcal infection may produce a disease identical in every respect with lobar pneumonia save that the distribution is not lobar, but lobular. Again, in broncho-pneumonia there may be a limitation of the disease to one lung, even to one lobe of the lung, with very few signs elsewhere, and it is only the course of the disease which shows that it is not a case of true lobar pneumonia. A classification, therefore, which is based on the local distribution of the disease cannot be regarded as satisfactory.

2. *The Bacteriological Conditions.*—Lobar pneumonia is in the vast majority of cases due to the pneumococcus, although in some cases a lobar pneumonia may be associated with streptococcal infection or the bacillus of influenza. Broncho-pneumonia, on the other hand, is usually streptococcal or staphylococcal in origin, or due to a mixture of these organisms. Further, it may be due to the pneumococcus, with or without the addition of other organisms. Tuberculous broncho-pneumonia is due primarily to the tubercle bacillus, but other organisms may be present. We find that in most cases there is a mixed infection in pneumonia. Even when one organism predominates and determines the nature of the attack—e.g., the pneumococcus—it does not always lead to the same type of disease. With

a bacteriological classification is very desirable, it is not at present available for clinical purposes.

3. *Primary and Secondary Pneumonias*.—A primary pneumonia is one in which infection attacks the lung directly, and a secondary pneumonia is one in which the pulmonary infection follows on some other local or constitutional disease. This is only another way of saying that in one case the soil is prepared by constitutional debility, and in the other by a definite preceding disease. In both cases the same organisms are at work, and the pathological changes are identical. One cannot recognize any difference in the course of the disease, nor can the two forms be distinguished clinically. The terms "primary" and "secondary" are often puzzling to the ordinary reader, because of the different meaning attached to them by different writers.

It may be stated that the classification of pneumonias in early life is much more difficult than in the case of adults, because the various forms are much more intermixed. An exact classification is at present impossible, but may come later with more definite bacteriological knowledge and the means of applying it clinically. At the same time, the various types can be sufficiently clearly defined by study at the bedside, and the difficulty of classification is more an academic than a practical one.

BRONCHO-PNEUMONIA.

INTRODUCTION.—There is no disease in early life which will meet the practitioner more frequently and test his diagnostic, prognostic, and therapeutical powers more thoroughly than acute broncho-pneumonia. It is most commonly met with, and in its severest forms during the first two years of life. From two to five years of age it is less common, and after the age of five years it is much less frequent and much less severe. Unlike lobar pneumonia, it attacks especially the weak, those infants which are marasmic or have been debilitated by some other illness. It may be ushered in with marked symptoms and run an acute course, or it may be found unexpectedly post mortem, without there having been any symptoms during life to raise suspicion of its existence. It is due to the action of different organisms—pneumococci, staphylococci, streptococci, and probably others. Its virulence, however, is not so much dependent on the particular organism as on the soil in which the organism flourishes. Previous disease and special infections would seem to render the pulmonary tissue of infants especially suitable for a virulent growth of these organisms. The association of broncho-pneumonia with measles and whooping-cough is responsible for the majority of fatal cases in these diseases. Taking all the forms of broncho-pneumonia together, it has been estimated that the death rate is 50 per cent., but this is probably an under-estimate. The disease, however, is not suited to any exact calculation as to the mortality, owing to the fact that it comprises many different forms of infection, and is not like lobar pneumonia, which is due in the majority of cases to one specific organism.

The common descriptive name of this disease is "broncho-pneumonia," but it is known under other titles. The name *catarhal pneumonia* emphasizes two of the leading features—namely, catarrh of the smaller tubes and pneumonic consolidation of the alveolar tissue. The term *lobular pneumonia* refers to the lobular distribution of the disease, as contrasted with lobar pneumonia. *Croupy bronchitis*

has proved a misleading and puzzling name, and had better be dropped. The reference here is to the fine rales or crepitations which appear to be produced in minute tubes, and when these are audible the disease is pneumonia, and not bronchitis only. The presence of these signs and the increasing distress of the patient soon show that the stage of bronchitis has been passed.

The study of this affection in childhood will be rendered clearer if a differentiation on the lines proposed by S. West be accepted. He recognizes the following three clinical varieties of broncho-pneumonia in children:

1. The disease is of gradual onset, and preceded by some affection of the air-tubes; the temperature is hectic in character; the course is prolonged and interrupted by frequent relapses; and the termination is by lysis.

2. The history and symptoms are similar, but the disease is of sudden rather than gradual onset.

3. There is no antecedent affection of the air-tubes; the attack is of sudden onset and short duration; the temperature is persistently high, and there is no marked tendency to relapse; the termination is by crisis; in fact, the clinical symptoms are those of croupous pneumonia, but the lesions those of broncho-pneumonia.

He applies the term "secondary broncho-pneumonia" to the first two classes, and the term "primary broncho-pneumonia" to the third, and regards these as different diseases both clinically and pathologically. He further points out that, while the secondary form stands in close relation with bronchitis and similar affections of the air-tubes, the primary stands in close relation with lobar pneumonia. The acceptance of West's classification will certainly tend to a clearer understanding of the various forms of broncho-pneumonia as they are presented clinically. While the bacteriological proof of the existence of such varieties is at present wanting, there is strong clinical evidence that what he calls primary broncho-pneumonia is really a form of pure pneumococcal infection. This subject will be considered further in connection with Lobar Pneumonia (p. 362).

Ætiology.—The general debilitating factors which have been referred to in connection with bronchitis, such as cold, stuffy rooms, deficient clothing, etc., are equally important in connection with the ætiology of broncho-pneumonia. The great majority of the cases occur during the colder months of the year, as in all forms of pulmonary disease.

The chief ætiological factor is the invasion of the pulmonary tissues by some micro-organism. The predisposing cause is some lowering of the patient's health or vitality. This may be brought about in various ways. Amongst these rickets holds a prominent place. The marasmic infant is always in danger of an attack of broncho-pneumonia. In many cases an attack of influenza paves the way to this disease. Amongst the specific infective fevers, measles and hooping-cough are rendered serious maladies by the frequency of this complication. It is not yet definitely known whether the specific organisms in these affections can directly cause broncho-pneumonia, or whether they merely render the lungs specially susceptible to a superadded infection. Acute gastro-intestinal disorders in infancy, and more especially epidemic summer diarrhoea, will often lead up to an attack of broncho-pneumonia.

The causative organisms may reach the lungs through the upper air-passages. They are always present in the mouth and naso-pharynx, and probably are specially abundant and virulent there in debilitated infants. Or the organisms may reach the lungs through the blood-stream. In the case of epidemic summer diarrhoea it

seems probable, although it is not proved, that some intestinal organism enters the blood-stream and gives rise to broncho-pneumonia.

SYMPTOMATOLOGY.—As contrasted with bronchitis, the gravity of an attack of broncho-pneumonia lies in the fact that not only the bronchi, but also the pulmonary tissues, are involved. There is not only obstruction to the free entrance of air into the lungs, but there is also obstruction to the circulation of blood through the lungs.

The fresh air cannot reach the blood, and the blood cannot reach the alveoli. There is thus a double cause for the dyspnoea which is such a marked feature of the disease. It follows that the strain thrown on the muscles of respiration is great, and the subjects of this affection, weakened by malnutrition or rickets, are often those whose muscles are least able to bear this strain. In this failure of the muscular power lies another cause of dyspnoea. The act of coughing is very necessary in order to clear the lungs and bronchi of the accumulated products of disease, but these patients have often very feeble coughing powers, and what they have are soon exhausted. Hence the bronchial tubes become blocked, patches of collapse develop, and the amount of pulmonary tissue available for breathing purposes is still further diminished.

Other organs are necessarily reacted on from the diseased condition of the lungs. Amongst these the heart is the most important, and its condition must always be carefully observed. The absorption of toxins from the lungs may lead to a state of general toxæmia.

A typical attack of broncho-pneumonia will proceed somewhat on the following lines: After a few days' slight illness, with the signs of bronchial catarrh, a sudden rise in temperature occurs, the breathing becomes more rapid, coughing becomes more constant, and the infant is evidently acutely ill. The dyspnoea increases, a look of marked distress is present, and prostration is evident. The physical signs in the lungs at this time may be disseminated crepitations only, without any definite dulness. The progressive dyspnoea becomes accompanied by cyanosis and drowsiness, the physical signs become more pronounced, scattered patches of consolidation appearing, and often coalescing to form large masses. Then come signs of failing respiratory powers, of failure of the right side of the heart, and of weakening cough. The lungs have become blocked with secretions and distended with blood. Finally come coma, convulsions, hyperpyrexia, and death. This is a common course—a fatal termination after an illness of from seven to fourteen days. On the other hand, there may be after a few days' illness a rapid cessation of all the symptoms and a gradual clearing up of the pulmonary signs. Or, again, a short attack of a week or ten days may be followed after an apyrexial interval by a relapse of the acute symptoms, with signs of the involvement of fresh areas in the lungs. This form of the disease, characterized by relapses, may persist for from eight to ten weeks, and may eventually terminate in recovery, even after one had almost entirely abandoned all hope of such an issue.

The onset of an attack is usually gradual, and the child does not appear to be acutely ill until the temperature suddenly rises. This is a marked contrast to the abrupt onset of acute lobar pneumonia in a previously healthy child. In some cases of broncho-pneumonia the onset may be sudden, accompanied by vomiting, convulsions, or other evidences of cerebral disturbance. Such an onset, although the physical signs may point to broncho-pneumonia, suggests that the primary organism at work is the pneumococcus. Again, the onset of broncho-pneumonia

during the course of another illness may come suddenly without any precursory signs. As a rule, however, the early stages are marked by the signs of bronchitis only, with slight cough and fever.

Broncho-pneumonia is a pyrexial affection, the temperature as a rule showing a considerable daily fluctuation, tending to reach its maximum in a week or ten days, and in favourable cases terminating by lysis. In fatal cases the temperature often rises to 107° or 108° F. before death. The height of the temperature is important, as marked pyrexia indicates severe illness, and is exhausting to an already debilitated infant. An average range of temperature in this disease is from 102° to 105° F. When relapses occur, the temperature shows a gradual

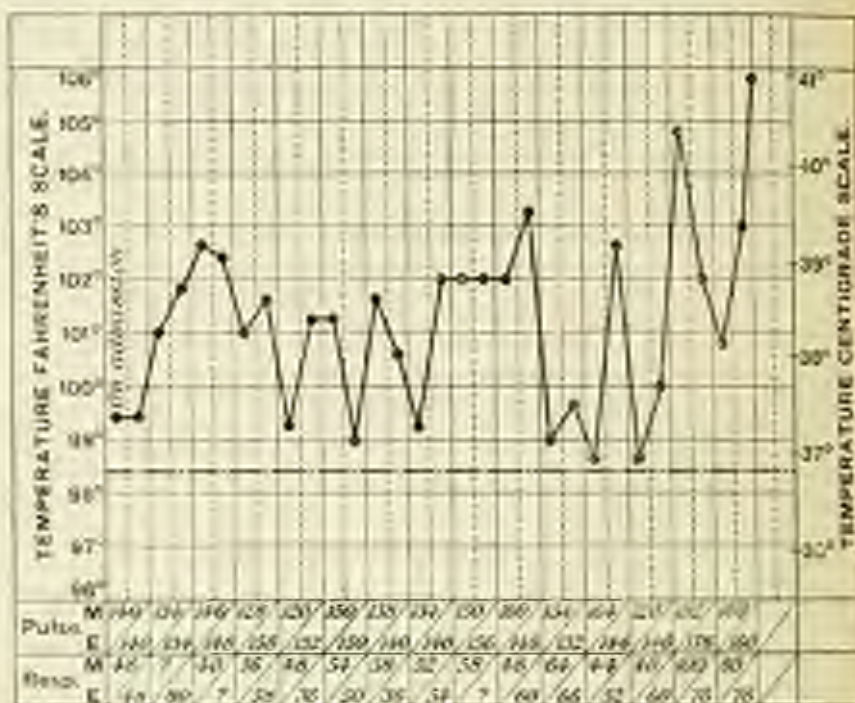


FIG. 25.—MALE, AGE SIX MONTHS: DAILY CHART OF RECTAL TEMPERATURE; DEATH.

rise for some days, and then a gradual return to normal. The temperature chart corresponding with the physical signs of the involvement of fresh areas is the high, and then their gradual clearing up. In some instances there may be no rise of temperature at all, and this occurs mainly in the case of extremely debilitated subjects. Thus Melville Dunlop found that the temperature was actually sub-normal through the whole course of extensive broncho-pneumonia in three atrophic infants. While the natural termination of the pyrexia is by lysis, cases occur of termination by crisis, after a clinical course which points to broncho-pneumonia and not lobar pneumonia. The termination by crisis and rapid recovery suggest strongly that the attack was due to infection by the pneumococcus only, the inflammation having a diffused instead of a strictly localized distribution.

The pulse-rate is always increased, running up rapidly to 120, 140, 160, or even higher. The factors that influence the pulse-rate are the temperature, the extent of the pulmonary inflammation, the amount of general toxæmia present, and of course the state of the heart.

Much information can be gained from observation of the nature and rapidity of the respiration when the patient is quiet. Coughing, crying, and restlessness, will entirely alter the conditions of respiration. The respiratory-rate is always increased, and mounts up quickly to 40 or 60 per minute. Higher rates may be present, even up to or above 100 per minute. It is probable that these higher rates are associated with some disturbance of the respiratory centre rather than due



FIG. 26.—FEMALE AGED ONE YEAR; DAILY CHART OF BRONCHO-PNEUMONIA FOLLOWING MEASLES; DEATH.

directly to the pulmonary affection. The pulse-respiration ratio may be found to be markedly altered, becoming 3 to 1 or 2 to 1 instead of the normal 5 to 1 or 4 to 1. As already stated, dyspnoea is a marked feature. The normal diaphragmatic breathing of infancy is reinforced by costal breathing, and gradually all the accessory muscles of respiration are brought into play. The child has a distressed look, and this distress is due to the special respiratory efforts. One can see this muscular over-action in the dilatation (inspiratory) of the alæ nasi, the descent (inspiratory) of the larynx, the raising of the clavicles and sometimes of the shoulders, and the strongly acting sterno-mastoid and scalene muscles. The chest has an appearance of being over-expanded, and this, as Harry Campbell has pointed out, is characteristic of all forms of dyspnoea. The first effort at the onset of dyspnoea is to get the chest fully expanded, and the tendency is for this distension to persist

as long as the respiratory muscles are over-acting. If cyanosis develops, the dyspnoea may become of such a character that the child, if old enough, may sit up in order to use his muscles more powerfully—a condition of orthopnoea. In some cases the dyspnoea may be as great as in laryngeal diphtheria, when tracheotomy is indicated, and, further, the physical sign of obstruction may be very similar. There may be great retraction of the soft parts below the ribs and above the clavicles, but this is due to the failure of the more or less consolidated lung to expand sufficiently to meet the thoracic expansion, and to the bronchial obstruction. While dyspnoea is present in all cases, its severity will vary greatly, and the factors which determine this are three in number: first, the amount of obstruction in the bronchi, and more especially the bronchioles; secondly, the extent of alveolar tissue which is thrown out of use by consolidation or collapse; and, thirdly, the extent to which the pulmonary circulation is blocked.

Cough is usually present through the whole course of the illness, sometimes very continuous, but more often intermittent. It may be loose from the abundant secretion present, or dry from the irritation caused by the pulmonary inflammation. Its force will vary with the strength of the patient, but as the vital powers diminish and the brain becomes clouded the cough becomes weaker, and may be entirely absent. The appetite is usually lost, and the digestive secretions are much diminished. Thirst is frequently very marked. Sweating may be present, and is to be encouraged, but as a rule the skin is dry and hot. Restlessness and sleeplessness are common when the disease is fully developed. The effort of crying is too great for the infant, with its respiratory powers already fully occupied, but a low moaning may be present.

Physical Signs.—Inspection of the chest and breathing will often enable one to form a provisional diagnosis. Next in importance comes auscultation, and then percussion. The auscultatory signs are often puzzling from their multiplicity and variability. As a rule both lungs are involved, although by no means symmetrically. In some cases the physical signs may be limited to one lung. The signs vary from day to day, sometimes from hour to hour, and in listening for them it is advisable to examine both when the child is breathing quietly and breathing deeply, as when crying or coughing. In the former case the finer crepitations are heard more clearly, although the areas of bronchial breathing may be overlooked; while in the latter the deep breathing brings out the bronchial element, and crying shows the altered state of the vocal resonance.

In the early stages the breathing may be harsh, vesicular, with rales and coarse râles formed in the bronchi. Soon begin to appear the characteristic fine crepitations formed in the bronchioles and alveoli. They are most abundant, as a rule, at the bases posteriorly, but tend to have a patchy distribution over the lungs generally. This condition of harsh breathing with crepitation may persist through the whole attack without further evidences of consolidation. In other cases patches of bronchial (or tubular) breathing may appear, and may extend, forming considerable areas, even to the involvement of the whole of one lobe. Sometimes the bubbling and fine râles may be so numerous as to conceal entirely the character of the breath sounds. Over the areas of bronchial breathing the vocal resonance is usually altered to a bronchophonic character. Sometimes the typical signs of a cavity in the lungs are present, but it must be remembered that these—namely, a cracked-pot note, amphoric breathing, and coarse râles—are often associated in children with pneumonic consolidation only.

In the early stages percussion shows little change in the resonance of the lungs. Even when areas of collapse or consolidation are present, as shown by the bronchial breathing, there may be little appreciable change in the percussion note. The reason is that there are still so many of the alveoli remaining unaffected amongst the scattered patches of consolidation, and some even emphysematous, that resonance is still maintained. It is not until considerable areas of consolidation are formed that impairment of resonance can be definitely made out, and even then the dulness is seldom so marked as in lobar pneumonia. While small areas of consolidation seem to vary rapidly, to change from day to day, the larger areas are much more persistent. They form slowly, and they are slow in clearing up. The presence of large areas of consolidation, as determined by physical signs, indicates a severe form of disease which will be accompanied by serious interference both with the pulmonary and the cardiac functions.

While pleurisy may occur during an attack of broncho-pneumonia, it is by no means a frequent condition, as may be judged by the absence of pain and friction sounds. Signs of pleuritic effusion are not usually present, and empyema is much less frequently met with than in the case of lobar pneumonia.

The heart and circulation generally are usually markedly affected by the progress of broncho-pneumonia. Owing to the congestion of the pulmonary vessels, the circulation in the lungs is carried on with difficulty, and the right side of the heart becomes embarrassed and dilated. Marked epigastric pulsation may be detected, along with an extension of the cardiac dulness to the right. Increasing difficulty in carrying on the pulmonary circulation will be shown by cyanosis and a progressive enlargement of the liver. The continued pyrexia and the presence of toxins in the blood will also have an injurious effect on the cardiac muscle generally, so that weakness of the left ventricle develops, the sounds weaken, and the pulse becomes small, feeble, and rapid. Owing to the cardiac failure, the brain becomes clouded, the nervous centres become less active, the voluntary power which maintain respiration and coughing are dulled, and the pulmonary condition becomes progressively worse.

One will often find, especially in the case of young infants, that the medullary centres appear to be involved in the toxic condition, as indicated by irregular forms of breathing or attacks of cardiac syncope. The breathing may be curiously cyclic, periods of complete intermission being followed by some deep respirations. Sometimes in an attack the patient will stop breathing and turn blue; the pulse cannot be felt, but the heart may be beating slowly. For a few minutes death seems to be imminent. A fatal result may follow, or a few gasping inspirations may be made, and the patient gradually recovers. In other cases the heart seems to be primarily affected, and extreme pallor comes on suddenly. Although these attacks may be repeatedly recovered from, they must be regarded as indicating a very serious complication and calling for stimulation.

Disturbances in the alimentary tract are common, and may increase the gravity of the disease. Diarrhoea is often present, and may rapidly reduce the patient's strength. Flatulent distension of the abdomen, associated with intestinal catarrh, may seriously impede the action of the diaphragm and increase the dyspnoea.

DIAGNOSIS.—Attention will at once be directed to the lungs from the cough and respiratory embarrassment which are present. In the early stage the diagnosis from bronchitis may be difficult or impossible, because the signs may be those of bronchitis only. With the spread of the disease to the bronchioles and the

involvement of the alveolar tissues, one can observe that the illness is more severe, the pyrexia greater, and the dyspnea more marked, than in ordinary bronchitis. The fine crepitations of broncho-pneumonia are not met with in bronchitis, and in the latter disease one does not find areas of consolidation with bronchial breathing and bronchophony.

The diagnosis between broncho-pneumonia and lobar pneumonia will be considered later (see p. 379). A differential diagnosis between tuberculous broncho-pneumonia and the non-tuberculous form can only be made clinically by finding tubercle bacilli in the sputum. There is a form of pulmonary tuberculosis which may simulate broncho-pneumonia—namely, acute miliary tuberculosis with extensive involvement of the lungs. The signs here are marked fever, great dyspnea, cyanosis, and many fine crepitations audible over both lungs. All these signs may be present in broncho-pneumonia, so that it may be impossible during life to distinguish between the two.

In the case of a weakly infant one is not surprised to meet with broncho-pneumonia, but in the case of an apparently healthy child some explanation seems to be called for. It may be that the attack marks the onset of measles, for broncho-pneumonia may develop before the rash appears as well as at any subsequent stage. Again, it may be that whooping-cough is the cause of the pulmonary trouble. During an attack of broncho-pneumonia the characteristic hoop may entirely disappear, and unless the practitioner is on his guard the primary disease may escape his notice, and may not have been recognized by the attendants. A third explanation may be that the disease is pneumococcal in origin. The pneumococcus seems to have the power of attacking successfully the lungs of healthy children, while the streptococci and staphylococci seem to require a soil weakened by disease. In other cases the explanation may lie in the fact that a latent tuberculosis has suddenly involved the lung, producing an acute tuberculous broncho-pneumonia.

PATHOLOGY.—Broncho-pneumonia is a disease in which all the component tissues of the lungs are involved. The bronchi are swollen and softened, the lumen is filled with catarrhal products, and the peribronchial tissues may show a great exudation of leucocytes. The bronchioles are specially involved. They are inflamed and swollen and softened, and contain pus or mucus. The resulting changes in the bronchioles have been described by Sharkey as "acute bronchiectasis," and by J. Møller as "bronchiolodectasis." The latter states that the bronchioles, which are unsupported by cartilage, become dilated, owing to a stretching of the elastic and muscular layers of the walls. This stretching is caused by the act of coughing, and rupture of the wall may take place, leading to an extravasation of purulent exudate into the surrounding tissues. Numerous purulent cavities may be formed, and produce the condition which is known as "honey-comb" lung (see Fig. 33, p. 388). The septa of the lung are inflamed, swollen, and crowded with leucocytes.

The alveoli are filled with leucocytes or blood or catarrhal products, but not with fibrin, as in lobar pneumonia. Through the lung tissues there may be scattered areas of hæmorrhage or localized abscesses, and the latter may lead to localized gangrene. Areas of collapse and of emphysema may be mixed up with areas of consolidation from alveolar inflammation. Inflammation may extend to the pleura, causing a pleuritis, and the glands at the root of the lungs are usually enlarged.

The disease spreads along the smaller bronchi, and this determines the lobular

distribution of the inflammation. It spreads irregularly over the lungs, but there is a natural tendency for it to gravitate towards the bases of the inferior lobes. The occlusion of a bronchiole may lead to collapse of the alveoli in the area supplied; and while this collapse may only be temporary if the air-passage is restored, it may, on the other hand, be altered into inflammatory consolidation by the action of the predominant organism which reaches the collapsed portion through the interstitial tissues or by direct extension from the bronchiole. The extension of the disease is often brought about by the inability of the child to clear the lungs, the *pus* or *mucopus* secreted being coughed out of one bronchus and passing into another.

As regards the bacteriology, it would appear that pneumococci, streptococci, and staphylococci, are the most common causative organisms, sometimes separately, but more often in association.

The following statistics by Eyre are based on an examination of ninety-five cases of acute lobular pneumonia and broncho-pneumonia in children:

Total number of cases yielding pure cultivations = 37.

Total number of cases yielding mixed cultivations = 82.

Micro-organisms isolated.	In Pure Cultures.	In Association with One or More Pathogenic Bacteria.
<i>Diplococcus pneumoniae</i>	22 times.	39 times.
<i>Streptococcus pyogenes longus</i>	23 "	29 "
<i>Staphylococcus pyogenes aureus</i> and <i>albus</i>	8 "	25 "
<i>Micrococcus catarrhalis</i>	1 "	12 "
<i>tetragenus</i>	0 "	7 "
Bacillus of Friedländer	4 "	7 "
<i>Bacillus influenzae</i>	3 "	15 "
— <i>peritumens</i>	0 "	2 "
— <i>pyogenus</i>	0 "	1 "
— <i>typhosus</i>	0 "	1 "
— <i>diphtheriae</i>	0 "	3 "

There is no evidence at present that an acute specific fever like hooping-cough or measles can directly induce broncho-pneumonia in a healthy child—*i.e.*, through the action of the specific toxin or organism. At the same time there is evidence that the organisms of broncho-pneumonia in these fevers have acquired a special virulence, and even a special infectivity.

Prognosis.—In a well-marked case of broncho-pneumonia the prognosis is always serious, and usually very serious. It must be within the experience of every clinician to have met with a considerable succession of cases every one of which proved fatal, so that he was almost reduced to despair as to the value of any treatment. Various factors have to be considered in forming a prognosis.

1. The state of the patient's general health and nutrition. A weak or marasmic infant usually succumbs to an attack of broncho-pneumonia. The presence of general disease like rickets, tuberculosis, or syphilis, will tell against the prospect of recovery.

2. The age. Under the age of twelve months the mortality is very high, probably from 70 to 90 per cent. In the second year of life it is distinctly less, and the prospect of life improves rapidly year by year, until after the age of five years the disease becomes much milder.

3. A *history of previous lung disease*. If the lungs have been weakened by previous disease, such as bronchitis, the form of inflammation is more likely to be severe.

4. The presence of an *acute specific fever*. As a complication of measles, hooping-cough, diphtheria, influenza, or acute infantile diarrhoea, broncho-pneumonia seems to develop a particularly virulent character.

5. The *type of disease*. Relapsing broncho-pneumonia usually terminates fatally from sheer exhaustion of the patient, but even after an illness of this type lasting for months recovery may take place. In the toxic form with great prostration and stupor, the prognosis is bad even although the physical signs may be few.

6. The *physical signs*. When both lungs are extensively involved or when large areas of consolidation are present, conditions which point to great limitation of the respiratory surface, the prognosis is bad.

7. The *form of organism present*. This cannot always be determined, but it would appear probable that the prognosis is best in the pneumococcal form, and worst in the streptococcal.

8. *Complications*.—The onset of diarrhoea, or of convulsions, or of marked cyanosis from cardiac failure, renders the prognosis very grave.

TREATMENT—Preventive Treatment.—Much can be done in the way of preventive treatment if one bears in mind that weak infants are extremely liable to this disease if exposed to any prolonged chilling of the surface of the body. An attack of what is apparently simple bronchitis in a delicate infant may rapidly develop into broncho-pneumonia in the absence of careful treatment.

The care of the mouth, nose, and throat, is extremely important, in order that a condition of sepsis may not develop. In the case of a delicate infant this is easily induced, and the ordinary micro-organisms take on a pathogenic function. It is more especially in connection with the specific fevers that the necessity of preventive treatment and the opportunity of carrying it out are presented. It is not going too far to say that the mortality from measles and hooping-cough could easily be reduced by one-half were means generally taken to prevent the occurrence of broncho-pneumonia. These diseases are but too often regarded by the lay public as merely feverish attacks, and when the temperature is normal the patient is regarded as well. He is accordingly allowed to be up and running about, possibly out of doors, and the result is an attack of broncho-pneumonia. In the case of these infective fevers the patient should be kept at rest for some weeks after the subsidence of all active symptoms, more especially bronchial symptoms, and until the medical attendant is convinced that the danger of pulmonary complications is over. Such precautions are absolutely necessary during the colder months of the year in this country, while in the warmer months rest should still be maintained although the patient may be allowed out of doors.

The Question of Infectivity.—A case of primary broncho-pneumonia does not show any marked degree of infectivity. It may be treated in a children's ward of a hospital without special risk to other patients, but in private practice it is advisable to keep other children out of the patient's room. In the case of secondary broncho-pneumonia accompanying measles or hooping-cough, it is desirable to keep the patient isolated as long as the pulmonary trouble continues. The specific fever would appear to add to the lung affection, not only a special virulence, but also an increased degree of infectivity. In a measles ward there may be an outbreak of

broncho-pneumonia of a virulent type, and evidently of a contagious character; while in an ordinary children's ward a case of broncho-pneumonia following measles may lead to an outbreak of other cases of pneumonia, usually of a very severe type. For these reasons it is well to regard and treat all cases of broncho-pneumonia accompanying or following measles or whooping-cough as distinctly infectious.

The *General Treatment* of the disease proceeds on the same lines as in other febrile illnesses in children. Rest in bed, the use of sufficiently warm body and bed clothing, and the maintenance of a steady temperature (82° F.) in the room, are essential. At the same time an abundance of fresh air must be supplied, as in this affection the patient has need of all the fresh air he can get, and stuffy rooms form the worst possible environment. If possible, two rooms should be in use, one for the day and the other for the night, so as to secure more thorough ventilation. The "open-air" treatment of broncho-pneumonia has proved very valuable, but is not advisable in cold and damp weather. Provided the weather is temperate and a place in the open air is available in which the baby's crib can be protected from wind, this method of treatment has in numerous cases proved life-saving. It has appeared to be specially useful in the protracted or relapsing cases, when no progress may have been achieved until the infant was transferred to a balcony and kept there night and day. Improvement as regards sleeping, appetite, coughing, and the pulmonary conditions, is often very striking. Means must of course be taken to insure that the child's body temperature is maintained by the use of plenty of blankets and hot bottles. A very interesting result has been obtained in this connection by Claude Keer, who treated a series of cases of whooping-cough by the open-air method throughout the whole illness, and found a marked diminution in the proportion of cases complicated with broncho-pneumonia, as compared with those treated in the wards of the hospital under the ordinary atmospheric conditions. A good many years ago the present writer was much impressed by the effect of the open-air treatment in forty cases of measles treated on board ship in a voyage between Egypt and Australia. The patients were kept on deck, protected from the sun and from cold draughts at night, and all made an excellent recovery, without the occurrence of a single case of severe pulmonary trouble. From my own experience of the balcony treatment of broncho-pneumonia in hospital cases, I can state that no harm has ever resulted, and several cases which while in the ward seemed to be going downhill began to mend as soon as the outside treatment was adopted. The chief difficulties in private practice are that the open-air method outrages all the domestic canons of treatment in this affection, the poultry and steam-kettle still holding sway, and the securing of the skilled nursing which is absolutely essential.

The older method of the steam tent and kettle has fallen into disuse, probably because it was used to excess. At the same time it may be employed with advantage for the relief of certain symptoms, such as spasm of the bronchi, laryngitis, and persistent irritable cough. The steam-kettle should not be used for more than half an hour at a time, and only for definite indications as above, for the disease itself is not benefited, and the patient may be much weakened by breathing continuously a hot, moist atmosphere. Melville Dunlop recommends that the upper half of the crib be surrounded with a tent, open in front so as to admit of the free access of air. Inside the tent are hung towels wrung out of a solution of one part of eucalyptus-oil to five parts of water. He is convinced that the evaporation of the moisture and the volatilization of the oil have a soothing effect on the

inflamed mucous membrane and greatly diminish the cough, and thinks it possible that the antiseptic action of the oil may tend to prevent the spread of the pneumonia to fresh portions of the lungs.

During the acute stage the food should be fluid, digestible, and given in small quantities every two or three hours. Gastric and intestinal complications are common and serious, and are best guarded against by dietetic measures. If the appetite is lost, it is much better to give weak fluid food than to produce gastric trouble by feeding up with concentrated patent foods. Albumin-water, whey, peptonized milk, and veal or mutton soup, may be given at first; and if the appetite is good, plain milk and barley-water, bread and milk, Benger's or Mellin's food and milk, and thin milk puddings, may be allowed. Water or barley-water can be given freely, and the more one can act on the skin and kidneys by the use of plain fluids, the better is the prospect of clearing the poison out of the system. As regards the fever, it is not often that a very high temperature is prolonged, and as the fever is usually of an intermittent type, no acute interference is called for in the way of anti-pyretic treatment. The use of anti-pyretic drugs is distinctly injurious. When a high temperature is associated with marked restlessness, delirium, or sleeplessness, a hot bath or a hot pack will often prove most beneficial. Hot sponging and hot packs are soothing and stimulating, and are of very great value in this affection. They should be given with as little disturbance of the child as possible. In the case of these young patients, it is to be remembered that their energies are fully occupied in carrying on the laboured respiration, and that therefore their strength should not be taxed in other ways. Carrying babies about, prolonged bathing operations, and fussing about constantly with positions, will tend to exhaust the powers of the patient, who should be resting quietly in bed.

Coughing is often troublesome, and in its treatment it is important to determine first the seat and nature of the irritation. If bronchial irritation without much secretion is present, as often happens in the early stage, steam inhalations for ten or fifteen minutes, hot fomentations to the chest for an hour, and hot drinks are indicated. At this stage one may expect benefit from such drugs as citrate of potash, carbonate of ammonia, and solution of acetate of ammonia. If the bronchial secretion is too free, with loose, bubbling rales all over the lungs, we follow another line of treatment. The emptying of the blocked tubes may be aided by partially inverting the child, by resting him with the head depressed, or by giving an emetic, such as 1 grain of sulphate of copper in a drachm of water. Emetics must not be used if the child is very weak, or at a late period of the disease, as they are too exhausting. Excessive bronchial secretion may be checked by the use of tincture of belladonna, 5 to 10 drops every four hours, or the solution of atropine in $\frac{1}{4}$ to 1 minims doses. Undoubtedly, belladonna is very useful in some cases of broncho-pneumonia in checking secretion and coughing, and it seems at the same time to stimulate the respiratory centres and affect favourably the whole course of the attack. Cassa has recommended the use of somewhat large doses of extract of belladonna $\frac{1}{2}$ grain every three or four hours. If the nature of the cough and the signs in the lungs suggest that irritation of nerve filaments through inflammatory action is the source of trouble, paregoric in 5 to 10 drop doses, or small doses of heroin or morphine, may be given.

In the case of broncho-pneumonia, we may rest assured that sooner or later the heart will suffer from the pulmonary obstruction, the prolonged fever, and the general toxæmia. With the view of easing up the heart, 2 to 3 drops of the

inctures of digitalis and *nux vomica* may be given thrice daily at an early stage of the attack. At the same time it must be kept in mind that digitalis has but a limited action in cases of acute febrile disease with cardiac weakness from toxæmia. Attacks of cardiac failure with dilatation of the left ventricle and irregular action may be treated with Nativelle's digitalin granules or *strophanthin* (gr. $\frac{1}{32}$) hypodermically. The right side of the heart suffers most from the pulmonary obstruction, and when it is dilating, as shown by cyanosis, increasing dyspnoea, and enlargement of the liver, depletion is called for. The application of three or four leeches over the hepatic region, followed by a hot fomentation to encourage further bleeding, will usually be found to produce marked relief. Free rubbing over the bases of the lungs posteriorly is indicated when the congestion and consolidation are marked in those regions. In very severe cases with marked cyanosis it may be advisable to try venesection, removing from 5 to 6 ounces of blood, so as to relieve the blocked cardio-pulmonary circulation. Stimulation must be pushed boldly in this disease. Strychnine and brandy stimulate both pulmonary and cardiac action, probably through the nervous system, and are invaluable in tiding a patient over an attack of broncho-pneumonia. Strychnine may be used in doses of from $\frac{1}{2}$ to 1 drop of the *liquor strychnine* every four hours, at first by the mouth, and later by hypodermic injection, if improvement has not followed. Brandy may be given in doses of 10 to 60 drops every three hours, according to the age and condition of the child. Its sedative action on the restless patient is often very marked. Oxygen bubbled through alcohol is of value in certain cases of pronounced cyanosis, but it is impossible to say beforehand whether it will improve the colour and breathing of the patient, which are the indications by which its value is to be tested. If there is much pulmonary catarrh present, it has sometimes seemed to have the effect of increasing the pulmonary secretion. We sometimes find that there is an increasing amount of respiratory distress, with dyspnoea and rapid breathing, which does not appear to depend directly on the state of the lungs or the heart. We have here probably to deal with a failure of the respiratory centre from toxæmia. In such cases atropine, as a direct respiratory stimulant, may be combined with the strychnine in doses of $\frac{1}{64}$ grain hypodermically every four hours for a child of three years.

There is a form of treatment which is not to be recommended, but which is sometimes carried out when a case of severe broncho-pneumonia is suddenly spring upon the young practitioner. He sees a child cyanosed, gasping for breath, with the lower ribs sinking in at every inspiration, and with all the indications for tracheotomy, save one—namely, stridor. Sometimes in his hurry this important sign is overlooked, and tracheotomy is performed, without, of course, affording any relief, as there is no obstruction to the entrance of air above the trachea.

Certain local complications call for notice. The mouth and naso-pharynx should be kept as free as possible from micro-organisms, which flourish during the illness and may be the source of fresh infection. A persistent nasal discharge and an infection of the middle ear can often be traced to a neglected condition of the mouth and naso-pharynx. The use of warm antiseptic lotions containing boroglyceride or Iod or Listerine will prevent these complications.

The course of treatment may be a long one, and the period of convalescence will also vary with the duration of the acute attack or attacks. Each relapse is to be treated on the same lines as the original one, and, as the patient has been already reduced in strength, special care must be taken to avoid anything which would

increase this weakness. The partiality of relatives to feeding-up methods, patent foods, and medicines, must be kept in check. When convalescence has been much hindered, more nourishing foods may be given, guided by the increase of appetite, and cod-liver-oil with malt or hypophosphites will prove useful. The complete resolution of the lungs may proceed but slowly even long after the fever and all active symptoms of disease have subsided. It is at this stage that a change of air to the hills or seaside will often complete the process of repair, which is so necessary if chronic pulmonary trouble is to be avoided.

When recovery takes place, it may be complete, usually after a somewhat prolonged convalescence, and the condition of the lungs may be again quite normal. On the other hand, in many cases the lungs never clear up entirely, and certain sequelæ remain. Of these, perhaps the most common is enlargement of the glands at the root of the lungs, which are thus rendered extremely liable to become a suitable nidus for the development of the tubercle bacillus later on. The bronchi may never recover their tone, and a condition of chronic bronchitis with dilatation of the bronchi may supervene. When a whole lobe or some other large area of the lung has been consolidated, resolution is often incomplete, and a state of chronic fibroid pneumonia is established. In some cases the damaged lung is attacked by the tubercle bacillus, and the disease then becomes chronic pulmonary tuberculosis. The treatment of these sequelæ will be found in other sections.

LOBAR PNEUMONIA.

INTRODUCTION.—Lobar pneumonia is a disease which is common at all periods of childhood. Some writers deny its frequency during the first two years, and more especially in the first year. It is, however, much commoner in the first year than is usually stated. The difference of opinion which exists on this subject arises from the difficulty which exists in distinguishing between broncho-pneumonia and lobar pneumonia by the physical signs only. If we use the term "a pure pneumococcal affection of the lungs" instead of "lobar pneumonia," and if we take the whole symptomatology of the disease instead of the physical signs only, this difficulty would disappear, and lobar pneumonia would be found to occur frequently during the first year of life. As matters stand, the published statistics of broncho-pneumonia and lobar pneumonia in children are quite unreliable, and vary according to the personal view of the collector. As it occurs in childhood, lobar pneumonia may run a course identical with that seen in adult life as regards the fever, the physical signs, the duration, and the whole course of the illness. On the other hand, there is more variety about the disease in childhood. The brunt of the infection may appear to fall on some other organ than the lung, and direct attention away from the primary source of illness. The physical signs may be so latent or so delayed in appearance as to raise a doubt as to whether the disease actually is lobar pneumonia. The complications which accompany it follow lobar pneumonia in childhood are much more common and more varied than in later years. Speaking generally, one may say that the younger the child the more likely is the pneumococcus to spread to other tissues of the body and produce local lesions outside of the lungs. An ordinary attack is usually well borne, and is recovered from rapidly. As contrasted with the disease in adult life, the cardio-pulmonary distress is of a comparatively trifling character through the greater part of the attack.

While many names have been applied to this disease in the past, such as "crepans pneumonia," "fibrinous pneumonia," etc., the term "lobar pneumonia" is now universally adopted. Perhaps, however, this term will in time be changed for one which emphasizes less the pulmonary affection, and expresses better the fact that it is a pneumococcal fever with local lesions in the lungs. There is much to be said for the inclusion of this affection amongst the general fevers, as has been done in some textbooks, and not amongst the special diseases of the lungs.

Lobar pneumonia is neither epidemic nor contagious in the ordinary sense of the term. In a well-ventilated hospital ward one does not see the disease spread to other patients. Certain cases have been recorded in which several members of the same family were attacked simultaneously or in rapid succession—a fact which points to a certain degree of contagiousness, which is probably exaggerated by unhealthy surroundings. It is by no means rare for the same child to suffer from more than one attack of lobar pneumonia, even within a year, so that an attack does not produce immunity for any length of time, and may even appear to predispose the system to fresh infection.

ÆTIOLOGY.—Lobar pneumonia is due to an infection of the lung by the pneumococcus. It is true that other organisms may produce an inflammation of the lungs which is lobar in distribution—e.g., the streptococcus, and possibly the influenza bacillus—but the typical disease, running a typical course, is due to pneumococcal infection.

Little is known about the predisposing circumstances. The organisms are probably always present in the mouth and naso-pharynx, and are more numerous, and therefore more dangerous, when these are in an unhealthy condition. Certainly the cold months of the year, and more especially the early spring, are responsible for the greater number of attacks. So that cold weather and exposure to cold may be regarded as important ætiological factors. Early infancy is no bar to pneumococcal infection of the lungs, which may occur even during the first three months of life. As contrasted with other pulmonary affections, a striking feature is the liability of well-nourished, healthy-looking children to be attacked. When a healthy infant is seized with what is manifestly pulmonary inflammation, the probability is that this will turn out to be lobar pneumonia rather than bronchopneumonia. In the course of, or convalescence from, infective fevers—e.g., measles and whooping-cough—while broncho-pneumonia is by far the commonest form of pulmonary inflammation, the occurrence of lobar pneumonia is by no means rare. In some cases a history of falling downstairs or other form of injury within twenty-four hours of an attack seems to suggest that traumatism may directly predispose the system to this disease.

Influenza is often complicated with lobar pneumonia. When influenza is epidemic, the association is usually clear, and the combination may lead to a very severe type of pulmonary inflammation. At other times it has too often been the custom to assume that pneumonia developing with unusual symptoms or running an unusual course was influenzal in origin. This assumption is in many cases groundless, and lobar pneumonia has sufficient variety in its own manifestations without calling in a supposititious influenza to account for them when they seem to be outside the ordinary course.

SYMPTOMATOLOGY.—As lobar pneumonia in childhood may pursue a course identical with that in adult life, it will not be necessary to enter fully into all the

ordinary signs and symptoms. We shall rather emphasize the characteristics of the disease as it occurs in childhood.

In the early stages of the disease the diagnosis is by no means easy, and more is to be learned from the history of the onset and the appearance of the patient than from an examination of the lungs. Especially is this the case in infants, for an infant lying quietly in its mother's arms may show definite and unmistakable symptoms of pneumonia. This same infant, after being undressed for an examination of the chest, may become such a terrified, shrieking, kicking mass of humanity that one can learn nothing. In many cases in the early stages there are no abnormal physical signs present in the lungs.

Lobar pneumonia usually begins as well as ends with a crisis. The sudden onset, with the development of acute symptoms, is quite as striking as the sudden termination, with the cessation of acute symptoms. A child has been apparently in good health, when suddenly, or after slight malaise, an attack of vomiting comes on; he shivers and complains of feeling cold, for rigors are not characteristically present in childhood; and there may be superadded epistaxis, or severe headache, or convulsions, or diarrhea. The important thing to note is that from the onset the child is definitely and visibly ill, and that within a few hours the skin is pungently hot, the face is flushed, the temperature has risen considerably, and the breathing is increased in frequency. According to the nature of the infection and the state of the system, he may become restless and delirious, or may be plunged into a drowsy or stuporous condition. This early stage is not usually under medical supervision, and it is important, therefore, to get a statement as to the exact condition at the onset and for the following twelve hours. In a certain number of cases, and more especially when another disease is already present, the onset may not be marked by any definite symptoms, and the insidious nature of the attack may suggest rather enteric fever or general tuberculosis than pneumonia.

In the early stages—i.e., during the first four or five days—the breathing is specially worthy of careful observation, as it presents many changes which are more characteristic than in the pneumonia of adult life. It is very important to observe it when the child is lying quietly, as the least emotion or physical disturbance brings in voluntary movements of respiration, which alter the character. Pure pneumonic breathing in childhood presents the following characteristics in the early stages:

1. The breathing is very frequent from the onset, increasing in rapidity out of proportion to the rise in temperature. The number of respirations may reach 40, 60, 80, or even 100 per minute.

2. In character the breathing is shallow or superficial, and from this fact the increased frequency may easily be overlooked. The bystander sees nothing in the face or in the chest movements of the patient to suggest tachypnea; but if he places a hand on the abdomen, the rapid, shallow breathing will be at once detected.

3. The breathing is chiefly abdominal, slightly lower costal, and scarcely at all upper costal. In other words, it is natural breathing accelerated. If upper costal movement is at all marked, one may be quite sure that there is some other cause than a patch of pneumonic consolidation.

4. An inverted type of respiratory rhythm is frequently present. The normal respiratory cycle is inspiration, expiration, pause, the pause being the imperceptible termination of the expiration. Very frequently in pneumonia this cycle is inverted, and becomes expiration, inspiration, pause. There is a distinct abnormality

about this process, both the expiration and the pause being accompanied by muscular action, and not merely passive, as in ordinary breathing. During expiration the air is forcibly driven out of the chest, a rapid inspiration follows, and during the pause the air is, as it were, held up in the chest, the vocal cords being closed. This phenomenon may occasionally be seen in children under other conditions, as, for example, after a fit of crying, but in pneumonia it may persist for hours or days. It is by no means, however, a constant symptom either in all cases or in any one individual patient; but when present it is of very great diagnostic value in at once suggesting pneumonia in a case which may have been obscure. In many cases of this inverted rhythm the breathing is quite quiet; but if the expiration is specially forcible, there may be audible "panting" or "grunting," or a nasal "whistle." This inverted type of breathing may be noticed on inspection of the patient, or it may first attract attention during auscultation. If one is auscultating a consolidated part of the lung, one may be struck by the fact that the bronchial or tubular element is louder during inspiration than expiration—i.e., the reverse of what usually occurs. The fact, of course, is that with the inverted type of breathing the apparently inspiratory sounds are really expiratory, and one has been misled by the altered position of the pause in the respiratory cycle.

Associated with this inverted rhythm there is a peculiar action of the *alae nasi* which is not seen in any other affection. A slight inspiratory dilatation of the nares is common in childhood under any excitement, and a marked increase of this dilatation when any obstruction of the respiratory passages is present. This last is characteristically shown in broncho-pneumonia. In all writings on lobar pneumonia one finds mentioned "an exaggerated movement of the *alae nasi*," and when this is described it is stated to be an inspiratory dilatation. It will be found that there is no such inspiratory dilatation of the nares in uncomplicated pneumonia, because there is no such obstruction of the air-passages as would produce it. Nevertheless, there is often in pneumonia an exaggerated movement of the *alae nasi*, but it consists of an expiratory dilatation. It occurs during or at the end of expiration, and it occurs only in association with the inverted type of expiration to which reference has been made. The method of its production is probably as follows: In the inverted type of breathing the expiration is forcible, and air is driven out with a certain explosive force, so that the air rushing through the nostrils distends the lax *alae nasi*.

3. In rare cases there may be observed irregular cerebral breathing. Some ten or twelve rapid respirations are taken, and then comes a complete pause or a temporary slowing, followed by rapid respirations again. This is sometimes described as "cyclic" breathing.

4. There is a complete absence of that distressed and anxious look on the patient's face which is usual in pulmonary affections associated with rapid breathing. The child may be sitting up in bed, breathing 90 to the minute, with a perfectly placid expression of face, and evidently not suffering from orthopnea; or the child may be lying down, looking as if suffering from a severe toxæmia, but not presenting that anxious and distressed look which is so marked a feature in pulmonary obstruction, as, for example, in broncho-pneumonia.

5. The signs of obstructive breathing are entirely absent. There is no over-action of the upper costal region, the sterno-mastoid and scalene muscles are not contracting actively, there is no supra-sternal or infra-costal retraction, and the chest is not kept fully or over expanded.

8. The change in the pulse-respiration ratio which is so characteristic of pneumonia in adult life is not so well marked in early childhood. The pulse-rate is usually high, running up to 120, 140, or 160 per minute, but without any symptoms of cardiac distress. Even in infancy one can determine that the respiratory rate has increased out of proportion to the number of cardiac pulsations. With a respiratory rate of 60 per minute one would expect under normal relations a pulse-rate of 240 per minute—a rate which is, of course, never attained. So that even in infants there is a certain retardation of the cardiac rate. In older children the characteristic pulse-respiration ratio of pneumonia in adult life may be exactly reproduced.

These different characteristics of the breathing—increased frequency, shallowness, absence of distress, inverted rhythm, and inverted action of the *abscus*—are so valuable in the diagnosis of pneumonia that one naturally goes a step farther and seeks for an explanation of them. The explanation of the respiratory changes usually given is that they are dependent on the pulmonary condition of consolidation and congestion. There are many difficulties in the way of accepting this view. The alterations in the breathing may be fully developed long before there are any recognizable pulmonary changes; they may persist unchanged while rapid advances in pulmonary consolidation are going on; and they may disappear more or less abruptly at the crisis, while the pulmonary condition is still unaltered. In pneumonia a small amount of pulmonary engorgement may be accompanied by great respiratory disturbance, while, on the other hand, a large area of consolidation may be accompanied by slight respiratory disturbance. The pain of pleurisy has been suggested by Eustace Smith and others as the cause of the "panting" or "grunting" breathing, with the inverted type of respiration, to which reference has been made. The child, it is said, holds its breath and lets go with a "pant" or a "grunt." This type of breathing, however, will be found to be characteristically marked when there is no pleurisy and no pain. If there is one thing one can tell decidedly about a child from the appearance, it is the presence or absence of pain, and in no condition is this more clearly shown than in pneumonia. It has also been suggested that the inverted rhythm is really an improvement on the normal type of breathing, in that it allows of the patient's retaining a larger quantity of fresh air in the lungs during the respiratory pause for the purpose of oxygenating the blood. The pause coming at the end of inspiration allows of a more prolonged and active interchange of gases. This explanation is at first sight rather plausible; but if it were correct, in all probability Nature would have made physiological breathing to be of this character. If anyone tries this for himself, it will be found that it is a most uncomfortable form of breathing to carry out voluntarily for any length of time, and yet in pneumonia it appears as an easy and natural action. As a useful working hypothesis, it may be suggested that the respiratory and cardiac phenomena of pneumonia are not primarily pulmonary or cardiac in origin, but are due to a disturbance of the respiratory and cardiac centres in the medulla. These centres are in close relation to each other, and also to the nuclei of the vagi nerves. Branches of the vagus pass from each lung to the medulla, and convey to the centre those impulses which regulate the respiratory movements in accordance with the requirements of the lungs. Other branches of the vagus pass from the medulla to the heart, and control the rapidity and strength of its movements. If we assume that there is some disturbance in these medullary centres, provoked either by the pneumonic focus acting through the vagus, or more

directly through the toxins circulating in the blood, we can easily find an explanation of the changes in the respiration and the heart's action which have been referred to. In the early stages the disturbance would appear to take the form of stimulation of the medullary centres, with resulting disordered action, while as the disease progresses the tendency is for a paresis or paralysis of the centres to develop.

Pain may be complained of over the site of the inflammation, and is due to accompanying pleurisy. It is by no means so frequently met with as in adult life, but when present it is of great diagnostic value in suggesting the nature of an otherwise obscure attack of fever. The presence of active pleurisy may be shown by the catchy breathing; the increase of pain on deep respiration, and by pleuritic friction. In many cases the pain is referred entirely to the abdomen, and all that can be learned from the young patient is that there is a "pain in the stomach." When localization is possible, the pain will be found to be in the abdominal wall on the same side as the lesion. Should this happen to be on the right side and in the region of the iliac fossa, a suspicion of appendicitis will arise. The combination of pyrexia, vomiting, and pain in the right iliac fossa, has sometimes led to this diagnosis being made and immediate operation carried out, and later it has been found that an attack of lobar pneumonia was the cause of all the symptoms. This mistake can easily be avoided if in the case of all attacks of acute abdominal pain in children a careful examination is made both of the abdomen and of the lungs. In lobar pneumonia one will find that, although pain and tenderness are present in the abdominal wall, there is not the marked abdominal rigidity and deep tenderness that accompany visceral disease, such as appendicitis. The pain is really referred from the costal nerves in the lower part of the pleural region to their peripheral endings in the abdominal wall, and is associated with a pneumonia of a lower lobe. Confirmation of this will be obtained on a careful examination of the lungs and of the nature of the breathing. In very rare cases one may find that the abdominal pain is due to an associated pneumococcal peritonitis, localized to the right iliac fossa or elsewhere. Here the diagnosis is by no means easy, and repeated examinations must be made. As a rule the referred abdominal pain in pneumonia subsides quickly when the patient is kept at rest. Another and very severe form of pain is that associated with diaphragmatic pleurisy accompanying pneumonia of a lower lobe, and is manifested by the extreme distress on even shallow breathing. Cough is not usually a troublesome symptom in uncomplicated lobar pneumonia. Many children pass through the whole illness, including complete resolution, with only a slight occasional cough. Resolution of the lung is usually carried out through the blood, and not by expectoration of the broken-down exudation. If catarrh of the larger bronchial tubes is present, as often happens, coughing is more frequent and troublesome. The characteristic sputum is not available for diagnostic purposes in the case of children, who rarely expectorate. Delirium and screaming may be present, and by some writers this symptom is regarded as being characteristic of an apical, as contrasted with a basal, pneumonia. It is difficult to obtain exact statistics, but as delirium may certainly occur in connection with a basal pneumonia, its value in determining the site of the exudation is not great. A well-marked *trake ciliolale* can often be elicited.

The temperature chart may be the typical one, the fever soon rising to 103° or 104° F., and oscillating but slightly along the line until the crisis occurs. On the other hand, a diurnal range of two or three degrees is not uncommon, and this is more frequently the case during the first two years of life than later. One must

also be prepared to meet with cases of pneumonia in which the temperature oscillates from day to day in a most uncharacteristic way, and makes the diagnosis uncertain, until there occurs a sudden cessation of all fever, usually between the fifth and seventh days of the attack. It is to be noted, however, that, as contrasted with other fevers of a hectic type, the temperature, even when irregular, does not during the course of the illness usually fall to normal or subnormal. A *pseudo-crisis* is a very common prelude to the real crisis in childhood, and is accompanied by a fall in the severity of the symptoms, which, however, return in full force with the subsequent rise in temperature. A crisis in two stages, as this may

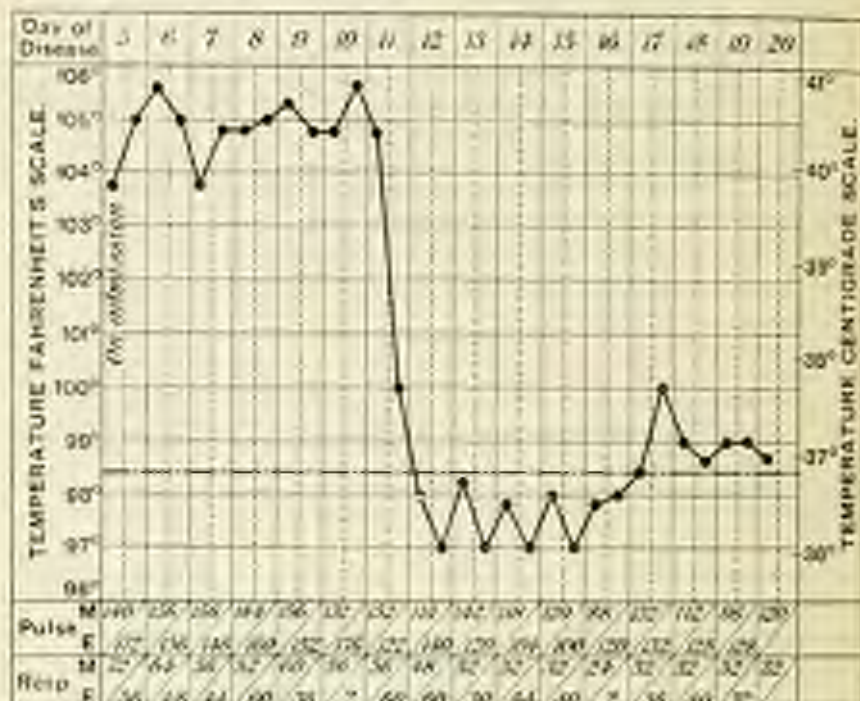


FIG. 11.—FEMALE AGED TWO YEARS: LOBAR PNEUMONIA OF RIGHT UPPER LOBE; RECOVERY.

Chart of a rather prolonged attack, with marked pyrexia, and subnormal temperature after the crisis.

be called, is probably better for the patient than a sudden drop of from seven to nine degrees in temperature, as one sometimes sees. The crisis occurs in the majority of cases between the fifth and the seventh days of the illness. It may occur as early as the second day or as late as the twenty-first. An abortive attack of pneumonia is one in which the crisis occurs on the second or third day, and this is quite a common condition in young children. The physical signs in the lung may be slight or absent, but all the other symptoms of the disease are present, and the early crisis is the only feature which marks this form from an ordinary attack of pneumonia. With a unilobar attack of pneumonia the crisis is rarely delayed beyond the tenth day, but may not occur until the fourteenth day. The prolongation of the illness

beyond the tenth day naturally increases the gravity of the patient's condition. Reginald Miller has classified the causes of protracted pyrexia as follows: (1) Protracted simple (non-spreading) pneumonia; (2) protracted spreading or recurrent pneumonia; (3) imperfect resolution; (4) bacterial complications other than pneumonia; and (5) toxic complications.

While the usual termination of the attack is marked by a crisis, cases occur in which it is by *lysis*. If the temperature is taken at frequent intervals during the critical period, it will be seen that the so-called "crisis" is a steady fall of tem-



FIG. 28.—FEMALE, AGED FIFTEEN MONTHS: LOBAR PNEUMONIA OF LEFT LOWER LOBE.

Daily chart showing normal course of disease, except for the markedly swinging temperature.

perature, lasting from twelve to twenty-four hours, and that the important feature is that this fall represents the end of the pyrexial stage. After the temperature has reached normal it may rise again, and this is usually due to the extension of the disease to another lobe, or to the development of some complication, such as empyema or crisis. In early life the crisis is not usually attended with the same tendency to collapse as in adult life, and if sweating or diarrhoea occur, it is not as a rule so severe. With the crisis the flow of urine becomes markedly increased, and the amount of chlorides passed in the urine, previously diminished, soon becomes excessive.

The presence of a marked leucocytosis may give valuable aid in the diagnosis in a doubtful case. The white cells in pneumonia are usually increased from 20,000 to 30,000, and may reach a much higher figure, but an absence of leucocytosis may be associated with either a very mild or a very severe case of pneumonia, so that the condition of the patient must be considered in settling this point. Herpes



FIG. 29.—MALE, AGED SIX YEARS: LOBAR PNEUMONIA OF LEFT LOWER LOBE; RECOVERY.

Chart showing that temperature, pulse, and respiration may run a corresponding course.

labialis is a valuable confirmatory sign in pneumonia, but is only exceptionally present. The herpetic eruption may be on the same or the opposite side to the lesion, it may be unilateral or bilateral, and may be so extensive as to affect the whole of one side of the face.

Diarrhoea is frequently present, as in all pyrexial affections in childhood, and is of no special importance when unaccompanied by other evidences of renal disease.

A condition of pneumococcal sepsitis will be referred to later. Percy Kidd has recorded the case of a boy, four and a half years old, who developed anasarca of the renal type without albuminuria nine days after the crisis, associated with erysipelas. This oedema passed off in a few days.

The physical signs in the lungs may be the same as in adult life, because the same three stages of exudation, consolidation, and resolution, occur in childhood. Certain differences are to be observed in childhood, partly owing to the clinical course of the disease, and partly owing to anatomical conditions. In many cases of pneumonia in infants and children the appearance of physical signs in the lungs is delayed until the fourth or fifth day, and in some they never appear at all. Without going the length of saying that pneumonia may run its course without any pulmonary exudation at all—for a local exudation in the lungs is probably the primary source of irritation in the vast majority of cases—it is certain that one meets with cases in which a brief impairment of resonance or a few crepitations are all the physical signs which are to be recognized in the lungs during the course of an atypical attack.

It is sometimes stated that the consolidation is so deeply seated that the physical signs are not recognizable at the surface, but this is much less likely to be the case in infants and children than in adults, owing to the small size of the chest. It is a common experience for one to examine a chest carefully without finding any physical signs of pneumonia, and on re-examination a few hours later to find extensive consolidation with all the usual signs. This sudden development of fibrinous exudation in the course of the disease is often very striking, and emphasizes the importance of frequent examination of the chest until the diagnosis is quite clear.

The first abnormal sign detected may be a *diminution* in the breath sounds over the affected area, as contrasted with the harsh puerile breathing over the opposite lung, and this may lead to an erroneous view that the area of exaggerated breath sounds is really the site of the pneumonia. Diminution of breath sounds is a much more important sign of disease in childhood than exaggeration. In some cases it may be necessary to adopt special measures in order to elicit the physical signs of pneumonia. If the child is breathing in a rapid and shallow manner, we may hear nothing but vesicular breathing without accompaniments; but if he be made to breathe deeply, as on coughing or crying, one can often hear a burst of crepitations or the high-pitched tubular breathing which clinches the diagnosis. Another method of securing the same result is to lay the child on one side of the chest and examine the lung which is uppermost. The air will be found to enter this lung more freely than when both lungs are acting equally. The child is then turned on the opposite side for the examination of the other lung. A change of position from lying down to sitting up, and vice versa, may so increase the depth of the breathing that latent signs are elicited. It is easy to tell an adult patient to breathe deeply, but with a young child both precept and example are frequently of no use. The conduction of signs from one side of the chest to the other is much more puzzling, and more common in childhood than in adult life. On auscultation of the chest one may hear the characteristic bronchial breathing and crepitations over both lungs, and this applies more especially to the back. It may be concluded that the patient is suffering from double pneumonia, while as a matter of fact only one lung is affected, and the sounds heard over the dull area are transmitted by the spine to the adjoining sound lung. More careful examination will show, not only that the signs are louder over one lung, but also that impairment of resonance

is confined to one lung. In the absence of dulness or impaired resonance one should not diagnose double pneumonia, however loud the transmitted sounds may be. In the examination of the chest in children the bases of the lungs are usually fully examined, but it is well also to remember the three apices—the apex of the upper lobe, the apex of the lower lobe, and the apex of the axilla—for in one or other of these places evidences of pulmonary consolidation are often found when they are absent elsewhere.

TYPES OF THE DISEASE.—As a rule one lobe of the lung is affected, and, taking the order of frequency, we find that the left lower lobe is most commonly affected, next the right upper, then the right lower, and lastly the left upper. At times one can distinguish a limitation of the disease to the middle lobe of the right lung, but as a rule it is involved with the right upper lobe. The disease is not always a



FIG. 50.—MALE, AGED EIGHT MONTHS; DOUBLE LOBAR PNEUMONIA, AFFECTING FIRST THE RIGHT LOWER AND THEN THE LEFT LOWER LOBE.

unilobar one, and a "creeping" or "wandering" type of pneumonia may occur. The initial attack may have run its course in the usual way, with a more or less complete crisis, but the symptoms of acute illness recur with high fever. If this is due to the creeping type of pneumonia, physical examination will show the signs of pneumonia in another lobe, either of the same or of the opposite lung. This process may be repeated until all the lobes of both lungs have been involved, and yet recovery may follow. In favourable cases rapid resolution takes place in the lobes which have been affected, so that their function is restored in time to replace the loss of function in others which have become consolidated. The result of this form of pneumonia is to prolong the disease, and the real crisis may be delayed for three weeks. It is sometimes said that this wandering or spreading form of pneumonia is characteristic of influenza, but it may occur quite independently

of any other signs of that affection. In some cases a double lobar pneumonia is present from the onset, one lobe of each lung being affected, and most commonly the two lower lobes. Such a condition is usually associated with a severe illness, both from the increased constitutional disturbance and the local interference with respiration and the cardio-pulmonary circulation.

While in most cases of pneumonia—even in those in which the objective pulmonary signs are quite latent for some days—it is possible for the unbiassed observer to diagnose with considerable accuracy the nature of the disease at an early stage, there occur others in which the poison seems to attack with virulence some other organ than the lung. The result of this is that we are apt to concentrate our attention on the organ chiefly affected as regards physical signs, and miss the real disease in the background. More especially is this the case in the cerebral type of pneumonia. Certain nervous phenomena are common in all cases of pneumonia in children. Amongst these are vomiting, headache, delirium, or convulsions, at the onset. In some cases these symptoms do not subside, but become more aggravated day by day. Hence the term "cerebral pneumonia" is in common use. The child may from the onset continue in a drowsy, lethargic state, crying out when disturbed, and wishing only to be left alone. One can often observe cases in which a drowsy languor or complete apathy persists all through an attack of pneumonia, and after the crisis the child seems to wake up as from a long dream, a wide-awake being again. Extreme tremulousness of the hands and of the tongue is often a marked feature. In other cases the nervous symptoms are of a more active type. There may be screaming and delirium at night. Headache may be severe, and convulsions, head retraction, opisthotonos, and rigidity of all the limbs, may be present. In short, the clinical picture of meningitis may be so exactly reproduced that one does not hesitate to make a diagnosis of that affection. As a matter of experience, we find that the above symptoms may be produced by (1) the poisoning of the cerebral tissues by the pneumococcus or its products; (2) an acute inflammation of the middle ear, frequently suppurative, and also pneumococcal in origin; or (3) actual pneumococcal meningitis. This last is extremely rare. As regards the first, the condition known as *menisgism*, if it is not recognized before the crisis, it will be apparent soon after, since the symptoms as a rule cease abruptly with the fall in temperature. The second condition—namely, inflammation of the middle ear—will be referred to later.

Another puzzling form of pneumonia is that in which the gastro-intestinal tract is the seat of great disturbance, probably from direct infection. Vomiting is present, the tongue rapidly becomes inflamed and furred, all appetite is lost, the abdomen becomes distended and hard, and sometimes tender, and rather severe and intractable diarrhoea sets in. When these signs are obstructive in a child manifestly ill, with continued fever, and when the signs of pulmonary disease are more or less latent, it is easy for the medical attendant to be led astray for a time, and a diagnosis of typhoid fever is sometimes made. The symptoms themselves do not call for any active treatment, save when tympanites is marked, and the pressure upwards of the diaphragm seriously impedes respiration in the later stages of the disease.

A further type of pneumonia in which the symptoms may at first puzzle one is that in which a condition of *sepsis* is present from the beginning of the illness, and the striking symptoms are hæmaturia, albuminuria, and dropsy. Advice may be sought because of hæmaturia or dropsy. An examination of the urine will

show the presence of albumin, blood, and possibly tube casts, while the pneumococci can often be found on staining a catheter specimen. If the signs of pneumonia are present and are recognized, the question will at once arise, Is this a case of pneumonia complicated with nephritis, or of nephritis complicated with pneumonia? If the signs of pneumonia are latent, one is easily led to make a diagnosis of nephritis only. Perhaps the chief diagnostic point in these cases is the elevated temperature, because a continued temperature of 103° or 104° F. is not common in acute nephritis. As a rule the course of the pneumonia is entirely unaffected by this complication, and with the crisis comes a rapid disappearance of the renal signs.

THE SECOND STAGE AND THE MODE OF DEATH IN PNEUMONIA.—As a rule the disease terminates favorably by crisis, but in certain cases there may be severe and alarming symptoms before the crisis, or the gradual increase of serious symptoms, with a fatal result. Whether in infancy, in childhood, or in adult life, the most common cause of a fatal termination in pneumonia is cardiac failure. Some have explained this on the ground that the heart is unable to carry on the circulation owing to the pulmonary obstruction. But the cutting off a part of the lung from the circulation, as happens in pneumonia, does not necessarily produce cardiac stress or cardiac failure, provided that, as usually happens, there is enough of sound pulmonary tissue left to keep up the oxygenation of the blood. At necropsy in fatal cases of pneumonia one will usually find quite enough of sound lung tissue for respiratory purposes, and will not find evidence of pulmonary obstruction sufficient to cause cardiac failure. The purely pulmonary theory as to the cause of a fatal issue in pneumonia, which assumes a mechanical failure on the part of the heart to carry on the pulmonary circulation, is not justified either by the clinical conditions or by the post-mortem findings.

A more reasonable theory is that which regards pneumonia as a profound toxæmia, producing disturbances through the system generally, and selecting more especially certain nervous centres. In these respects the disease may be compared with diphtheria, which is first of all a general toxæmia, and secondly shows a special affinity for certain nerve-centres. In pneumonia a patient may die from the profound toxæmia, being in a prostrate condition from the onset, and never showing any signs of rallying. This is not a common mode of death in childhood, although it may occur.

In the first stage of pneumonia the cardiac and respiratory centres in the medulla show signs of stimulation, and in the second stage they tend to become paretic, or even paralyzed. As regards the respiratory centre in the medulla, its action can be reinforced through the cortical centres of the brain, which bring into play all the accessory muscles of respiration. This second stage is marked clinically by an alteration in the breathing. The excessive frequency disappears, and as the breathing becomes slower the shallow abdominal breathing is replaced by deeper breathing in which the upper thoracic region takes the chief part. The placid expression of the patient's face changes to one of anxiety and distress, and all the muscles of respiration show laboured action, which did not formerly exist. These conditions may be due entirely to a failure of the medullary centre to carry on the work, or they may be aggravated by extensive consolidation of the lung or by the supervention of cardiac weakness. In most cases the respiratory powers will be sufficient to carry the patient safely through the crisis, in others cardiac failure supervenes before the respiratory powers are exhausted, and in some cases it may happen that death comes directly from respiratory failure.

As regards the effect on the heart of paresis or paralysis of the cardiac centre in the medulla, there is no auxiliary mechanism, central or peripheral, which can be called into play, as in the case of respiration. The inhibitory action of the vagus is diminished or cut off, and the second stage of pneumonia is marked clinically by an increased rate in the cardiac pulsations. If relief is not obtained, either through the natural termination of the disease or through artificial aids, the result will be that the heart runs on uncontrolled, and finally runs itself out. Hence we can understand why it is that a fatal issue is much more common in pneumonia from cardiac than from respiratory failure. Myocarditis and cardiac weakness from involvement of the heart muscle in the general toxæmia would appear to be much less common in childhood than in adult life.

In some cases only the first stage is present, the crisis supervening before the second is reached. The second stage begins as a rule about the fifth or sixth day of the disease, and may be termed the "pre-critical stage." The symptoms may be alarming, and the condition of the patient may cause anxiety for a day or two, but in the case of otherwise healthy subjects recovery as a rule takes place.

COMPLICATIONS.—Certain complications may arise during or after the acute stage of pneumonia. Some of these are due to an extension of the disease by direct continuity, and others are due to infection carried by the blood-stream. *Lobar pneumonia* is responsible for from 80 to 90 per cent. of all cases of empyema in childhood. This complication should always be suspected and looked for when the temperature continues irregular after the crisis. Many cases which appear to hang fire owing to delayed resolution of the lung are really dependent on the presence of pus in the pleura. The pus may be lying free in the pleural cavity, or shut off by adhesions, or between two lobes, or between the diaphragm and the lung. Very rarely there may be loculated collections of fluid, some purulent and some serous. Simple serous effusion to an extent recognizable by physical signs is not common in pneumonia, and the fluid is usually purulent from the outset. A definite diagnosis can only be made by the exploring needle, which one should not hesitate to use in the presence of suggestive local conditions. Even if there should be no fluid, but only consolidated lung, it is sometimes surprising to find that active resolution sets in, apparently through the stimulus of the needling process. In some cases of empyema the pus is so thick that the exploring needle may fail to detect its presence, and it may be necessary to open the pleura to confirm the diagnosis. The tendency to empyema seems to be most marked in infancy, and the majority of the cases occur during the first three years of life (see *Empyema*, p. 306). *Otitis media* is another complication common during or after the acute stage, and is often overlooked, owing to the absence of localizing symptoms. It arises from the passage of the pneumococcus from the naso-pharynx to the middle ear or through the blood-stream. Pain in the ear may be complained of, but is more frequently absent. On the other hand, hyperpyrexia, delirium at night, restlessness, and even the series of symptoms comprised in the term "meningism," may be directly due to acute inflammation of the middle ear. In the presence of these symptoms a careful examination of the ear should not be omitted. *Pericarditis* is a most serious complication, and one that is easily overlooked. Like other inflammations of serous membranes in pneumonia, it tends to be suppurative. Although it may be due to direct spread of infection, it is not necessarily associated with pneumonia of the left lung, as the blood-stream may convey the infection

from the right lung. The localizing symptoms are usually slight; pericardial friction is rarely heard; pain is not present; and, unless the area of cardiac dulness is being constantly watched, it is extremely likely that the pericarditis will be overlooked. It is often associated with empyema, but may occur separately. Pericarditis should be suspected when, after the crisis, the child seems to be much more ill than the physical signs would account for. Peritonitis is another complication, although a rare one. It may arise with the onset of pneumonia or develop at any period of the attack, and is almost invariably purulent in character. The onset is usually manifested by pain and the other signs of peritonitis. The whole peritoneum may be involved, or the disease may be localized for a time, but the abscess formed will tend to rupture into the general peritoneal cavity.

The systemic infection of pneumonia is also shown by the occasional occurrence of arthritis, meningitis, and subcutaneous abscesses. An arthritis may be manifested by inflammation and effusion in one or more joints. The effusion is usually purulent, but may be serous, and examination of the fluid will show pneumococci in abundance. The larger joints are chiefly affected—the hips, shoulders, knees, and ankles—and the distribution may be bilateral or unilateral. The chief incidence of this complication is in infants. True meningitis is a rare complication, and may be easily confused with the condition of meningism already referred to. If the clinical signs of meningitis, as opposed to meningism, are not sufficiently clear, lumbar puncture will probably aid the diagnosis. Josephine Hemmaway found in an examination of the cerebro-spinal fluid in twenty-two cases of pneumococcal meningitis, that the fluid was turbid, the cells were chiefly polymorphonuclear and the pneumococci were plentiful in the smears and grew readily in culture. In one case, however, which was proved on autopsy to be due to pneumococcal meningitis, three punctures showed normal fluid in the acute stage. Suppurative meningitis may also occur either as a complication of pneumonia or secondary to empyema. The signs of purulent meningitis are often very latent, such as a sudden attack of vomiting or the occurrence of a convulsion, and yet at the autopsy the whole surface of the brain under the arachnoid may be found covered with greenish-yellow pus. Subcutaneous abscesses may form during or after the acute stage of pneumonia, and may be single and large or numerous and small. When a large extent of the body is affected, the condition usually terminates fatally from toxæmia.

PATHOLOGY.—The pathology of lobar pneumonia is the same in childhood as in adult life. The pneumococcus is the cause of the disease, and its presence in childhood, as contrasted with adult life, seems to produce more bronchial catarrh, and a marked tendency to secondary infections in various serous and synovial membranes, with pus formation. A primary pneumococcal infection of the lung is followed by definite pathological changes and a well-defined clinical course. The mode of infection is probably by direct extension from the naso-pharynx or mouth to the lung, or possibly to the blood or lymph stream. It has been suggested that in pneumonia the infection may pass from the bowel through the thoracic duct and the right side of the heart to the pulmonary capillaries, a route which some hold is the normal one in the case of pulmonary tuberculosis. There is no direct evidence of any such mode of infection in pneumonia.

A pneumonia lobar in distribution may be caused by other organisms than the pneumococcus, but the course of the temperature and the disease generally will usually differ considerably. A boy of five years old was seen on the eighth day of

as illness, and found to have definite signs of a right basal pneumonia. The attack lasted for fifty-three days, the fever being markedly irregular and of the hectic type. The pneumonia was of the creeping type, spreading from the right lower lobe to the left lower lobe, thence to the right upper lobe, and finally to the right middle lobe. Streptococci were obtained from the lung by puncture on the thirteenth day of the illness, and from the blood on the forty-second day. Other atypical forms of pneumonia, although lobar in distribution, have been found to be associated with the influenza bacillus, the *Bacillus coli communis*, and the staphylococcus. The atypical nature of the attack and the prolonged course will usually suggest that some organism other than the pneumo-

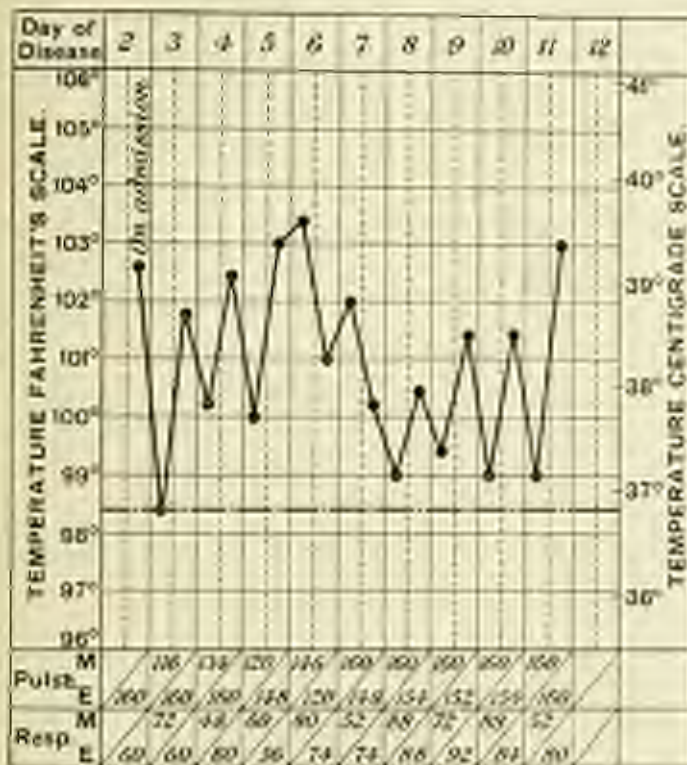


FIG. 22.—FEMALE, AGED ONE YEAR AND SEVEN MONTHS; LOBAR PNEUMONIA AFFECTING THE WHOLE OF THE RIGHT LUNG; GENERAL TOXEMIA; DEATH.

coccus is the cause, and the organism can often be determined by puncturing the solid part of lung and examining the blood obtained.

Pneumonia may be directly associated with a general infection of the blood and acute pneumococcal septicaemia, which may be extremely virulent, leading rapidly to a fatal issue. In such a case the lung may show signs of rapid breaking down, with abscess formation and even gangrene. On the other hand, in ordinary cases of lobar pneumonia the pneumococcus does not show any tendency to pus formation in the pulmonary tissue itself, while the secondary infections are chiefly purulent.

DIAGNOSIS.—The extent of the general disturbance in pneumonia, and the degree of pyrexia, together with the absence or latency of physical signs, has often led to a diagnosis of some general disease, like enteric fever, scarlet fever, or influenza. It is well to remember that the local signs may be absent for the first few days, and even up to the crisis, and that they may never be fully developed as regards a whole lobe; while repeated examination of the lungs should be carried out in doubtful cases, and early diagnosis may have to depend on the general symptoms. Of these the chief are the sudden onset in good health; the rapid rise in temperature, and high level maintained; the quickened, shallow, and often panting breathing; and the appearance of the child. In enteric fever the onset is more gradual, the temperature tends to rise gradually from day to day, and the altered respiration is not present. If the diagnosis is not clear at the end of a week, the application of Widal's test should settle it. Scarlet fever is characterized by a sudden onset with vomiting, but the condition of the throat and the rash, the lower temperature, and the absence of respiratory changes, should serve to distinguish it. Influenza, like pneumonia, has often a sudden onset, and may be accompanied by various



FIG. 37.—MALE, 4 AND THREE MONTHS: LOCAL PNEUMONIA AND PULMONARY ABSCCESS. DRAIN.

Daily chart showing the pneumonia pyrexia, the abscess pyrexia, and finally a normal temperature. Post mortem the whole of the right lung was in a condition of unresorbed pneumonia and mottled with small abscesses. The base of the left lung was similarly affected. Several pleuritic adhesions. Glands at the root of the lungs swollen, but not tuberculous.

indefinite symptoms, but the acute stage is short-lived, the pains and prostration are not found in pneumonia, and a few days' observation ought to settle any difficulty. An acute pyelitis or a systemic infection due to the *Bacillus coli* is often accompanied by high temperature, rigors, and rapid breathing, which may closely simulate the features of an attack of pneumonia. A bacteriological examination of the urine and the detection of the *B. coli* may be the only means of differentiation during the early stages.

Of local diseases other than pulmonary, the most likely to cause difficulty are acute tonsillitis and acute gastro-enteritis. Tonsillitis frequently manifests itself by a sudden rise of temperature to 104° or 105° F., the patient looks ill, but makes no complaint of pain in the throat or elsewhere. Consequently, if the throat is not examined in all such cases, an erroneous diagnosis of pneumonia may be made. Acute gastro-enteritis may usher in an attack of pneumonia, and the symptoms may for some time point so clearly to disturbance of the alimentary tract that the lungs are not examined. In pneumonia the course of the temperature is different, the breathing is probably characteristically altered, and a careful examination of the lungs will usually reveal some definite physical signs.

The chief diagnostic difficulty arises in distinguishing between lobar and broncho-pneumonia during the first three years of life. If it is held, as many hold, that lobar pneumonia is rare under two years of age, and very rare under one year, this difficulty will not cause much trouble. The physician will diagnose all his cases in infants as broncho-pneumonia, and his statistics will show a percentage of recoveries which must be extremely gratifying. On the other hand, if one does recognize and distinguish lobar pneumonia in infancy, the statistics of that disease will show a most satisfactory number of recoveries, while those of broncho-pneumonia will be most distressing as regards the mortality. Each of these diseases—lobar pneumonia and broncho-pneumonia—may run a perfectly characteristic course in infancy, so that no difficulty should arise in connection with the diagnosis. Other cases may be more mixed in their features, so that one has to consider the diagnosis more carefully, and this hesitation may even be prolonged until one has had an opportunity of studying the onset, the course of the disease, and the termination. Certain general distinguishing features may be summarized as follows:

Broncho-Pneumonia.

1. Common during first two years of life.
2. Subjects of weak constitution or debilitated by disease.
3. Frequently secondary to other acute illness.
4. Onset slow, with symptoms of bronchial catarrh.
5. Temperature rising progressively and showing marked irregularity.
6. Symptoms of pulmonary distress early.
7. Breathing rapid, deep, laboured, costo-abdominal.
8. Signs of bronchitis and patchy consolidation in both lungs.
9. Progressive signs of pulmonary embarrassment.
10. Cardiac trouble secondary to pulmonary lesions.
11. Duration of illness indefinite; relapses common.
12. Termination of fever by lysis.
13. Suppuration in other tissues rare.
14. Prognosis bad.
15. Usual termination—death.
16. Sequelæ: empyema uncommon; chronic fibrinous pneumonia usually due to this disease.

Lobar Pneumonia.

1. Less common.
2. Subjects usually healthy and well-nourished.
3. Usually primary disease.
4. Onset sudden without preceding bronchial catarrh.
5. Temperature rising suddenly and maintaining high level.
6. Symptoms of pulmonary distress late or absent.
7. Breathing rapid, shallow, not laboured, abdominal only.
8. Signs of consolidation in one lobe of one lung.
9. Progressive signs of disturbance of respiratory centre.
10. Cardiac trouble secondary to disturbance of cardiac centre.
11. Duration of illness, five to ten days; relapses uncommon.
12. Termination by crisis.
13. Suppuration in other tissues common.
14. Prognosis good.
15. Usual termination—complete recovery.
16. Sequelæ: empyema common; fibrinous pneumonia rare.

These distinctions apply generally, but they do not necessarily render the diagnosis easy in individual cases, and more especially in the early stages. It is well to bear in mind certain differences in the clinical course of the two affections. In broncho-pneumonia there is much coughing, rapid and laboured breathing, and usually an expression of marked respiratory distress. In lobar pneumonia, on the other hand, the cough trouble is trifling or entirely absent, the breathing is usually rapid, but is not laboured, and the expression of the patient's face shows no sign of respiratory distress. In broncho-pneumonia we find definite local (pulmonary) disease, with few signs of systemic disturbance; while in lobar pneumonia we find much systemic disturbance, with few local (pulmonary) signs to account for them. In both diseases cardiac weakness may supervene, but whereas in broncho-pneumonia the cardiac trouble is secondary to the disturbed circulation in the lungs, in lobar pneumonia it is secondary to toxic weakening of the medullary centre. A pneumonia which is primary and limited to the right upper lobe is a *croupous pneumonia*.

Still further difficulty arises in connection with that class of case which Samuel West has described as "primary broncho-pneumonia," but which resembles lobar pneumonia in all respects save that the primary affection is lobular in distribution. The following are the distinguishing features of this affection, according to West:

1. The child is often robust and healthy.
2. The fever commences abruptly, and is often ushered in by convulsions or nerve symptoms; it is of shorter duration, and terminates, as it begins, suddenly, often by well-marked crisis. The temperature is of a higher range and less remittent type.
3. There is no great tendency to relapse.
4. Recovery is rapid.
5. The mortality is small.
6. There is no antecedent bronchitis, and often no coincident bronchitis; and though occasionally in the later stages of the disease signs of bronchitis may present themselves, they are obviously secondary.
7. The general symptoms are often much more severe than the local ones, so that the pulmonary affection may be more or less masked.
8. The nervous symptoms may be marked, and the patients be unconscious or delirious, and it is in this group of cases that the difficulties of diagnosis from meningitis most frequently present themselves.

It will be admitted that the symptomatology of this affection is undoubtedly that of lobar pneumonia, while the signs in the lungs may be only those of a somewhat disseminated inflammation. The explanation of the pathology as given by West is probably correct—namely, that this affection is due to a pneumococcal invasion of the lung, differing only from lobar pneumonia in the distribution of the lesions. Should this type of disease be generally recognized and accepted as a clinical entity, the result would be that the present confusion in the minds of many as to the varieties of broncho-pneumonia would disappear. Considered clinically, broncho-pneumonia would be distinguished by the predominance of the local over the general symptoms of disease, while lobar pneumonia (including primary broncho-pneumonia) would be distinguished by the predominance of the general over the local. Considered bacteriologically, broncho-pneumonia will probably prove to be due to a streptococcal or staphylococcal infection, with some-

ness a pneumococcal superadded, while lobar pneumonia (including primary broncho-pneumonia) is due to the pneumococcus alone.

PROGNOSIS.—An attack of lobar pneumonia, limited to one lobe, and running its course without complications, usually ends in complete recovery. The illness may be a severe one, the prostration great, the temperature high, and yet after the crisis the child is quickly restored to health. On the other hand, until this successful result has been attained one can never tell what the course of the disease may be, as there are many complications which may alter entirely the prospect of recovery.

The age of the patient is important. Under two years of age the prognosis is not so good, largely owing to the fact that certain complications are specially common at this period. These include empyema, pericarditis, arthritis, and other suppurative affections. An infant already weakened by the primary illness is not well fitted to stand such complications, which usually require surgical treatment.

The important complications have already been discussed, and they are chiefly responsible for the fatal cases of lobar pneumonia. In other words, it is not the pulmonary infection, but the pneumococcal extension of the disease, which adds gravity to the prognosis. The extent of lung tissue involved is also of importance, and when several lobes are involved at the same time the general condition and the cardiac weakness may become serious. The duration of the attack has a direct bearing on the prognosis, and with a crisis delayed beyond the tenth day signs of increasing weakness will develop from the prolonged pyrexia and toxæmia. The crisis, however, may be delayed as long as the twenty-first day, and yet a complete recovery follow.

TREATMENT.—In an ordinary case, and in the majority of cases, if the patient is kept under favourable conditions as regards rest in bed, fresh air, warmth, and suitable diet, the disease will run its course to a favourable termination. No treatment of pneumonia has yet been devised which will shorten or influence the natural course of the disease, and many forms of treatment have fallen into well-merited discredit. Success with vaccine treatment may come in the future, but at present the course of an acute attack does not seem to have been much influenced by this method. Although we have no specific treatment, and the usual course of the disease is favourable, it is none the less necessary that every case should be carefully watched—first in order to relieve troublesome and weakening symptoms, and secondly to detect and treat complications which may arise.

The general treatment of an attack may be summarized as follows: The patient is kept in bed, clothed in a flannel nightdress, with or without some extra flannel or cotton-wool about the chest, according to the season of the year. The supply of bedclothes should be sufficient to preserve warmth, but not to be a burden or to cause sweating. Any chilling of the extremities is to be guarded against by the use of hot bottles. The bed should not be placed in a corner or close to the fire, or covered with a tent, but in an open part of the largest available room, where fresh air can play freely about it (without draught), so that the lungs may have a plentiful supply of cool, fresh air. A diet of milk and barley-water, with some nutrient broth or chicken soup, is to be ordered, and a febrifuge mixture containing citrate and acetate of potash is to be prescribed. In many cases the above, with

the help of good nursing and the treatment common to all acute specific fevers, will carry the patient safely through the illness. Certain symptoms may call for treatment.

1. *Pain about the Chest*.—If the pleuritic pain is manifestly causing the child suffering, a local application in the shape of a turpentine fomentation or lanced pessetice may be applied over the painful area for half an hour or until the skin is thoroughly reddened. This may be repeated some hours later if necessary. The practice of poulticing or fomenting the whole chest continuously throughout an attack of pneumonia, and irrespective of definite symptoms, has now fallen into disuse. It was based on a wrong pathology: it had no beneficial effect on the course of the disease, and it distinctly did harm by disturbing the patient, interfering with the freedom of the respiratory movements, and depressing the heart. On the other hand, the interrupted use of hot, moist applications for the relief of special symptoms is of great value. These local measures will usually serve to relieve the patient, except in that very painful variety associated with diaphragmatic pleurisy. In this condition the application of a few leeches (two or three) over the affected area, combined with strapping of the affected side, may secure relief and rest. But if they fail to do so, then opium or morphia must be given. Opium is distinctly contra-indicated in the later stages of pneumonia, because of its toxic effect on the respiratory centre; and as that centre is always affected to a greater or less extent in pneumonia, one should refrain from giving it for any symptoms save very severe pain. We have to consider whether the patient will not be more weakened by the pain and restlessness than by the opium, and decide accordingly.

2. *Coughing* may at times disturb the patient. The amount of coughing probably depends on the extent of lung involved. If there is a whole lobe involved, there will probably be a good deal of coughing from the irritation of the exudation. If only a small focus is involved, the irritation will be slight and the coughing trifling. It may be either of the short, hacking pneumonic type, or bronchial from the presence of catarrh. In both of these the interrupted use of hot local applications, as described above, will be beneficial. The position of the patient in bed must also be attended to, as frequently the coughing only occurs in certain positions, and is relieved by a change. The use of steam inhalations may also check an irritable condition of the bronchi. For the relief of persistent coughing, with or without much secretion, belladonna in doses of π v. x. of the tincture or $\text{R} \frac{1}{4}$ i. of the liquor atropinæ will be found advantageous. It is especially indicated in pneumonia because (1) of its checking secretion, (2) dulling the sensory branches of the vagus throughout the lungs, and (3) stimulating the respiratory centre. One can also safely and beneficially give 5 to 10 minims of paregoric occasionally for a hacking cough.

3. *Pyrexia*.—The ordinary temperature of pneumonia, averaging from 103° to 104.5° F., requires no treatment. In children the height of the temperature is not an indication of the gravity of the disease, and the rapid recovery after the crisis shows that the pyrexia is not *per se* a source of weakness or danger. Prolonged continuous pyrexia, say after the seventh day, is probably weakening, but should not be interfered with by drastic measures, as the pyrexia may directly bring the crisis nearer. The medical profession is quite unanimous in deprecating the use of antipyretic drugs in pneumonia. In some cases the temperature rises to 105° or 106° F. without the patient showing any signs of distress. Here, again, as

treatment is called for. But if great restlessness supervenes, with sleeplessness, symptoms which we trace to the presence of hyperpyrexia, then some interference is called for. In the case of infants, the employment of a hot bath, which is rapidly cooled to 85° F., will frequently be followed by a fall of temperature of from 1° to 2° F., and the restlessness will disappear. In older children sponging in bed with hot or tepid water is the corresponding treatment, and, if extreme nervous disturbance is present, the temporary application of cold-water cloths or Leiter's tubing to the head. It will often be found, also, that the application of a hot fomentation to the chest, combined with drachm doses of liquor ammoniac acetatis, has a distinctly antipyretic effect. An ice-bag to the chest has its advocates, but my own experience has led me to avoid extremely cold applications in childhood and infancy. When confronted with a rise of temperature amounting to hyperpyrexia, one must not assume at once that this is due to the pneumonia. Disturbances of an entirely different kind may be present in other parts of the system, and may be sufficient to raise the temperature by a few degrees. Such a disturbance will frequently be found in the gastro-intestinal tract, and consequently, in the absence of other definite causes, it is well to order a couple of grains of calomel or grey powder in case of hyperpyrexia with restlessness.

4. *Sleeplessness*.—Pursuing the plan of securing rest to the patient, we shall find it necessary to take action if sleeplessness occurs. Probably there is nothing more exhausting to the child's whole system than want of sleep. Delirious sleep is very common in pneumonia, and is probably not injurious. On the other hand, sleeplessness is often accompanied by active delirium, and marked prostration follows. We have already referred to the presence of middle-ear inflammation, and this condition should always be looked for, and, if necessary, treated when delirium and sleeplessness are present. Cold water to the head or hot applications to the chest may produce relief of these symptoms, but constant fussing about the patient with local application is apt to increase the condition. Probably the free use of bromide of ammonium in 10-grain doses, or chloral hydrate in 5-grain doses, is at once the most effective and the least harmful treatment. A dose of brandy, ʒi. to ʒss., in hot water, will often act like a charm.

5. *Symptoms preceding the Crisis*.—The symptoms of pneumonia are as a rule most pronounced and most threatening for the twenty-four or forty-eight hours preceding the crisis, and possibly for a short time after it. This is very strikingly shown in hospital practice, where so many pneumonia patients are admitted shortly before the crisis. The breathing and coughing may then have been observed to get worse, the restlessness greater, or the lethargy and prostration more pronounced. Sometimes an increase in the nervous symptoms may have excited alarm; sometimes blueness of the face or a tendency to faint may have been noticed. These or other signs have led the parents to seek medical advice for the first time. The same conditions are often present in cases which have been carefully treated from the first, and therefore it is important to be on the watch for the symptoms of the pre-critical or second stage of pneumonia. The longer the crisis is delayed, the more severe will the symptoms of the pre-critical stage become. This is the stage at which the active treatment of pneumonia should commence, as contrasted with our previous treatment for the relief of symptoms. Not that even at this stage do we actually treat the disease, but only certain results of it which are of vital importance.

Signs of cardiac weakness are to be carefully watched for, and we have to note

whether the failure is primarily in the left or in the right ventricle. There are many elaborate methods described for determining these points, but clinically the simplest method is the best. If dilatation of the left ventricle is taking place, as the result of cardiac failure, we shall find the apex-beat extending outwards to the left, the first sound at the apex becoming weaker, the pulse tension falling and the rate increasing, and a tendency to faintness and pallor appearing. At the onset of these signs the use of brandy, strychnine, and ether, is indicated in doses increasing in amount until we get a definite improvement. If the condition is more serious or is rapidly advancing, the hypodermic injection of strychnine is as serviceable in children as in adults. The injection causes little discomfort to the patient, and the only remark I have to make about the dose is that enough is not usually given. For an infant one year old in the pre-critical stage of pneumonia, liquor strychnine in 1-minim dose by the mouth, or $\frac{1}{2}$ -minim dose hypodermically, every four hours will produce good results. The reaction of the system to strychnine is much less easily produced in the profound toxæmia of pneumonia than under normal conditions. Consequently the drug should be pushed until we get the desired improvement, or until some symptoms of the physiological action, such as twitching, have been induced. If dilatation of the right ventricle is present with cyanosis, dyspnoea, and overaction of the right side of the heart, then some depletion of blood is called for. A very useful test as to the presence of this condition is the gradual enlargement of the liver, and clinically the increase in size of the liver will be found to be a very exact guide as to the degree of engorgement of the right side of the heart. This may be accompanied by signs of oedema in the affected lung or the sound lung. The application of three or four leeches over the hepatic region, followed by a hot fomentation to encourage further breathing, will usually be found to produce marked relief. This treatment is further aided by a dose of calomel (2 or 3 grains) followed by a saline, and by the use of alcohol and strychnine as cardiac tonics. Instead of the leeching, we may employ dry cupping over the bases of the lungs posteriorly, especially when pulmonary engorgement is present. It is also when cyanosis and right-sided enlargement of the heart occur that the use of oxygen inhalations, or, better, of a hyper-oxygenated atmosphere about the patient, may be of distinct service. In the treatment of pneumonia generally the advantage of oxygen inhalations is by no means clear, and the patient does not appear to be suffering from a defective supply of oxygen; but in cyanotic conditions oxygen certainly gives relief in some cases, and presumably benefits the patient. When the cardiac and respiratory centres in the medulla seem to be failing, we must use strychnine, brandy, and atropine freely.

As a rule, in lobar pneumonia there is no special call for the "open-air" or "haloey" treatment, and the nursing difficulties are increased. But in several cases, and where the nursing can be efficiently carried out, it will be found that open air is one of the best stimulants and tonics at the disposal of the physician, while at the same time it promotes sleep and appetite.

In an ordinary attack of pneumonia the crisis is not such a critical period as in the case of adults, and it is rare to see extreme prostration, sweating, or diarrhoea, at that period. None the less, even in straightforward cases one must be ready to stimulate the patient if necessary. After the crisis the breathing and cardiac rate soon become normal, and within twenty-four hours the danger of cardiac failure in the case of a child is usually over. Melville Dunlop has noted the occurrence of a slow pulse-rate, 40 to 60 per minute, as not uncommon during the

convalescent stage, and regards it as an indication of the profound toxic influence of the pneumococcus on the cardiac muscle.

Convalescence, in the absence of complications, is usually rapid and complete within a week. The physical signs of consolidation have usually passed off by that time, and even if they have not, there is no benefit gained by keeping the patient in bed. In the convalescent stage the deeper respirations induced by exercise and an open-air life will tend to promote resolution of the lung.

The special suppurative complications must be dealt with on surgical principles. After evacuation of the pus and drainage a troublesome sinus may persist, and here the use of an autogenous vaccine may prove beneficial. The organism found is usually the pneumococcus, but there may be a superadded streptococcal or staphylococcal infection, and the vaccine must be prepared accordingly.

CHRONIC INTERSTITIAL PNEUMONIA.

SYNONYMS.—*Corbosis* of the lung; *Chronic fibroid phthisis*; *Bronchiectasis*.

ETIOLOGY.—A condition of chronic inflammation of the lung or lungs in which the leading characteristic is an overgrowth of the interstitial tissue is by no means uncommon in childhood. This affection in its most typical and common form is a sequel to an attack of broncho-pneumonia in early life, more especially when associated with measles or whooping-cough. It is very often diagnosed as tuberculosis of the lungs, and, while the tubercle bacillus may in some cases be the chief irritant, it is only exceptionally so. The disease in its clinical course differs in all respects from the ordinary forms of pulmonary tuberculosis in childhood. Certain other causes may lead up to this condition, but they are all rare as compared with broncho-pneumonia. There may be after an attack of pleurisy a chronic inflammatory thickening of the pleura, with extension along the septa of the lungs in the neighbourhood. An empyema which has burst through the lung may set up a chronic fibroid inflammation. In some cases it may be that a chronic bronchitis will lead to a peribronchitis and excessive growth of connective tissue with fibrosis, and even to bronchiectasis. Still has suggested that some of the unilateral fibroid affections of the lung in later childhood may represent an advanced stage of the syphilitic pneumonia of infancy, which is itself a very rare disease. Auld has recorded a case of fibroid pneumonia in a child of eight years in which the initial lesion was apparently acute lobar pneumonia, the lung being previously healthy. The association of acute lobar pneumonia with fibroid pneumonia is admittedly very rare, and it seems probable that in such cases the acute pneumonia was due to some other organism than the pneumococcus.

As the progress of the disease is extremely slow, it is often difficult to say whether the affection is tuberculous or not in origin, but at any stage of the disease tubercle bacilli may attack the damaged lung. If there is a history of a previous attack of broncho-pneumonia, followed by a chronic cough and impaired health, the presumption is that the disease was not tuberculous in origin, as cases of tuberculous broncho-pneumonia are always fatal. The previous history may not reveal anything more than an attack of measles or whooping-cough; but if pulmonary troubles follow from such an illness, again the presumption is that the disease is not tuberculous. The differentiation clinically of the two types of disease may be rendered

very difficult or impossible. The clinical signs and the clinical course may be identical. Tubercle bacilli may not be obtained, either from an absence of sputum or from their non-detection in the sputum, and yet the disease may be primarily tuberculous. On the other hand, tubercle bacilli may be obtained, and yet be only the evidence of a superadded infection on a non-tuberculous pneumonia. Puncture of the lung for bacteriological purposes, which might help to clear up the diagnosis, is not advisable in this disease, for reasons to be stated later. Fortunately, the usual course of the disease and the treatment are the same whether the disease is primarily tuberculous or not, and even the prognosis does not appear to be much influenced by this factor.

SYMPTOMATOLOGY.—If the patient has been under observation during and after an attack of broncho-pneumonia, it will be found that the lungs had been slow in clearing up, and that some part or parts had never cleared entirely, while the patient's general health had never been completely restored. In many cases, however, the child comes under medical care for symptoms which were not regarded as dependent on any previous illness. The clinical history of the disease may be divided into two stages.

In the first stage, the history is one of cough, lassitude, loss of flesh, and increasing shortness of breath on exertion. This may have been present for some six or twelve months. The cough is usually hacking in character, often paroxysmal, and may be so severe as to induce vomiting. If there is any sputum it is probably mucopurulent, but not excessive. Intermittent pains in the chest may have been complained of, due to the pleurisy often associated with this disease. The temperature, apart from intercurrent complications, is not above normal, and may be sub-normal. Sweating is not usually at all excessive. A very careful examination of the lungs may be necessary in order to discover the signs of disease. While both lungs may be affected, the disease is much more frequently limited to one lung. While the upper lobe may be affected, the disease usually originates in the lower. A contracting fibroid apex of the lung is almost always, if not invariably, due to tuberculosis. As regards the two lower lobes, the left is much more frequently affected than the right, and the presence of the heart, acting mechanically by pressure, may have something to do with the frequency of interstitial pneumonia on that side. Inspection of the two sides of the chest may show deficient expansion on the side of the lesion. On percussion the note over the base of the affected lobe is dull, and the dullness extends a varying degree up the lobe, according to the extent of lung tissue involved. Sometimes the note is quite flat, and suggests the presence of fluid, but the absence of any displacement of the heart to the opposite side negates that. The flat note is due to the thickening which has taken place both in the pleura and the lung. The signs heard on auscultation vary from time to time in a most puzzling manner. Perhaps the most common condition is an absence or marked diminution of the breath sounds over the dull area, and a total absence of all accompaniments. At times the breathing may be bronchial in character, and this is usually most marked about the inferior angle of the scapula. Râles of a medium or coarse type come and go. The lung which has appeared to be silent one day may be the seat of clear bronchial or cavernous breathing on the next. The presence or absence of secretion in the bronchial tubes of the affected area will determine the nature of the accompaniments heard. Pleural friction of a coarse or medium type is often audible over the dull area. The physical signs are usually much more marked at the back and side of the chest than at the front.

In the second stage, which is reached after the disease has lasted for some time—a year or more—various secondary changes develop along with the progress of the disease in the lungs. The child is usually under-developed and of poor physique, there being apparently a real retardation of normal growth from this disease. The fingers and toes are often clubbed and the extremities dusky. In the early stages the bronchi have been softened and weakened by chronic bronchial catarrh. Violent fits of coughing tend to produce dilatation of these weakened bronchi. As the fibrosis of the lungs progresses, the bronchi are still further dilated by the contraction of the new fibrous tissue surrounding them, and a condition of bronchiectasis is established. Mucopurulent accumulates in these dilated tubes, and is coughed up with difficulty. An added bacterial infection may lead to the production of foul-smelling pus in the bronchiectatic cavities. A comparison of the two sides of the chest will often show a definite flattening, with diminished expansion on the side affected. The apex-beat and the heart are displaced outwards and upwards towards the axilla of the affected side. The area of lung tissue involved shows a slow but steady increase until probably the whole of one lobe is involved, and as the years pass the whole lung may come to be affected. There is an increase of the area of dullness and of the degree of dullness on percussion. The breath sound may be weak or absent in one part, and bronchial in another. The lung becomes more extensively attached to the chest wall by pleuritic adhesions, its power of expansion is much diminished, and it shrinks by the contraction of the new fibrous tissue formed. Although marked thickening of the pulmonary tissues may have occurred, no definite evidence of this is visible on examination by the X-rays, according to the writer's experience. The contraction of the fibrous tissue leads to the drawing over of the mediastinal contents, which are movable, and to the drawing in of the chest wall. The heart is still further displaced, and it may even be found that the sound lung is drawn across the mid-line and is resonant beyond the sternum on the affected side. The diaphragmatic surfaces of the pleura are usually adherent on the diseased side, and the diaphragm itself is drawn upwards, with elevation of the liver or the stomach according to the side affected. Muscular wasting from disuse or failure of development may be seen in the muscles of the thoracic wall on one side, and the spine is often curved with a lateral convexity towards the side of lesion.

While the course of the disease is essentially chronic, it is liable at all stages to acute developments due to the occurrence of acute bronchitis or acute congestion about the area of lung affected. If the patient has not been under observation previously, such attacks may prove very puzzling as to their nature and course. As regards the bronchitis, one will find an area of lung tissue showing signs which are not common in simple bronchitis. While the purely bronchial symptoms subside quickly under treatment, the dullness remaining suggests that probably some acute tuberculous process is present. As regards the attacks of acute congestion, there will be present a high temperature, the symptoms of pneumonia, and the evidences of consolidation in one lobe. Yet in the course of a few days the whole of the acute symptoms pass off. Unless the signs of chronic disease are recognized, one is apt to keep on treating the case as one of unresolved pneumonia, and to continue waiting for a complete resolution, which does not take place. The effect of each such attack of bronchitis or pneumonia is to lead to a further extension of the disease in the lung affected.

Chronic interstitial pneumonia may run a prolonged course for many years,

and may even persist until the patient is well advanced in adult life. It is rare for the lung to clear up entirely, or for a complete cessation of further inflammation and fibrosis to occur, if the signs have lasted over twelve months. The gradual extension of the disease may steadily weaken the patient, and death may be brought about by an attack of bronchitis or pneumonia. In tuberculous cases an acute tuberculosis may develop and prove fatal, while in cases of bronchiectatic cavities a cerebral abscess may carry off the patient. In the case of older children hæmoptysis to a considerable extent may occur.



FIG. 22.—PHOTOGRAPH OF A SECTION OF A LUNG OF A CHILD OF THREE AND A HALF YEARS, EXHIBITING THE CONDITION OF BRONCHITIS.

(From case described by H. M. Fischer, *Trans. Path. Soc. Lond.*, 1901, lii, 192.)

PATHOLOGY.—The pathology of the disease is the same as in adult life. There is some bacterial infection present, acting specially on the interstitial tissues of the lung and the pleura, and causing a cellular proliferation which is converted into fibrous tissue. While the tubercle bacillus is the active factor in some cases, the exact organism in other cases is not known, although it is probably the same as induced the preceding attack of bronchopneumonia. Probably there is a special tendency in the subjects of this affection to the formation of fibrous tissue, which is manifested on irritation, just as one sees an overgrowth of scar tissue in some cases of vaccination. In one way this overgrowth of fibrous tissue is beneficial, in that it shuts off the organism and produces a difficult barrier to its further extension. Hence the slow course of the disease and the rarity of general infection. In young and growing children the deformities resulting in the chest wall and the displacement of the thoracic viscera from the contracting fibrous tissue are much more marked than in adult life.

DIAGNOSIS.—The diagnosis rests on the chronicity and febrile nature of the disease, together with the more or less active signs in the lungs, the evidence of pleural thickening and pulmonary consolidation, and in the later stages the deformity of the chest wall and the displacement of intrathoracic organs. Clubbing of the fingers is a strongly confirmatory sign, and is usually present when the disease has lasted for some time.

The disease is often diagnosed as chronic pulmonary tuberculosis, but, as already stated, the whole course of the illness is quite different from that of the ordinary form of tuberculous disease of the lungs in children. Even the presence of tubercle bacilli may only be the evidence of a superadded infection which does not have much effect on the natural course of the disease or produce the ordinary changes which follow a tuberculous lesion in the lung (see *Pulmonary Tuberculosis*, p. 400).

The marked flatness of the percussion note and the absence of breath sounds may lead to a diagnosis of pleural effusion. This error may easily be avoided if one notes first of all the flattening of the chest wall, and next the displacement of

the heart and thoracic organs towards the side affected. In pleural effusion the heart is displaced away from the affected side. An error in diagnosis of this nature may prove serious if an attempt is made to settle the question by an exploratory puncture. However harmless the use of the exploring needle may be and is in cases of pleural effusion, or lobar pneumonia, or broncho-pneumonia, there seems to be a very definite and special risk in the case of chronic fibrinous pneumonia. During the past few years a number of fatal results from this procedure in cases of fibrinous pneumonia in childhood have been recorded. The explanation is not quite clear; for while in some extensive bleeding into the lung had followed, in others no such condition was found at the necropsy, and the cause of death seemed to be some form of reflex shock. These results have been so direct and striking that it would certainly seem to be advisable to refrain from puncturing a fibrinous lung, and it should be quite possible to distinguish the condition from pleural effusion without this test.

While the X-rays do not show much change in the lung affected until the pulmonary thickening is very advanced, they are of great value in showing the altered position of the adjoining contents of the thorax and the limited movement of the diaphragm on the side affected.

Prognosis.—The prognosis will depend on the progress of the disease, the surroundings of the patient, and the absence of complications. It is only in slight and early cases that a complete cure can be reasonably hoped for, while on the other hand there is as a rule no immediate danger to life. The disease will in most cases run a very chronic course, which is not incompatible with prolonged life, although the patient's general health and physique may never be up to the normal standard.

The surroundings as to climate, protection from exposure to cold or wet weather, and domestic life, will largely determine the prognosis, according as they are good or bad. The complications which may affect the prognosis unfavourably are severe and recurring attacks of bronchitis or pulmonary congestion, and the development of large pus-containing cavities.

Treatment.—The important part of the treatment is to place the patient in such surroundings as will tend to check the further progress of the disease, and if possible lead to the healing of the affected portion of lung. Residence in a warm, dry climate for a prolonged period is the best means of securing this result, and much benefit may be expected both as regards the direct improvement of the lung condition and the prevention of fresh bronchial and congestive attacks. Open-air treatment should be encouraged as far as possible. Nourishing foods and tonics of the nature of cod-liver-oil, iron, and strychnine, are beneficial. Complications, such as pleurisy, bronchitis, and pneumonia, call for the treatment suitable to these disorders. Gymnastic exercises directed to the full expansion of the chest should be carried out, but regulated so as not to overtax the patient's strength.

When a condition of bronchiectasis is present, with dilated and pus-containing tubes, the emptying of these may be the source of much difficulty and coughing. The emptying of the tubes may be aided by laying the patient across the bed, with the head and chest over the side, when the coughing will be much more effective. Inhalations of carbolic acid, creosote, or turpentine, help to check the secretion and disinfect the dilated tubes. Surgical treatment in the way of draining the bronchiectatic cavities is not advisable.

CONGESTION AND EDEMA OF THE LUNG.

Pulmonary congestion is a common condition in connection with all acute diseases of the lungs, such as bronchitis, broncho-pneumonia, and lobar pneumonia. The clinical signs by which it might be recognized in a separate form are concealed by those of the primary disease. The congestion is often of a fleeting character, and may be suggested by an access of dyspnea, a rise in temperature altering the normal course of the chart, whether pyrexial or otherwise, and some rales, diminished breath sounds, and impaired resonance over a part of the lung which appeared healthy. The disappearance of these symptoms and signs within a few hours suggests that they may have been due to an attack of congestion, but on the other hand they may have been due to a collapse of some portion of the lung. While one recognizes acute pulmonary congestion as a secondary result of acute disease of the lung, the condition cannot be separated clinically from the primary disease.

Sudden congestion of the lung or lungs may also occur as part of that mysterious disease known as "erythema multiforme exudativum," and cause great respiratory distress. It may also arise in connection with asthma. Passive congestion of the lungs is common in cases of cardiac disease and renal disease, but these present in childhood no differences from the same conditions in adult life.

The more special condition in early life to which attention may be directed is that of acute primary congestion of the lung. That sudden attacks of illness may arise in infancy or early childhood, with symptoms like those of lobar pneumonia, but which disappear suddenly, is familiar to all. Some energetic therapeutists have regarded such cases as examples of lobar pneumonia which have been rapidly cured by their therapeutic skill. Others, including Herzog, have regarded them as examples of abortive lobar pneumonia. Holt has pointed out that such attacks, evidently due to congestion of the lungs, may occur and recur in the course of malarial fever, and may be permanently cured by the administration of quinine. Cadet de Gassicourt has described the condition as the acute idiopathic pulmonary congestion of early life.

An attack begins with a sudden rise of temperature, the patient feels cold, looks ill, and possibly complains of pain in the side or abdomen. The breathing becomes rapid, possibly dyspnoic, and a cough with expectoration of mucus may develop. There may be headache, or delirium, or vomiting, or convulsions. On physical examination of the chest during the first twelve or twenty-four hours there are no definite signs. The resonance may appear to be slightly impaired in one part; there may be some loose rales, and possibly a certain blowing element about the breathing, all of which conditions may be present occasionally in the lung of a healthy child. The physical signs do not increase to any extent. The diagnosis cannot be made with any certainty, and is still in suspense, when, after one or two days of manifest malaise, the temperature suddenly falls, sweating or free diuresis may occur, and complete recovery rapidly follows.

Although this affection cannot be definitely pronounced to be lobar pneumonia, there is very strong support for the view that it is a form of pneumococcal infection, with congestion of the lung. The symptoms point to a general blood-infection, which is soon checked by the resisting powers of the body. The sudden onset and

the termination by crisis are like a pneumococcal invasion. The changes in the breathing without definite physical signs are comparable only to those found in connection with definite pneumonia. Herpes labialis is sometimes present, and the pneumococcus has been found in the expectoration. A marked leucocytosis has been found by some observers. At the same time, while in this country the pneumococcus is probably the commonest causal organism, it does not follow that it is the only one. Holt has found that the *Plasmodium malarie* may induce similar attacks. Possibly the *Bacillus coli* may do so also. The important point to remember is that such attacks may occur and recur in children during or apart from any other active disease, and may cause considerable difficulty in diagnosis. They usually last from one to three days, very rarely more than five, and do not call for any treatment other than rest in bed and a febrifuge mixture.

Acute Oedema of the lungs may occur in childhood, but is uncommon. The symptoms and etiological factors are the same as in adult life. Amongst infectious diseases, the presence of acute scarlatinal nephritis takes the first place. Amongst mechanical causes, the removal of pleuritic exudation too rapidly or in too great quantity may lead to acute oedema of the lung on the affected side, or of both lungs. While the condition is always a serious one, it is frequently recovered from. Treatment is to be carried out by means of poultices or fomentations to the chest, bleeding, and the use of vaso-dilator medicines, including brandy.

TUBERCULOUS BRONCHIAL GLANDS;

INTRODUCTION.—In considering the subject of pulmonary tuberculosis in childhood, one must include also tuberculosis of the lymphatic glands lying about the root of the lungs. These glands are situated in close relation to the trachea and œsophagus at the bifurcation of the trachea, and around the bronchi as far as their third or fourth division in the lungs. They form a correlated group, which may be called the "tracheo-broncho-pulmonary lymphatic system." In childhood they may become tuberculous, and from their enlargement cause definite symptoms by pressure on surrounding organs, or, as is more common, they may be the starting-point of a tuberculous infection of the lungs or of the system generally.

A primary infection of the bronchial glands by the tubercle bacilli is much more common than a primary tuberculosis of the lungs. The question has been much discussed as to how the infection reaches these glands. Some have held that there is an extension of tuberculous infection downwards from the glands of the neck to those of the thorax. In childhood the cervical glands are often tuberculous, but there is no evidence that this infection tends to spread directly downwards into the thorax, or that the subjects of tuberculous glands in the neck have any special proclivity to pulmonary tuberculosis. It was assumed for some time that the thoracic glands became infected through the intestine via the mesenteric glands. A difficulty arose in this connection when it was pointed out that the thoracic glands usually showed a much more advanced state of disease than the mesenteric glands, and that in many cases the latter were not affected at all. On the basis of some experimental work it was next suggested that tubercle bacilli passed

through the intestinal wall, then through the mesenteric glands, and eventually reached the thoracic glands, to develop actively there without leaving behind any trace of their somewhat circuitous route. This theory is difficult to disprove, but it is opposed to the ordinary course of tuberculous infection in other parts of the body. While pathologists are still divided in opinion as to the usual portal by which tubercle bacilli enter the system in early life, the view most commonly held at present by clinicians is that the bronchial glands are infected through the air entering the lungs. Tubercle bacilli present in this air are caught somewhere in the trachea, the bronchi, the bronchioles, or the air vesicles, and passed on by the lymph stream to the glands at the root of the lungs. In the glands the bacilli may be destroyed, or they may develop and cause marked glandular enlargement. This is a process comparable to what one sees in the case of the cervical glands, which become infected through the passage of tubercle bacilli from the mouth or nasopharynx. In both cases, also, it is to be noted that there is frequently no tuberculous lesion at the site of entrance of the bacilli into the system. In all probability the healthy lung of a healthy infant is by no means susceptible to tuberculous infection directly through the air stream, and can protect itself from the growth of tubercle bacilli in its tissues by passing them on to the glands. It is different, however, when the lung has been weakened and the soil prepared by an attack of bronchitis, broncho-pneumonia, or some specific fever. These are the two processes by which pulmonary tuberculosis is brought about: either directly through the settlement and growth of tubercle bacilli on some diseased part of the lower passages, or indirectly through the bronchial glands which have become tuberculous.

We shall return later to the direct infection of the lung, and in the meantime shall pursue the course of the tubercle bacilli which have entered the gland or glands. The gland, if it does not succeed in destroying the invaders, becomes enlarged and tuberculous. Other glands in the neighbourhood are similarly affected. In course of time the whole gland may become caseous and break down. These caseous glands are a very characteristic feature at autopsies on children, whether dying from tuberculosis or some other affection. On section, one finds the whole or a part of the gland occupied by a soft, cheesy-looking material. Around the glands there is inflammatory thickening, a peri-adenitis, which in time becomes tuberculous also, matting the glands together and invading the surrounding tissues. From this tuberculous stronghold the tubercle bacilli are enabled to attack the lung more successfully and in greater numbers than on their first passage through the lungs. The tuberculous growth may invade a small bloodvessel, and tubercle bacilli enter the blood-stream. This will bring about a general tuberculosis, with milary tubercles in the lungs. Again, a bronchus may be involved, its wall softened, and finally ulcerated through, with the result that the lobe or lobes supplied by the bronchus become tuberculous. This may result in a localized and chronic form of pulmonary tuberculosis, or it may lead to a disseminated and acute form. The glandular mass at the root of the lung may extend by a succession of tuberculous deposits radiating outwards and faroses through the pulmonary tissue. A considerable mass of tuberculous growth is then found near the root of the lung, which may itself soften and break down in part, and in the areas beyond this there may be scattered tuberculous deposits mixed up with healthy pulmonary tissue. If life is prolonged, the whole lung becomes involved. These are some of the routes by which the return journey, as it may be

called, of the tubercle bacillus to the lung is brought about, and by which tuberculosis of the lungs is established. From the glands the tuberculous lesion may also spread by direct extension to the pleura, the pericardium, or the mediastinal tissues. Local abscesses may follow the breaking down of the glands, which from their size are apt to cause pressure symptoms, and often rupture into the trachea or bronchus or pleura.

Direct infection of the lungs is not usually brought about when the pulmonary tissue is healthy. The type of tuberculosis common in adult life, in which the lesion is started a short distance below the extreme apex of the lung, is rare in early life. After the age of eight or ten years this lesion is more frequently met with. Still, it must be kept in mind that, although the occurrence is rare, the typical lesions of pulmonary tuberculosis in adults may be met with even as early as the first year of life, and they are apparently due to direct infection through the air-passages. Probably in such cases the soil has been prepared by the previous existence of tuberculosis in the parents, and the surroundings have been favourable to tuberculous infection. Direct infection may also arise in connection with any disease which lowers resistance in the pulmonary passages, such as bronchitis or broncho-pneumonia, especially when associated with a specific fever, such as measles or whooping-cough. Even in such cases, however, it may be that the infection comes from the tuberculous bronchial glands. Tubercle bacilli present in these glands may be stirred into activity by the constitutional disturbances, and may find an entrance into the damaged tissues of the lung. On the other hand, an attack of bronchitis or pneumonia causes an inflammatory swelling of the bronchial glands, which lowers their resistant powers, and may enable tubercle bacilli to settle and flourish in them.

SYMPTOMATOLOGY.—Were it possible to diagnose and to cure the condition of tuberculous bronchial glands, a great deal of pulmonary tuberculosis would be prevented. At present the diagnosis, especially in the early stages, is extremely difficult, and if treatment has to be deferred to the later stages it is seldom successful. The changes in the glands themselves have already been referred to. The symptoms and signs referable to their enlargement are general and local. The general symptoms are those common to all forms of tuberculosis, such as wasting, anaemia, irregular temperature, loss of appetite, and lassitude. The pyrexia as a rule is not great, save when suppuration has taken place in the glands. An irregular rise of from 99° to 100° F. at night is much more common. A marked condition of irritability is often a feature of this disease. One or all of these symptoms may be entirely absent in the early stages, and until the disease is sufficiently advanced to produce marked constitutional effects; so that, at best, from the general symptoms one can only suspect tuberculous disease of these glands, when a careful examination has failed to reveal any cause for the symptoms elsewhere.

The local signs are due to pressure on and irritation of the surrounding tissues—namely, nerves, bloodvessels, trachea, bronchi, and oesophagus. The signs are usually much more marked on one side of the chest than the other, and extension of the glandular enlargement takes place more frequently on the right than on the left side. Pressure on the nerves at the root of the lungs not infrequently leads to a harsh hacking cough, apt to occur in paroxysms, which may closely simulate whooping-cough. In young children without any sign of disease in the nasopharynx or lungs to account for it, this hacking paroxysmal cough is very sugges-

tive of enlarged bronchial glands. This form of cough is sometimes associated with attacks of dyspnoea and cyanosis, due to spasm of the trachea or bronchi and simulating closely an asthmatic seizure. Pressure on the bloodvessels is not usually so marked as to induce a persistent dilatation of the superficial veins, but a "Eustace Smith bruit" may be heard. This is supposed to be due to pressure on the large veins, and is heard on auscultation over the upper part of the sternum when the head is thrown well back. Considerable doubt has been cast on the value of this sign. Carey Coombs has found a murmur heard over the sterno-clavicular region to be common in association with anaemia and febrile states generally, and even in apparently healthy children. He regards the bruit as due to flattening of the veins against the transverse processes of the cervical spines. Fetterolf and Gittings suggest that the bruit depends on the persistence of the thymus, which can exert pressure on the vessels as they arise from the arch of the aorta. All that can be said is that the "Eustace Smith bruit" in a well-marked and persistent form is a rare sign, which may be associated with pressure from enlarged bronchial glands. Pressure on the trachea may cause a stridor, both inspiratory and expiratory, a clanging cough, and attacks of dyspnoea. To cause this the pressure must be considerable, and is perhaps most frequently present when the glands have broken down and formed an abscess. Pressure on the bronchi may cause more definite signs, because the pressure is usually on one side, and one lung only is affected. If the pressure is on the main bronchus, the breath sounds may be exaggerated or diminished, according as the degree of pressure is slight or great. The percussion note over the lung may be less resonant than on the other side. A more distinctive condition is that in which the pressure is on some branch of the main bronchial tree, so that the altered breath sounds are lobar in distribution. In young children the bronchus supplying the upper part of the right lung seems to be specially frequently affected. A condition of bronchial breathing above and below the right clavicle, with some impaired resonance, but without any accompaniments, is highly suggestive of bronchial pressure, provided that these signs are constantly and persistently present. In childhood quiet breathing often leads to a partial and temporary collapse of portions of the lung, which must not be mistaken for any pathological change. Pressure on the oesophagus may lead to difficulty in swallowing, but is very rarely found. Other aids in localizing a tuberculous lesion in the bronchial glands have been sought by means of percussion and the X-rays. Percussion over the upper part of the sternum and on each side of it may reveal an area of impaired resonance which has been held as indicating this condition, but it is a very late sign. There is a difficulty also in excluding the thymus as an equally likely cause. Posteriorly, percussion over the roots of the lungs may show a difference of resonance on the two sides, with bronchial breathing more extensively heard on the diseased side. This ought to be carefully examined for in all suspected cases. The X-rays do not appear to be of much value in moderate enlargement of the glands at the root of the lung, but when the glands lying in the lung are enlarged they may be visible on screening or photographing as irregularly disturbed patches. If the glands are calcareous, the X-ray picture will be much more definite.

The Prognosis in any given case in which the disease has advanced to such a stage that it can be recognized with accuracy must always be guarded, because of the tendency to a spread of the disease leading to primary or general tuberculosis. During the first five years of life tuberculous glands tend to soften,

caseate, break down, and disseminate their contents, and it is not until later childhood that fibrosis and calcification are developed as safeguards against general infection. The extension of the disease is an ever-present contingency which must render the prognosis grave in all cases. At the same time it is clear, from post-mortem evidence in children dying from other diseases, that tuberculous disease of these glands may last for a long time without any active development or extension, and that the glands may pass into a state of latent and inactive tuberculosis. During childhood there are many risks of the lighting up of this latent tuberculosis into activity, such as the specific fevers, attacks of acute pulmonary disease, etc., and very often this happens in cases in which the tuberculous disease had not even been suspected beforehand.

TREATMENT.—In cases suspected or diagnosed, the treatment common to all forms of tuberculosis should be carried out. Benefit may be expected in the early cases from the prolonged administration of the iodide of iron, iodide of potash, and arsenic.

PULMONARY TUBERCULOSIS.

INTRODUCTION.—The various types of pulmonary tuberculosis are apt to be so intermingled both clinically and pathologically that only broad distinctions can be given. The acute *miliary* lesions may be found in association with a chronic mass of caseous consolidation. Superadded infections by the pneumococcus or the streptococcus may complicate the clinical picture and produce additional lesions in the lungs. In many cases it is impossible to tell whether an acute bronchopneumonia is due to the tubercle bacillus or some other organism until the necropsy. Speaking generally, all acute tuberculous lesions of the lungs prove fatal. The chronic forms when slight in extent may be recovered from, as is shown by the healed lesions found post mortem in children dead of other diseases. The pulmonary disease may not prove fatal directly, but by the dissemination of the infection through the system causing general tuberculosis or tuberculous meningitis. The disease is very frequently distributed over both lungs. In other cases it begins in one lung and spreads to the other. Again, it may be limited to one lung only. The association with tuberculous disease of the bronchial glands usually leads to the involvement of one lung at first.

¶ The various forms of pulmonary tuberculosis in childhood tend to run a more acute and more rapid course than in adult life. This is especially the case during the first two years of life, less so up to the eighth year, after which the tendency to the more chronic forms predominates. The pulmonary lesions may either be the result of general tuberculosis or may be the starting-point of general tuberculosis. In childhood pulmonary tuberculosis, tuberculous pleurisy, and tuberculous peritonitis, are often associated.

¶ But little reliance as a rule is to be placed on the symptoms preceding the illness. Children are frequently brought for advice in whom pulmonary tuberculosis is suspected by an anxious parent, but in whom the symptoms are due to other causes. On the other hand, an attack of pulmonary tuberculosis usually develops when least expected, and this is explained by the fact that it is an acute extension of a previously latent glandular infection. Such things as chills, severe colds, an attack of measles or whooping-cough, etc., do not cause tuberculosis, although

they may prove the spark which determines the explosion of tuberculous activity. Experience has shown that with a tuberculous focus in some other part of the system, whether the glands, the joints, the peritoneum, or elsewhere, the possibility of pulmonary infection must always be kept in mind. The well-known tendency in adult life for chronic pulmonary pathosis to develop as a terminal factor in other wasting diseases, such as diabetes, is not common in early childhood, but may be met with typically after the age of eight or ten years.

1. Acute Miliary Tuberculosis.—This is a general infection in which the lungs may be involved. The tubercle bacilli are carried in the blood-stream to the lungs, where they form glistening grey granulations about the size of a large pin's head. The granulations may be dotted about at intervals, or may seem almost to occupy the whole of the lung tissue. The clinical picture of the disease varies greatly. There may be (1) no symptoms or signs of pulmonary involvement, or (2) symptoms but no signs of pulmonary involvement, or (3) symptoms and signs of pulmonary involvement.

(1) It is certainly very striking at a necropsy to find the lungs studded with grey granulations, which had caused no symptoms or signs of pulmonary disease during life. Outside the granulations the lung tissue is apparently healthy.

(2) There may be marked symptoms of pulmonary involvement in the shape of cough, dyspnoea, and increasing cyanosis, and yet on physical examination of the chest no alteration in the breath sounds, no accompaniments, and no signs of consolidation. In the case of a continued fever, suggestive of general tuberculosis, and without signs of other disease, this marked dyspnoea and cyanosis should suggest miliary tubercles in the lungs so extensive as to interfere with the aeration of the blood. There is probably in addition acute congestion of the pulmonary tissue surrounding the granulations.

(3) To the symptoms of cough, dyspnoea, and cyanosis, there may be added definite physical signs. The breath sounds may be very harsh and accompanied by numerous moist sounds, and there may be patchy consolidation with bronchial breathing. The clinical picture of acute broncho-pneumonia may be reproduced, and no doubt such is the disease with the tubercle bacillus as the active organism. At the necropsy the scattered granulations are the only features which distinguish the disease from ordinary broncho-pneumonia.

These various pulmonary conditions are only part of a generalized tuberculosis which is always fatal. The disease is met with frequently during the first two years of life. There is no curative treatment, and at most one can but relieve distressing symptoms with opiates.

2. Tuberculous Pneumonia.—This affection may occur either in an acute or in a chronic form.

The acute form resembles closely the ordinary broncho-pneumonia of infancy. Even after watching a case closely for some weeks it may be impossible to say whether the disease is tuberculous or not. The necropsy often shows that what was regarded as an ordinary attack of broncho-pneumonia was really tuberculous, and vice versa. The recovery of the patient may show that a case diagnosed as tuberculous broncho-pneumonia was due to some other infection.

The distribution of the lesions may be lobar or lobular. They are often started by a direct extension of tuberculous disease from the glands at the root of the lungs. One lung only may be affected at first, but if the patient lives long enough

the other lung will almost certainly be involved. Not infrequently the disease begins as an acute pneumonia of the pneumococcal type. The physical signs and symptoms are those of lobar pneumonia, and blood drawn from the affected area may give a pure culture of the pneumococcus. The temperature may for a time be that of lobar pneumonia, but the crisis does not occur at the usual time. Instead of a crisis we find the temperature continuing high, probably with marked oscillation. The consolidation spreads, and, after involving the whole of one lung, may extend to the other. Partial resolution of the solid areas may occur, but as a rule there is never the complete clearing up that one meets with in ordinary lobar pneumonia. The distinction between this condition and a "creeping" pneumonia may be extremely difficult or impossible until the parts are examined at the

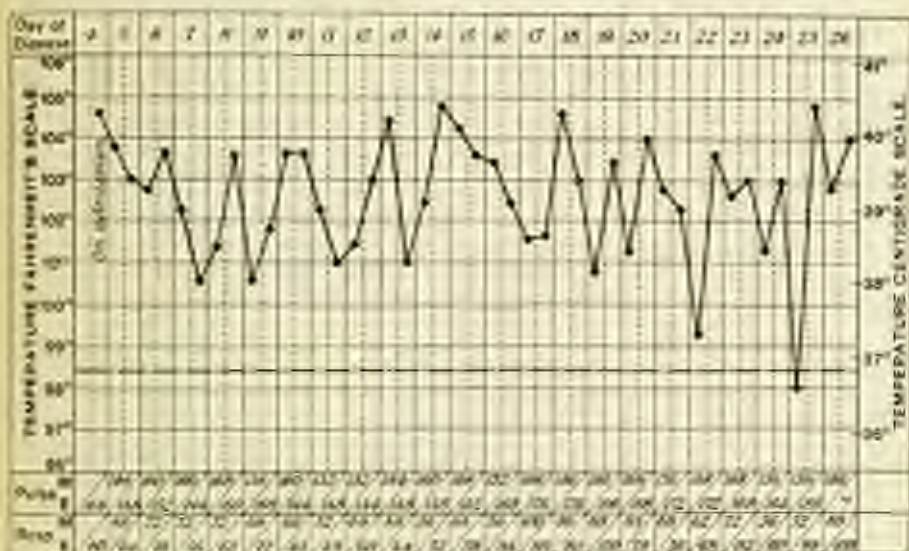


FIG. 31.—MALE, AGED TWO YEARS AND EIGHT MONTHS: TUBERCULOUS PNEUMONIA; DYSENTERY.

Disease started acutely as pneumonia of right lower lobe, and pneumococci were found on exploratory puncture. The whole of the right lung became involved, and later the most of the left. Post mortem, generalized pneumonia, many abscess cavities, pneumothorax, and empyema of right side. Glands at the root of the lungs were caseous.

autopsy. If recovery follows, one assumes that the disease was not tuberculous. A streptococcal pneumonia, with its usual lobular distribution may after the early stages become complicated by tuberculous infection. The pleura tends to be extensively involved in tuberculous pneumonia, and pleuritic adhesions usually form over wide areas and tend to become very dense. Occasionally pneumothorax occurs, and may be recognized clinically. It is due to the bursting into the pleura of a tuberculous abscess in the lung, and an empyema follows. Both pneumothorax and empyema are usually limited in extent from surrounding pleural adhesions.

PATHOLOGY.—The pathological lesions vary greatly in different cases. The glands at the root of the lungs are commonly found in a state of advanced caseation, and may have formed a tuberculous mass extending into the lung. The lungs contain scattered areas of tuberculous deposit which tend to caseate, and may

form multiple abscess cavities. The areas of tuberculous infiltration may have a lobar or lobular distribution. This may depend on the presence of a pneumonia of a lobar or of a lobular type, or it may depend on the manner in which the disease has spread from the bronchial glands. Secondary infection may be responsible for localized patches of inflammation which are not evidently tuberculous. Scattered miliary tubercles may be seen in parts of the lung which seem otherwise healthy. The bronchi and bronchioles may be acutely inflamed and tuberculous, while their contents may be mucus-pus or pus. As in all acute pulmonary affections in childhood, areas of collapse and of emphysema are common.

SYMPTOMATOLOGY.—The general and local signs of this disease are the same as in other forms of broncho-pneumonia. Irregular fever, wasting, cough, and vomiting, are usually present, but have nothing characteristic about them. Some cases may show only signs of diffused bronchial catarrh and the so-called "capillary bronchitis"; others have in addition signs of patchy consolidation, and others signs of extensive consolidation. The signs of cavitation may or may not be present, according to the size and the distance of the cavities from the surface. It must be remembered that in children all the signs which are held to be characteristic of a cavity may be present in simple consolidation of the lung. Cardiac weakness and dilatation may develop as a result either of the obstruction to the pulmonary circulation or of the pyrexia and toxæmia present. The liver and spleen are often enlarged. Speaking generally, one may say that the spleen is more commonly enlarged in the tuberculous than in the other forms of pneumonia. The course of the disease is always towards a fatal termination, due to the pulmonary affection, tuberculous meningitis, general tuberculous, or exhaustion.

The chronic form is distinguished only by the more chronic course of the attack. To a certain extent this may be due to the absence of secondary infections of a virulent type, such as the pneumococcus, streptococcus, etc. The temperature chart shows a much lower range of fever than in the acute form. The extension of the disease from the root of the lungs takes place more slowly and in a more limited manner. Some scattered tubercles develop slowly in the lungs. The symptoms may be coughing and dyspnoea on exertion, and on examination one may find signs of bronchial catarrh, with copious sputa. The patient looks ill, wastes, and becomes listless and inactive. Sooner or later the child sinks, death taking place from exhaustion, or the acute form of tuberculous pneumonia, or general tuberculosis.

3. Massive Pulmonary Tuberculosis.—In some cases one may find the tuberculous infection spreading out from the root of the lung, and involving the pulmonary tissue like a solid *onc* growth. A whole lobe or more may come to be involved, all the pulmonary tissues in this area being converted into a solid yellow mass of caseous matter. On physical examination, the note may be absolutely dull over this area, while the breath sounds are entirely absent or possibly bronchial in character. The presence of fluid in the pleural cavity is suggested by the flatness of the note, but exploratory puncture gives a negative result. The constitutional disturbances may be slight. A similar condition of massive involvement may be caused, as Still has pointed out, by the bursting of a caseous gland into a bronchus, so that the whole area supplied by that bronchus becomes a solid mass of caseous matter. The illness usually terminates by the extension of the disease in an acute form either through the lungs or through the system generally.

These three forms—acute miliary tuberculosis, tuberculous broncho-pneumonia,

and remote pulmonary tuberculosis—have a dread significance from the fact that they are all secondary to tuberculous elsewhere. Given a small tuberculous focus in the lungs of a primary character, the resistant powers of the organism would probably combat the invaders successfully, and the disease might run its course unrecognized from the slightness of the symptoms. But we have in these three types the results of a successful attack on the system at some previous time. The tubercle bacilli have entrenched themselves in the bronchial glands or elsewhere. From their stronghold they are able to invade the pulmonary tissues, either by a general and rapid advance through the blood-stream or a bronchus, or more slowly by a gradual extension of the tuberculous process from the root of the lungs. Treatment directed to the lungs has no effect on the immense reserve forces in the glands. One feels that, unless the stronghold in the glands can be rooted out, victory is bound to be in the end with the tubercle bacillus.

4. Chronic Phthisis Pulmonum.—This, the common form in adult life, is rare in infancy and early childhood. Its incidence begins to be more common after the age of ten years. None the less it is sometimes met with even in the first year of life, with the typical localized tuberculous deposit, caseation, and cavitation. It is in the early years of life that the diagnosis is likely to cause most difficulty, because of the rarity of the condition. The primary focus of disease is frequently in the right upper lobe, but the lower lobes are much oftener the starting-point of the disease than in adult life. The clinical course, the symptoms, and the signs, are the same as in adults, with the difference that the progress of the disease tends to be more rapid, and the occurrence of a general tuberculosis is more common. Although complete recovery may follow, as post-mortem evidence shows, such an event is rare, save when the lesion is small and so slight as to have given rise to few, if any, definite symptoms. When the signs of chronic phthisis are well marked in childhood, the disease usually progresses to a fatal termination. Laryngeal tuberculosis may occur in this form, and in a manner similar to that of adult life. Hemoptysis is rare, and is more frequently associated with a cavity than with an early stage of lung disease. Ulceration of the bowel is common, and is probably due to the swallowing of the sputum. Even in older children expectoration is never so thoroughly carried out as in adults.

The onset of this disease—"consumption," as it is commonly termed—is much dreaded and frequently diagnosed by the lay public. Every form of chronic cough is regarded as of pulmonary origin, and advice is sought in order to have the diagnosis confirmed or refuted. Wasting, lassitude, loss of appetite, and other symptoms, are regarded as pointing strongly to consumption. It is only by means of a careful examination, not only of the lungs, but of the whole respiratory passages, that one can form a definite opinion as to the presence or absence of chronic phthisis. It is well to bear in mind at the outset that this form of tuberculosis is not common in childhood. The exciting cause of the cough is much more frequently found in some non-tuberculous lesion of the nose, naso-pharynx, trachea, or bronchi. It is impossible, in the absence of definite physical signs in the lungs, to make a diagnosis of chronic phthisis with any certainty. In the absence of any indications of disease in the respiratory passages and lungs, one is often justified in suspecting latent tuberculosis of the bronchial glands, and these should be examined so far as clinical methods will allow (p. 293). When the symptoms are entirely general, such as wasting and slight pyrexia, the other systems must be examined, as the disease may be situated in some other part of the body.

5. Chronic Tuberculous Pulmonary Fibrosis.—This condition was referred to in connection with chronic interstitial pneumonia. There is no doubt that primary tuberculosis is often diagnosed in cases of chronic pneumonia in which the tubercle bacillus plays no part. The leading features of pulmonary tuberculosis in childhood are the acuteness of the symptoms, the absence of fibrosis, the tendency to cavitation, and the rapid course to a fatal termination. Chronic pulmonary fibrosis, on the other hand, is characterized by the chronicity of the disease and the absence of acute symptoms, apart from complications, while there is no tendency to cavitation, and there is always a marked degree of fibrosis present. We have therefore to assume that in cases of chronic tuberculous fibrosis there is either some peculiar action of the tubercle bacillus, or some special predisposition on the part of the patient to the formation of fibrous tissue in the lungs. Neither of these assumptions is supported by the clinical or post-mortem evidence in connection with other forms of pulmonary tuberculosis in childhood. Chronic fibroid pneumonia is not infrequently met with during the ages of two to six years (when acute tuberculous disease of the lungs also prevails), and can usually be definitely traced to an infection other than that of the tubercle bacillus. Definite tuberculous pulmonary fibrosis is rare at this period of life, as judged both by clinical and post-mortem evidence. Nor can it be admitted that the finding of tubercle bacilli in the throat or sputum is positive evidence that the lung affection is tuberculous. In a case of chronic fibroid pneumonia there may be a secondary or superadded infection by the tubercle bacillus, just as in the case of tuberculous pneumonia there may be a secondary infection by the streptococcus or the pneumococcus. The presence of tubercle bacilli does not seem to affect to any marked extent the course or symptoms of the pulmonary fibrosis, probably because the fibrosis renders the soil extremely unsuitable for its development. In early childhood, therefore, the course of a fibroid pneumonia will be found to be much the same whether the tubercle bacillus has or has not been found to be present. The diagnosis, prognosis, and treatment, are the same in all cases, and has been already considered in connection with chronic fibroid pneumonia (p. 389).

In later childhood, after the age of eight years, the tendency to chronic form of pulmonary tuberculosis begins to manifest itself, and in these a leading feature is the formation of fibrous tissue. This is Nature's method of shutting off a localized tuberculous process. At this period of life we may meet with a tuberculous lesion near the apex or in one of the lower lobes which becomes encircled by fibrous tissue, and as the patient and the lung grow the fibroid area will produce those local changes described as occurring in chronic fibroid pneumonia. The active disease may have ceased entirely, and the symptoms will depend on the extent to which the pulmonary tissue has been destroyed. In a tuberculous subject the future prospects are clouded by the risk of a lighting up of the old tuberculous focus, which may have a cavating centre, and of a fresh infection of the lungs by the tubercle bacillus.

THE DIAGNOSIS OF PULMONARY TUBERCULOSIS.—In cases of pulmonary tuberculosis a certain amount of weight is to be attached to the family history and previous health. If there is a parental history of tuberculous disease, we may consider that the patient has a natural predisposition to the disease. If other children in the family have died of the disease or suffered from any form of tuberculosis, it is probable that the home surroundings are favourable to the spread of this infection. In considering the previous health of the patient, a history of

wasting, cough, and loss of appetite for some months before the appearance of active symptoms, is suggestive of some latent tuberculous lesion. Inquiry should also be made as to whether the patient had suffered previously from any form of tuberculous disease, be from any acute pulmonary disease associated with menses or hooping-cough. Certain acute diseases of the lungs, and especially those in connection with the specific fevers, cause inflammatory changes in the glands at the root of the lungs, which frequently pave the way to tuberculous infection. A history of tuberculous peritonitis, hip-joint disease, glandular abscess, etc., should always render one suspicious of tubercle if active pulmonary signs appear. Too much stress must not be laid on the appearance of the patient when he is of the so-called "tuberculous" type. Such children are quite as liable as others to acute pulmonary affections of a non-tuberculous character.

The course of the disease may give some aid in the diagnosis. In subjects under the age of two years the disease runs a rapid course after the appearance of active signs; consequently, at this age an acute pulmonary affection lasting for more than a month is probably non-tuberculous. Definite periods of cessation of acute symptoms followed by exacerbations are less characteristic of tuberculous pneumonia than of ordinary broncho-pneumonia. Recurrent attacks of bronchitis or pneumonia are probably non-tuberculous, although they may tend in time to the development of glandular tuberculosis. As the child grows, the tendency to the more chronic forms of pulmonary tuberculosis increases, but up to the age of ten years they are exceptional in a well-marked form. Where widespread pulmonary signs have existed for a year without causing death, tuberculosis can almost certainly be excluded as the cause of the disease.

It is but rarely that the symptoms and physical signs give unequivocal evidence of tuberculous infection. The temperature may be as high and as irregular in one form of pneumonia as in the other. Wasting, anæmia, and sweating, are common to all acute pulmonary infections. The signs of bronchial catarrh and of patchy or extensive consolidation are the same, whatever the specific cause may be. In tuberculous pneumonia, pleurisy leading to firm pleural adhesions is more frequent than in ordinary broncho-pneumonia, and if definite signs of pleurisy can be made out the disease is probably tuberculous. Unfortunately, the signs of pleurisy are often absent. If there is definite evidence of cavity formation, the disease is almost certainly tuberculous. When the pulmonary signs are limited to one lung or to one part of a lung, tuberculosis should be suspected. Still says that the limitation of active tuberculosis to one lobe when there are signs of enlarged mediastinal glands is highly suggestive of ulceration of a caseous gland into a bronchus, especially so on the right side.

That valuable sign in adults of early tuberculous infection of the lungs—namely, hæmoptysis—is rarely met with in children at any stage of the disease. Confirmatory evidence may be obtained by the presence of definite tuberculosis elsewhere. Tubercle of the choroid is quite characteristic, and is found most frequently in connection with acute miliary tuberculosis, less commonly in connection with the chronic types. Examination of the blood shows as a rule the signs of secondary anæmia, with a normal or diminished number of leucocytes. In the acute forms of pulmonary tuberculosis this may be altered by the presence of secondary infections.

The most definite evidence of pulmonary tuberculosis is the presence of tubercle bacilli in the sputum. In the case of children one is met with the difficulty of

obtaining any sputum. This difficulty may be overcome when the child is over five years of age by a little careful training in the method of expectoration. In the case of younger children the plan introduced by Holt may be employed. Coughing is excited by irritating the pharynx, and the sputum thus brought into view is caught on a bit of muslin or gauze. The cough may be started by rubbing the pharynx with the end of a pair of forceps wrapped in muslin. The mucus and mucus-pus thus obtained may be stained for tubercle bacilli. Another method, according to Holt, which is suitable for infants and young children, is to pass a stomach-tube well into the oesophagus and strain the mucus which adheres to it when withdrawn. By inverting a child during a paroxysm of coughing, a considerable mass of mucus may be coughed into a receiver. In testing these methods in seventy-six cases of pulmonary tuberculosis, Holt found tubercle bacilli in over 80 per cent., although in over half of them the disease was not advanced, as judged by the symptoms and physical signs. In most of the cases repeated examinations were necessary. It need hardly be added that the absence of tubercle bacilli from the sputum is not to be regarded as proof that the disease is not tuberculous.

The von Pirquet and the Calmette tests have recently come into common use. They are both of limited value, in that they indicate the existence of a tuberculous lesion, past or present, but give no information as to its locality. They are therefore of no positive value as regards a doubtful case of pulmonary tuberculosis. On the other hand, if the reaction is negative, it is probable that the disease is not tuberculous. Both of these reactions seem to be unreliable in cases of acute *milary* tuberculosis, which so frequently develops in connection with pulmonary tuberculosis. The von Pirquet test is much to be preferred to Calmette's in the case of children, as severe conjunctivitis may follow from the use of the latter.

The diagnostic method of using tuberculin by means of injection is not often carried out in early life. It is suitable only for cases with an afebrile temperature, as the characteristic reaction is a febrile one. It merely indicates the presence of a tuberculous focus. For its use Koch's original tuberculin in doses of $\frac{1}{10}$ to $\frac{1}{5}$ milligrammes may be employed either subcutaneously or by the mouth. Holt tested 102 infants with tuberculin, and got a positive reaction in 34. The dose, given subcutaneously, varied from $\frac{1}{10}$ milligramme to 6 milligrammes. Out of the 34 giving a positive reaction, 30 were shown positively to be the subjects of tuberculosis. The average temperature reaction in 15 cases was 103.3° F., reached in thirteen hours. He has not seen any bad effects from these injections.

While the diagnosis of pulmonary tuberculosis may present great difficulty in its acute forms, in but too many cases the development of tuberculous meningitis or general tuberculosis will settle the problem. From the practical standpoint, it is more important that chronic affections characterized by cough or some bronchial catarrh should not be diagnosed and treated as tuberculous disease of the lungs on insufficient evidence. After an acute affection like influenza, bronchitis, bronchopneumonia, etc., there may persist for some time cough, an irregular temperature, and some physical signs in the lungs, which are not necessarily tuberculous, and which will eventually clear up under suitable treatment. The systematic examination of school-children has led to the discovery of many cases in which pulmonary tuberculosis has been suspected owing to the presence of indefinite signs and symptoms; and while such children may require care, and will be much improved by hygienic surroundings and good food, it is not justifiable to label them as *infected* from pulmonary tuberculosis, with the accompanying isolation and ostracism

which follows such a diagnosis. Remembering the many other causes of such symptoms in childhood, we will do well to make as careful an examination as possible to determine the existence of definite signs of tuberculosis, and to withhold such a diagnosis in the absence of unequivocal signs. More especially are such diseases as chronic fibroid pneumonia, bronchiectasis, chronic pleurisy, and neglected or undiagnosed empyema, likely to be mistaken for chronic pulmonary tuberculosis, because of the general appearance of the patient and the physical signs present.

PROGNOSIS.—In the first two years of life the prognosis is bad in all forms of pulmonary tuberculosis, largely owing to the fact that the disease is acute and tends to become generalized. The same tendency is manifested with diminishing frequency up to the age of eight or ten years. In all acute forms the prognosis is very bad. After the age of five years localized tuberculous disease of the lungs may recover, as the process tends to become more chronic and the resistant powers of the system more fully developed. Such lesions may be cured entirely, but the prognosis is rendered uncertain by the extreme liability of these patients to fresh infection.

TREATMENT.—There is only one method of treatment which is of any definite value in connection with pulmonary tuberculosis in early life, and that is prophylactic treatment. As previously stated, the symptoms of pulmonary tuberculosis usually appear, not as the result of a direct infection from without, but as an extension of disease from the bronchial glands or other part of the body in which the tubercle bacilli have already established themselves. We are therefore dealing with a secondary lesion so far as the lungs are concerned. Therapeutic measures, to be successful, must be directed to the glands already affected, and should be carried out before the lungs are involved. Here we are met with two difficulties—first, that the glandular disease is entirely latent in the great majority of cases, and, secondly, that no curative treatment of the glandular lesion is at present available. The preventive treatment, to be complete, would consist in the exclusion of tubercle bacilli from the mouth and nose of infants and children. This may be a counsel of perfection, but a great deal can be done by a wise control of the feeding and home surroundings. Fresh pure air by day and night, and fresh, wholesome, nourishing food, if generally employed, would do much to diminish the frequency of pulmonary tuberculosis in early life. On the other hand, stuffy rooms, a dusty atmosphere, and contaminated baby-soothers, are common means by which a child is infected. Relatives or nurses who are suffering from consumption often communicate the disease to infants. A diet of patent foods, starchy and otherwise, fails in building up a strong constitution, and the system becomes more liable to a successful attack by the tubercle bacillus. Attacks of bronchitis, broncho-pneumonia, and other pulmonary affections, cause enlargement of the bronchial glands, and render them less able to resist the growth of tubercle bacilli. These are the factors which must be borne in mind in connection with the preventive treatment of pulmonary tuberculosis.

The treatment of acute pulmonary tuberculosis varies with the form of disease. In the acute miliary form, no treatment is at present known which will in any way influence the disease or save the patient. In the forms of which tuberculous pneumonia may be taken as the type, the treatment should be carried out on the lines which have been indicated in connection with the therapeutics of bronchitis,

pleurisy, pneumonia, etc. We are limited practically to the relief of symptoms and to maintaining the patient's strength. It is only in the chronic and localized forms of pulmonary tuberculosis that more direct and more hopeful treatment can be carried out.

The chronic forms of the disease appear usually after the age of five years. While the treatment of tuberculosis generally is equally suited to sufferers from pulmonary tuberculosis, there is little in the treatment which is special to the lung condition. Sanatorium treatment is not available or suitable for children as a rule, but everything that is embraced in the term "open-air" treatment should be carried out. Great resistance to this will be encountered in domestic circles, and mothers will strongly object to a delicate child sleeping in the open air, or with windows wide open in all weathers, on the ground of the risk of catching cold. With a little experience of the benefit derived they will soon be converted. During the pyrexial stage rest in bed or on a couch should be maintained, but when no fever is present exercise in moderation will be beneficial. The natural movements of a child playing in a garden are better than any gymnastic exercises or physical drill. The open air of the mountains or seaside, according to individual temperaments, is much to be preferred to that of towns or valleys. At the same time, in the colder months of the year residence in a warm climate or in some sheltered country place is better than exposure to sea-fogs, strong east winds, or a constant damp atmosphere.

The diet should be a full and nourishing one, but stuffing with food is not to be encouraged. Nor is it advisable to coax a young patient to eat food when the appetite is lost. It is better to give the stomach rest for a few days, to order a few doses of calomel and a bitter tonic containing *nux vomica* and an acid. When the normal appetite returns the food will be better digested and better assimilated. The proteins and the fats should be fully represented in the dietary. Raw-meat juice, beef soup, beefsteak, and mutton, are to be ordered in larger amounts than are required in ordinary healthy childhood. Eggs, milk, cream, butter, and cod-liver-oil, should be taken regularly. The meat essences and dried milks and patent foods with strange names may be entirely dispensed with, to the benefit of the patient. The meals should not be too frequent, and if an ordinary diet is being taken, it is better to give three meals a day, with some milk between-times if desired. The appetite is very apt to become impaired if the child is dosed with cough mixtures and opiates to stop coughing. As a rule the coughing is beneficial, and a really troublesome and excessive amount of cough can usually be checked by a simple fruit lozenge, a hot fermentation to the chest, or the inhalation of steam.

The direct treatment of the disease in the lungs has not yet been successfully accomplished. The inhalation of volatile antiseptics, either from an oral inhaler or by living in an atmosphere impregnated with the vapour, is depressing to the patient, and cannot be said to be productive of any good. The use of tuberculin injections as a curative measure is still on trial, and no very conclusive results have been obtained. Apparently pulmonary tuberculosis proves more resistant to this form of treatment than most of the other forms of localized tuberculosis. At present it may be said that the best chance of healing a tuberculous lesion in the lungs is by increasing the natural resistant powers of the patient through rest, fresh air, and good food. The course of treatment must in every case be a prolonged one, and it is only in the case of well-to-do families that the care and attention which may be necessary for one or two years can be given.

PULMONARY GANGRENE.

INTRODUCTION.—Gangrene of the lung is a rare condition in early life. As a rule the disease is secondary to some preceding affection of the lungs, and is the result of a septicaemic infection. The breaking down of lung tissue and the characteristic odour of the breath and expectoration are due to the action of certain anaerobic bacteria. The gangrenous areas may be diffused through the whole lung or in both lungs, but more commonly in the case of children they are limited to one part of the lung. In this area there may be only one gangrenous patch, with softened, irregular outline, and a central cavity filled with broken-down lung tissue in a putrid condition and emitting a most offensive odour. In other cases there may be several gangrenous patches adjoining each other, often lying in close proximity to the pleural surface, and showing by their distribution that they were the result of septic emboli in some branches of the pulmonary artery.

ETIOLOGY.—The likely subjects of this condition are those weakened by disease, either general or local. Amongst the general diseases, the specific fevers, and more especially measles, are the leading causes. In the case of children there seems to be a special tendency to localized gangrene in connection with these fevers, as shown by the occurrence of gangrene in other parts than the lung—e.g., the mouth and cheeks, vulva, etc. Amongst local causes the presence of broncho-pneumonia, and to a less extent of lobar pneumonia, seems to predispose to gangrene. At the same time it is clear that some of the cases described as gangrene following pneumonia were due to a gangrenous infection of the lung to begin with, and that the so-called "pneumonia" was really an inflammatory reaction around the gangrenous area.

Septic foci in various parts of the body may lead to septic embolism and pulmonary gangrene. In children, suppurative otitis media, mastoiditis, and osteomyelitis, are the commonest causes. A putrid condition of the mouth, as in septic stomatitis or cancrum oris, may lead to pulmonary gangrene by the direct transference of organisms through the respiratory passages. The presence of a foreign body impacted in a bronchus may lead to ulceration and gangrene of the lung.

SYMPTOMATOLOGY.—There are three leading symptoms by means of which gangrene of the lung may be recognized—namely, (1) a gangrenous odour of the breath; (2) the expectoration of foul-smelling matter, containing shreds of broken-down lung tissue; (3) copious haemoptysis. The putrid odour of the breath and expectoration are quite characteristic, but must be distinguished from the offensive odour which accompanies cases of bronchiectatic cavities. Here the smell of the breath and of the expectorated pus may be very strong and disagreeable, but has never the putrid smell of pulmonary gangrene. As copious haemoptysis is a very rare condition in children, in the absence of other recognized cause, such as a tuberculous cavity or hemorrhagic disease, a sudden haemorrhage is very suggestive of pulmonary gangrene.

In many cases, however, all these leading signs are absent. The disease runs its course without these characteristic symptoms, and the illness may prove fatal without any suspicion having been aroused as to the primary trouble.

The general symptoms of acute septic disturbance are present. The tempera-

ture is high and usually hectic, the pulse is rapid, sweating and rigors may occur, anemia is marked, great prostration, loss of appetite, and wasting, are common, and complications such as diarrhea may occur. Cough is usually present, and tends to be paroxysmal in character. If there has been a pre-existing pneumonia, the physical signs of that disease will seem to afford sufficient explanation of the general disturbance. If there has been no previous lung disease, the physical signs in the chest are at first slight. There may be signs of pulmonary edema. Later some impaired resonance may follow, and the dulness may gradually get more marked. The signs of a cavity may become clear. Pleuritic friction and pain in the side may develop. Sometimes the pleura gives way, and the septic matter, reaching the pleural cavity, may lead to empyema or pyopneumothorax. None of these signs, in the absence of a direct communication between the gangrenous area and the air-passages, is sufficiently definite to make the diagnosis clear, so that at most one can only suspect gangrene from the condition of the child, which seems serious and of proportion to the physical signs.

TREATMENT.—As the signs of pulmonary gangrene may be entirely latent, and the condition may only be discovered at a necropsy, in many cases there will be no indications for treatment other than those directed to the primary source of infection, or the accompanying indications of pulmonary disorder. When the offensive odour of the breath and the nature of the expectoration, if any, have indicated the disease, the patient should be isolated, whether the treatment is being carried out at home or in a hospital ward. A gangrenous pneumonia is particularly infectious to other children suffering from lung trouble. Antiseptic inhalations by means of turpentine, creosote, etc., should be employed. If there are definite indications of empyema or a localized abscess of the lung, surgical intervention is called for. This will only be likely to succeed if the gangrenous area is near the pleural surface of the lung, with one cavity which can be opened and freely drained. If there are diffused abscess cavities, as in embolic cases, surgical treatment will probably be ineffectual. When a foreign body is the exciting cause, its removal by means of coughing or with the aid of the bronchoscope may lead to recovery, which is more likely than in other cases of gangrene, owing to the fact that these patients are usually in a better state of health.

General measures in the way of fresh air, nourishing foods, and stimulants, must not be neglected, and are often the only means at the disposal of the practitioner. As a rule an attack of pulmonary gangrene, supervening on an illness which has already severely lowered the resisting powers and strength of a child, will run a rapid course to a fatal termination.

PULMONARY ABSCESS.

The lungs may be the seat of secondary abscesses from pyæmic foci in other parts of the body—e.g., bone diseases. Certain cases of severe broncho-pneumonia are associated with multiple small abscesses in the lungs. These are forms of acute abscesses which do not lend themselves either to diagnosis or treatment.

Suppuration in the lung is also found in connection with bronchiectatic and tuberculous cavities, as in adult life. A foreign body impacted in a bronchus may lead to an abscess of the lung and a fatal result, unless the foreign body is removed or coughed up, when recovery may follow. The rupture into the lung

of an abscess in the vicinity, such as an empyema or a suppurating bronchial gland, may lead to a pulmonary abscess.

The more special type of abscess in early life to which reference may be made is that associated with pneumococcal pneumonia, usually of the lobar type. Considering how frequently the pneumococcus leads to suppurative lesions in adjoining tissues, the pleura and the pericardium, it is rather strange that it should so seldom lead to suppuration in the lung itself. Still, this complication sometimes arises in the course of or after an attack of lobar pneumonia. If the abscess is deeply seated, the clinical signs will probably be of a general character only—viz., irregular temperature, high leucocyte count, sweating, and increasing cachexia. Rupture into a bronchus may occur, and pus containing some pulmonary tissue be coughed up, with temporary or complete relief of all the symptoms. If the abscess approach the pleural surface of the lung, there will be certain local signs present. These are usually very similar to those of an empyema—namely, marked impairment of resonance, diminished breath sounds, and weak vocal resonance. From an encysted empyema or an interlobar empyema the diagnosis may be impossible, as in both cases there is a collection of pent-up pus, which in the case of an abscess of the lung lies inside the visceral pleura, and in the case of an empyema lies outside. In distinguishing an abscess of the lung from an empyema lying free in the pleural cavity, one gets some assistance from the fact that in the latter case there is usually displacement of the heart away from the affected side. Exploratory puncture may be employed, and if pus is obtained a diagnosis of empyema will probably be made, and it is not until the pleura has been opened for the purpose of drainage that the mistake is discovered. In some cases an abscess of the lung bursts through the pleura, producing a secondary empyema. If the pus is not evacuated by natural or artificial means, the tendency is towards a fatal termination from increasing cachexia and weakness.

THE TREATMENT of a pneumococcal abscess of the lung is surgical. When the abscess is single, as usually happens, the prognosis is good under surgical treatment. The abscess cavity is to be opened and drained, after its exact situation has been determined by exploratory puncture. Even if exploratory puncture has failed to localize any pus, one may feel justified from the general and local signs in advising that the pleura should be opened and the lung explored digitally. Should the pleura have been opened with the view of relieving an empyema, and no pus be found, the possibility of a pulmonary abscess as the cause of the local signs should be kept in mind and further exploration carried out.

NEW GROWTHS IN THE LUNG, PLEURA, AND MEDIASTINA.

These may for convenience be taken together. Primary growths in these structures are rare; secondary growths are much more common. A primary sarcoma may arise in the lung or in the mediastinum, where it will probably be a lymphosarcoma. Secondary tumours are usually multiple, and reproduce the character of the primary growth—e.g., melanotic sarcoma, osteosarcoma, etc. As a rule these secondary growths do not give rise to any localizing symptoms. The suprasternal or axillary glands may be involved in cases of intrathoracic tumours, but more frequently they show no enlargement. Primary tumours of the pleura are extremely rare.

The diagnosis is usually made from the physical signs, and an absolute diagnosis may be very difficult until these are well marked and the disease extensive. A new growth in the mediastinum will usually produce signs of irritation and pressure, such as coughing, distension of the veins of the chest, oedema of the upper extremities and face, dyspnoea and stridor, suffocative attacks, etc. As the tumour grows there may be manifest dulness on percussion over the sternal region and beyond it, displacement of the heart, and possibly pleuritic friction or effusion. The more rapidly the tumour grows, the more marked are the symptoms, while in the case of slowly-growing tumours it is astonishing how extensive a growth may be without causing any distress to the patient or marked pressure signs.

Tumours of the lung as a rule cannot be diagnosed until they are near enough to the surface to produce some alteration in the breath sounds or the percussion note, or pleurisy. The breath sounds over an area of the lung, which tends to increase in size, may be diminished, or absent, or bronchial in character, according to the conducting power of the new growth. The percussion note is impaired, and tends to become quite flat when the growth extends to the pleural margin. At this stage the physical signs closely resemble those of fluid effusion, and this diagnosis is frequently made. Repeated exploration with a needle, when the results are negative, should suggest the probability of a pulmonary new growth. Pleurisy is very often present either as dry pleurisy or more commonly as pleurisy with effusion, which is often considerable in amount. On aspirating this fluid, cells of the new growth may be found. The chest wall is often pressed outwards by a new growth of the lung, so that, on comparing the two sides of the chest, one can see a localized bulging on the affected side. The tumour may grow through the pleura and chest wall, and appear as an external swelling, destroying the tissue it passes through, bone included. Hemorrhage from the tumour with hæmoptysis is not common in childhood. The growth of the tumour in size may lead to the displacement of the mediastinal tissues, and pressure symptoms, such as dyspnoea, cyanosis, and dilatation of the superficial thoracic veins, may appear. Radiography may aid the diagnosis when the tumour is of some size. The course of the disease is always to a fatal termination, and if intercurrent disease, such as bronchopneumonia, does not carry off the patient, constitutional symptoms—wasting, anaemia, etc.—supervene.

HYDATID DISEASE.

Hydatid disease of the lungs is rare save in those countries where the parasite is common. The symptoms are for some time those of indefinite pulmonary disease, and they may simulate closely those of a new growth. The cysts are usually unilocular. There may be a considerable amount of coughing with mucopurulent or bloody expectoration, in which the hooklets may be found. Aid in the diagnosis may be obtained by examination of the blood, which shows a marked degree of eosinophilia, and by examination of the liver, which may contain other hydatid tumours. Pleural effusion is often an accompaniment, and renders the diagnosis more difficult. If the cyst is near the surface, and can be punctured, the characteristic fluid may be obtained. Spontaneous recovery may occur through rupture of the cyst into a bronchus. As a rule the disease terminates fatally from some pulmonary complication, unless surgical treatment prove effective by the opening and evacuation of the cysts.

ACTINOMYCOSIS OR STREPTOTRICHOSIS OF THE LUNG.

This is an affection which in many respects resembles tuberculosis in its pathological and clinical aspects, but differs in the mode of extension of the disease, which is not limited by the natural anatomical division of organs, but progresses after the manner of a malignant growth. It is properly classed amongst the infective disorders; and while the lung or pleura may be the primary seat of infection, these structures are more commonly involved by an extension of the disease from some adjoining organ—e.g., the liver. The clinical course of the disease varies greatly according to the site and spread of the local lesions. It is a rare affection at any age in this country, but the first case diagnosed during life was that of a boy of nine years. The history of this case, recorded by Sir Douglas Powell, illustrates well the clinical course of the disease and the difficulty of diagnosis, as shown in the following abstract:

The boy's father was a milker and milk-carrier, and the boy himself had been amongst cattle. Six weeks before admission to hospital he had become languid and disinclined for work or play, and had complained of some pain in the right side of his chest when touched or washed. A little later there were slight headache and a hacking cough, but no expectoration or hæmoptysis. Gradual loss of flesh, night sweats, and shortness of breath, had supervened. The right side of the chest was found to be 1 inch greater in circumference than the left, and restricted in movement. There was a tender, smooth swelling about the right nipple, obliterating the intercostal spaces, and in the posterior axillary line a larger swelling, tender, brawny, and suggestive of an early stage of pointing empyema. There was dullness over the lower two-thirds of the right lung, with absence of breath sounds and vocal resonance. From the hectic temperature and physical signs empyema was diagnosed. Two exploratory punctures were made, but only a little blood was obtained. The swellings increased in size, and there was some glandular enlargement in the right axilla and cervical region. A diagnosis of malignant new growth in the thorax was made. Later the chest was explored after resection of a rib; the finger entered a mass of soft material; some clotted, brain-like matter escaped, mixed with blood, and in this matter the ray fungus was found on microscopical examination. Some time later one of the swellings burst externally, and thick, blood-stained pus escaped, containing many white granules, as in bovine actinomycosis. These granules were also found in the sputum. Eventually the patient died from exhaustion. At the necropsy the lower part of the right lung was found to be converted into tough fibrous material, with irregular spaces filled with pink substance like altered lung tissue. The upper lobe was plentifully studded with grey nodules, like tubercles, which were also scattered over the left lung. The right pleura contained a large mass of soft material, paleaceous and casey-coloured. There was a similar mass between the liver and the diaphragm. The dorsal vertebrae and the ribs around were also involved in, and eroded by, this new growth.

SYMPTOMATOLOGY.—In the case of children the symptoms present will usually suggest either empyema or a malignant growth of the lung. Empyema is very often present as a complication when the actinomycosis has involved the pleura, although sometimes the effusion is serous. The extension of the growth into the

subcutaneous tissues of the chest wall suggests at first an abscess, and when no pus is obtained on puncturing, but free bleeding takes place, one is likely to fall back on a malignant growth as the probable diagnosis. When the apex of the lung is involved, from infection through the blood-stream, the physical signs will suggest pulmonary tuberculosis. More commonly the lower part of the lung is involved from extension through the diaphragm. The signs present will suggest bronchitis or broncho-pneumonia, or tuberculous disease.

DIAGNOSIS.—The characteristic organism may be obtained from a discharging sinus, from pus in the pleura or localized abscess, or from the expectoration. In such a case an absolute diagnosis can be made. The pus is usually viscid, with numerous white specks which, under a lens, are seen to be more or less spherical granules with a coarsely granular surface. These granules consist of the organism embedded in a layer of pus cells.

In the absence of any such material for examination, the diagnosis may be very difficult or impossible. Some chronic disease of the lung or pleura will be diagnosed on the physical signs, and there is at present no absolute means of determining its exact character, unless the history of the patient's surroundings, and possibly of some other case of actinomycosis in the neighbourhood, may suggest a correct diagnosis.

TREATMENT.—Much benefit, and even complete cure, may follow the use of iodide of potassium in full doses, one or two drachms per diem. Pleural suppuration and local abscesses must be dealt with surgically. The employment of vaccines has been successful in some cases. A nourishing diet and tonics are useful in all cases, but, unless the disease can be checked by the above measures, death from exhaustion or some complication terminates the illness.

DISEASES OF THE MEDIASTINA.

The various affections of the mediastinum, as far as we have to deal with them here, are of importance clinically because of their effect on the surrounding structures. The chief effect is caused by pressure, and this is largely a question of the size and situation of the lesion. In other cases there may be such a matting of the tissues as to interfere with the function of the surrounding structures. Again, there may be an extension of the mediastinal disease into the surrounding tissues, as in the case of inflammation, tuberculosis, and new growths.

1. Mediastinitis.—There is a peculiar form of spreading inflammation of the mediastinal tissues which is sometimes met with in children, and produces a series of symptoms which come in time to form a fairly clear clinical picture, but which may cause considerable difficulty in the diagnosis. In some cases there would appear to be some special infective organism at work, while in other cases the affection is undoubtedly tuberculous. The most common starting-point of the inflammation is in the lymphatic glands. In other cases the disease would appear to be an extension from the pericardium (mediastino-pericarditis) or from the pleura (mediastino-pleuritis). There is usually a slow extension of the inflam-

matory process, and the cells poured out are gradually transformed into fibrous tissue. This tissue hardens and contracts, and interferes with the normal action of the large bloodvessels, nerves, and air-tubes, in the mediastinum.

A boy of eight years came under observation because of swelling of the abdomen and shortness of breath on exertion, symptoms which had been present for six months. His previous health had been good on the whole. Three years previously he had an attack of pleurisy, with effusion on the left side. As an infant he had suffered from rickets and bronchitis, and at the age of three years he had been troubled with a paroxysmal cough.

The boy was rather thin, with some puffiness about the eyes, and a dusky colour on the cheeks, lips, and tongue. There was no oedema of the extremities, but the fingers and toes were blue and slightly clubbed. A prominent feature was the large globular abdomen containing a large quantity of free fluid. On dipping the liver could be felt to be enlarged. The respirations were short and rapid, but were not accompanied by any signs of dyspnoea or by coughing. There was distinct impairment of resonance on the lower part of the left lung, with deficient air entry. The heart and kidneys were apparently healthy.

As the fluid in the abdomen was increasing and the breathing was becoming laboured, the fluid was drawn off to the extent of 106 ounces. The liver was then felt to be enlarged downwards to the level of the umbilicus, and to be hard and smooth. The spleen was not enlarged.

While under observation he developed a cough, which became so paroxysmal in character that whooping-cough was suspected. Vomiting followed the coughing on some occasions, and he had most extensive subconjunctival hemorrhages in both eyes, but there was no hoop or epistaxis. The signs in the chest were those of a mild bronchitis, and there was extremely scanty, frothy, and at times blood-stained, expectoration. He had occasional attacks of pulmonary oedema, affecting one or sometimes both bases. Later there was found to be some impairment of resonance over the upper part of the sternum, which gradually extended on each side until an extensive area of dulness was present. Coarse pleuritic friction became audible in the sternal region, extending laterally into each axilla.

The boy remained under observation for more than a year, during which the disease progressed slowly. The abdomen was tapped six times in all. His general health remained fairly good, but he was unable to take any exercise because of the coughing and dyspnoea which followed.

The clinical course of the disease varies in different cases according to the structures involved. There is frequently, as in the above case, an attack of inflammation of the lymphatic glands in the mediastinum at some previous period. While these glands are becoming larger and matted together, and the inflammation is extending, there may be a latent period so far as definite symptoms go. We cannot make out these changes by direct examination, and it is not until there are some signs of pressure on the air-passages, the bloodvessels, or the nerves, that we can formulate a diagnosis. Pressure on the superior vena cava is shown by prominent veins in the arms and neck, by puffiness of the face, and by duskiuess in the lips, cheeks, and tongue. Pressure on the inferior vena cava is shown by enlargement of the liver and ascites. Pressure on the pulmonary vessels is shown by pulmonary oedema, dyspnoea, and catarrh. Pressure on the bronchi or the nerves is shown by paroxysmal cough and pseudo-asthmatic attacks. In a later stage of the disease there may be definite dulness under and on each side of the

sternum, showing an extensive consolidation of the mediastinal tissues. The signs of thickened pleura and pericardium may be found on physical examination.

The general symptoms of the disease are debility, wasting, and stunted development. There may be occasional rises of temperature at intervals, marking some exacerbation or extension of the inflammation, and there may be an irregular temperature in the tuberculous cases, but as a rule the affection is not characterized by pyrexia.

The enlargement of the abdomen with ascites is very often the first symptom to attract notice, and may lead to a diagnosis of cirrhosis of the liver or tuberculous peritonitis. In some cases a real peritonitis is present, due either to a direct spread of the inflammation through the diaphragm or to a general infection, of which the mediastinitis is a part. As other serous membranes—e.g., the pleura and pericardium—see apt to be involved, the affection is sometimes described as polyserositis or polycritheminitis. Another common symptom in chronic mediastinitis is the *pulsus paradoxus*—i.e., the pulse becomes smaller during inspiration.

The disease tends to run a prolonged course of some years, but pressure symptoms may progress to such an extent as to cause death directly and rapidly. The ultimate termination is bound to be a fatal one, and the tuberculous or non-tuberculous nature of the infection does not seem to make any difference.

TREATMENT can be directed only to the relief of symptoms. Ascites requires to be relieved by tapping as often as necessary. Severe coughing can only be checked by opiates such as heroin or morphine. Effusion into the pleura or pericardium may require to be removed. As regards medicines for the underlying disease, mercury and iodide of potassium may act as a temporary check on the inflammatory process. (See also Chapter VIII., p. 473.)

2. Abscess.—The chief cause of abscess in the mediastinum is the breaking down of tuberculous glands in the tracheo-bronchial region. Pus is formed, and as the abscess increases in size it causes symptoms of irritation and pressure. The respiratory passages in the neighbourhood may be pressed on, as manifested by paroxysmal cough, attacks of dyspnoea, stridor, and cyanosis. The signs, in short, may be very much the same as in chronic mediastinitis, and an abscess may be a complication of this affection. As a rule an abscess is characterized by an irregular pyrexia and by attacks of symptoms of acute respiratory distress, followed by remissions. In other cases an abscess of the mediastinum may be the result of extension of tuberculous disease of the spine. In the course of a pyramic illness or of some pyogenic disease of the lungs a mediastinal abscess may occur, usually through glandular infection.

While the DIAGNOSIS of the pressure symptoms may be comparatively easy, the determination of the presence or absence of pus may be very difficult. The development of acute pressure symptoms in a chronic case is very suggestive of abscess. The discovery of Pott's disease of the spine in an acute state may give valuable aid. A marked degree of leucocytosis is suggestive of pus formation.

The results of obstruction of the respiratory passages from pressure may lead to death from exhaustion following dyspnoea and cyanosis. Not infrequently the abscess bursts into some adjoining structure. Rupture into a bronchus or the trachea may lead to the rapid coughing-up of the pus and recovery, or to suffocation, or to acute pulmonary disease. Rupture into the pleural cavity will lead to the development of an empyema. Rupture into the oesophagus may be followed by

complete recovery. It is impossible to foretell what route the pus will pursue, and the best result for the patient is that it should burst through a bronchus, so as to give at least a chance of the complete evacuation and cure of the abscess.

There is no medical treatment for suppuration in the mediastinum. If an abscess can be localized with sufficient accuracy, the aid of the surgeon may be called in with a view to its being opened and drained. Aid in the localization of an abscess may be obtained by means of the X-rays.

3. Enlarged Lymphatic Glands.—The most important form of this affection has already been described—namely, tuberculous enlargement of the tracheo-bronchial glands (see p. 391). In other diseases, such as syphilis and Hodgkin's disease, there may be a definite degree of enlargement, producing irritative signs, but such is rarely the case.

In all acute and chronic pulmonary affections enlargement of the mediastinal glands tends to occur. This tendency is specially marked in connection with the pulmonary complications of measles and whooping-cough. It has been maintained by some that enlargement of these glands is the chief lesion and cause of the symptoms in whooping-cough, a theory which can be supported by a good deal of evidence. After an infective illness of the lungs, the persistence of the cough may be due to enlargement of the tracheo-bronchial glands. This view is further supported by the occurrence of attacks of dyspnoea, and spasm of the larynx, when there is no pulmonary disease or affection of the upper respiratory passages to account for the symptoms. On the other hand, one cannot help suspecting that in such cases the acute pulmonary affection had kindled into activity a latent tuberculous affection of the glands which had already existed for some time. We have at present no means of distinguishing between tuberculous and non-tuberculous enlargement of these glands. The frequency of tuberculous infection is so great that it is advisable to treat all patients with symptoms of glandular enlargement in the mediastinum as tuberculous. There is no doubt that the signs and symptoms of enlarged mediastinal glands may be present, and subside under treatment, and this probably occurs even in tuberculous cases. The whole subject of the symptomatology has been considered under the heading Tuberculosis of the Bronchial Glands (p. 391).

The softening and breaking down of enlarged bronchial glands may lead to the invasion of the adjoining hollow structures, trachea, bronchus, or œsophagus. If the gland ulcerates through the œsophagus, the contents may pass down into the stomach and recovery follow. If the gland breaks through into the trachea or bronchus, acute suffocative symptoms develop. Sometimes the whole mass is so soft that it can be coughed up, but in other cases the solid part of the gland remains fixed, and death from suffocation follows. This is one of the causes of sudden death in infants, and may occur even when no suspicion had been aroused as to the existence of enlarged bronchial glands.

4. Enlargement of the Thymus Gland.—Amongst the affections of the mediastinal organs leading to pressure on the respiratory passages must be mentioned enlargement of the thymus. Although the view that actual mechanical pressure of the trachea can be produced by an enlarged thymus has been hotly disputed, there is no doubt that such actually occurs. The trachea has been found at necropsies to be flattened out by this pressure, and during life the active symptoms have been relieved either by raising or resecting the gland. The chief symptoms are

strides, usually both inspiratory and expiratory, of a harsh, sawing character; and attacks of intermittent dyspnoea with cyanosis. The condition is met with during the first two years of life. The diagnosis may present considerable difficulty, but in addition to the symptoms mentioned above there may be definite impairment of the percussion note over the manubrium sterni. In some cases the enlarged thymus extends into the neck, and can be felt above the sternum. Radiography may show a marked shadow in the region of the thymus. There is no medical treatment for this condition, and the only hope of relief for severe cases lies in surgical intervention. The manubrium sterni may be freed from its attachments so as to relieve the pressure, or the thymus may be drawn up into the neck, and either partially resected or stitched there amongst the subcutaneous tissues. (See also Chapters IX. and X., pp. 548 and 580.)

CHAPTER VIII

CARDIO-VASCULAR DISEASE IN CHILDHOOD

E. J. FOYNTON

INTRODUCTORY.

CONGENITAL HEART DISEASE.

ACQUIRED HEART DISEASE:

RHEUMATIC HEART DISEASE.

MALIGNANT, ULCERATIVE, OR PROGRESSIVE ENDOCARDITIS.

HEART DISEASE DUE TO OTHER INFECTIONS:

SCARLET FEVER.

PERITONOCOCAL INFECTION.

DIPHTHERIA.

TYPHOID FEVER.

TYPHOID FEVER.

MEASLES.

INFANTILA.

CONGENITAL SYPHILIS.

HEART DISEASE DUE TO OTHER INFECTIONS—continued.

MENINGOCOCCAL INFECTION.

CHRONIC HEART DISEASE.

FUNCTIONAL AFFECTIONS OF THE HEART.

DISORDERS OF RHYTHM.

FUNCTIONAL BRUISES.

MALIGNANT GROWTHS OF THE HEART AND PERICARDIUM.

HEART DISEASE THE RESULT OF CHRONIC PULMONARY DISEASE.

THE HEART IN RENAL DISEASE.

VASCULAR DISEASE:

DISEASES OF THE ARTERIES.

DISEASES OF THE VEINS.

RAYNAUD'S DISEASE.

INTRODUCTORY.

ALTHOUGH the cardio-vascular affections of childhood are less varied in their nature and less complex in their causation than those of adult life, yet in this country, with its changeable climate, heart disease is not only frequent, but among the poorer classes often very severe. Those who have experience of both special and general hospitals clearly recognize how much of the chronic heart disease met with in the adult is to be dated from acute illnesses in early life.

From the first it must be emphasized that in childhood the vast majority of these cases are the result of some infection, for, without embarking upon any controversial statements as to the causation of rheumatism, it will be maintained here that the cardiac lesions of this nature are the outcome of an infective process. Congenital heart disease, together with cases the result of anaemia, dyspepsia, nervous disturbances, renal and pulmonary diseases and strain, form in comparison but a small group, and even in this we find examples where we have good reason to suspect the agency of some infective disease. To the writer this generalization appears as one of great interest and importance, for it carries with it the hope that the future will bring increasing possibilities of preventive and specific treatment.

The importance of infective processes is clearly illustrated by an analysis of 600 cases met with in an out-patient department under the writer's observation.

- In 351 cases there was active rheumatism.
 In 93 cases there was active chorea, with no obvious manifestation of rheumatism at the time of the first observation.
 In 68 cases the disease was congenital.
 In 34 — the condition followed immediately upon scarlet fever.
 In 38 — the causation was uncertain.
 In 6 — there was active tuberculosis.
 In 6 — measles was the antecedent.
 In 5 — diphtheria was the antecedent.
 In 5 — influenza was the antecedent.
 In 5 — erythema nodosum (two of these were doubtful).
 In 3 — pneumonia appeared to be the cause.
 In 3 — epilepsy appeared to be the cause.
 In 2 — renal disease.
 In 1 case congenital syphilis.

These figures cannot be used to illustrate more than the general statement that infections are of outstanding importance in the history of heart disease in childhood. They cannot be used as evidence of the relative frequency of these infections, for acute diphtheritic cardiac lesions and pericardium would not be included, such cases being limited to out-patient practice. We can, however, from an investigation such as this, extending over twelve years, get a general idea of the predominance of rheumatism in this country. Heart strain, on the other hand, is more possible of study in the public school class than in the poorer members of the community; and functional disorders are also more frequent in the well-to-do.

Tonsillitis and its relation to heart disease in childhood is worthy of special notice, for we possess some very definite data upon this point, which may be succinctly stated as follows:

1. A micrococcus found in rheumatic lesions, and capable of producing similar lesions in rabbits and monkeys, is present in the tonsils in rheumatic tonsillitis.
2. It may be present in the unhealthy tonsils of the rheumatic who have suffered from definite attacks of acute rheumatism even when the active disease is quiescent.
3. Isolated from the tonsils of the rheumatic it will produce, upon intravenous injection into animals, heart disease, including endocarditis, myocarditis, and pericarditis.

These data are significant when we put side by side with them the results of an analysis of the active manifestations of rheumatism in 500 children at the time of their first visit to hospital. The relative frequency of these manifestations was as follows:

Heart disease	350
Arthritis and pains	248
Chorea	245
Scarlet throat	157
Nodules	39
Rashes	36

The clinical association of heart disease and tonsillitis is very apparent from this analysis when it is added that no case of tonsillitis was included in which there was not heart disease and at least one other rheumatic manifestation. Clinical

observation has repeatedly proved the fact of heart disease following upon a tonsillitis, and it is not claimed for one moment that all such cases are necessarily rheumatic; but when we bear in mind the occurrence of cardiac lesions following the angina of scarlet fever, measles, and diphtheria, we cannot fail to realize how such importance must be laid upon unhealthy conditions of the throat as factors in the causation of heart affections in childhood. It is unhealthy conditions of the throat, and not acute tonsillitis only, that must be considered in this study of heart disease, and it is hardly too much to state that for the prevention of *necrosis cordis* it is one of the most promising lines of investigation we possess at the present time. More attention, also, will probably be directed in the future to the possibilities of chronic nasal discharge, or ear discharges, or pyorrhea alveolarum, as sources of infection.

The Classification of Cardiac Infections.

The prominent part taken by infections in producing heart disease in the young has an important bearing upon the method of description that will be adopted in this article, for a study of these shows that they tend to damage all parts of the heart. Some doubtless attack one part more than another, but they may all on occasion produce endocarditis. Rheumatism tends essentially to produce this lesion. Diphtheria, on the other hand, damages most particularly the non-muscular structures, while the pneumococcal infections are liable to produce pro-pericardium.

Accordingly the writer will substitute for isolated descriptions of endocarditis, myocarditis, and pericarditis, an account of the various forms of acquired heart disease under their specific causation—e.g., rheumatic heart disease, pneumococcal heart disease, etc.—following a classification to be presently defined.

This method is by no means perfect, but it possesses the great advantages that it gives a more accurate conception of any particular form of *morbus cordis* and avoids bringing the mechanical aspect of cardiac affections into too great prominence. At the same time it does not minimize the importance of the fact that in some cases endocarditis, in other myocarditis, and in others pericarditis, may be the outstanding lesion, for when such is the case the particular lesion will be dealt with in more detail. Thus, in the description of diphtheritic affections myocarditis will be specially dwelt upon; in pneumococcal affections, pericardial effusion; in rheumatism, valvular affections.

When the activity of these various infections is destroyed, and as a result scars are left in the heart, the conditions produced are very much alike whatever the original infection, and thus we have a section which includes such conditions as chronic quiescent valvular disease, chronic myocardial weakness, and adherent pericardium.

It is, however, most necessary that we recognize in childhood that such purely mechanical disabilities as these are comparatively rare, for we generally find them complicated by recrudescences of the original infection. In other words, we repeatedly find that the cause of cardiac failure at this age is not a question solely of the breakdown of a damaged pump, but a complex process dependent in part upon this, and in part upon a superadded active infection.

It will be well here to define what is meant by the term *myxædema*, which will be frequently employed. It is used as a general expression to imply heart failure from whatever cause, however acute and however transient.

Clinical Examination of the Heart.

The methods of examination in childhood are identical with those in adult life, and include inspection, palpation, percussion, auscultation, radiography, and the use of the polygraph and of electro-cardiographic investigations.

The Normal Heart in Childhood.—There are some peculiarities in the normal heart dependent upon anatomical and physiological details which require brief mention.

The weight of the heart at birth averages $\frac{1}{2}$ to 1 ounce; this doubles itself by the end of the second year. At five years the weight is 2.5 ounces, and at fourteen years 5 ounces. The relation to the general body-weight is proportionately greater in childhood, and there are two periods at which the weight of the heart increases rapidly—at the end of the first year and at puberty.

In infancy the wall of the right ventricle approaches in thickness to the left, but as the pulmonary circulation becomes established the cavity increases, although the thickness of the wall itself remains much the same up to the sixth year, by which time the left ventricle has doubled the size of its walls.

At birth the orifices of the aorta and pulmonary artery are larger in proportion to the cavities of the ventricles than at any other time of life, and the valves of the arterial system is also relatively large.

Horsmann gives the following numbers as representing the varying relations of the volume of the heart to the circumference of the aorta:

- In early infancy as 25 to 31.
- At the commencement of puberty as 140 to 56.
- At the end of puberty as 200 to 16.

From infancy to adolescence the volume of the heart increases twelvefold, that of the aortic orifice threefold.

It is evident, then, that in the early periods of life there is provision for a particularly easy systemic circulation and a low blood-pressure.

The pulse in childhood is more rapid than in the adult, and the following numbers are useful guides:

- At birth, 140 to 150 per minute.
- At one year, 120 to 100 per minute.
- At five years, 100 per minute.
- At ten years, 80 in the male, 90 in the female, per minute.

In early infancy both the rate and regularity of the pulse are easily influenced by the movements of respiration, by crying, and in sleep, and to a lesser degree this holds good throughout early childhood.

The blood-pressure, as estimated by Kolosova, averages from—

1 to 2 years,	80 to 85	millimetres of mercury.
3 " 4 "	85	" " "
5 " 7 "	90 " 95	" " "
8 " 10 "	95 " 100	" " "
11 " 13 "	100 " 110	" " "

H. D. Rolleston has shown that the blood-pressure in the aorta central differs but little from that in the peripheral arteries.

The position of the impulse and area of deep cardiac dullness have been made the subjects of many investigations, and there is general agreement that the impulse in early life lies farther to the left in its relation to the mammary line and at a higher level than in the adult. As a general rule, it may be stated that at the seventh year the impulse is about the nipple line, and below this age outside it. Sawyer gives 60 to 70 per cent. of infants one to two years of age as having the apex external to the vertical nipple line. Much, however, must necessarily depend upon the development and shape of the chest, and also upon position. In the erect posture the impulse sinks. It also moves to the right or left as the child turns from one side to the other. In the horizontal position the impulse is usually in the fourth space at the fifth year, after which it gradually sinks to the fifth space.

Physical Examination in Heart Disease.—The physical examination of the cardio-vascular system in heart disease requires, if possible, greater care in childhood than at any period of life, for at this early age we recognize how much is dependent upon accurate observation as distinguished from information obtained from the patient.

The pulse is not so instructive as in adults, because arterial degeneration is infrequent, and gross irregularities of its rate and strength are not so common. Yet it may be of much importance, as when, for example, in acute pericarditis there is costantal rapidity and progressive diminution in volume. In renal disease, again, arterial spasm can be often detected, and the blood-pressure may be obviously high.

Inspection of the chest is very helpful for cardiac hypertrophy in the young soon produces precordial bulging, and this may also be observed when there has been a copious pericardial effusion. In childhood we meet with extremely wide areas of visible impulse associated with valvular disease and adherent pericardium.

Palpation is of particular value in localizing thrills the result of congenital or acquired disease.

A high degree of accuracy can be obtained with *percussion*; and as dilatation of the heart is one of the most important of all cardiac lesions in childhood, a study of the deep cardiac dullness is of great value. The student will find it most helpful if, after light percussion, he marks out the limits thus defined with an outline pencil, for in this way he will gradually achieve great precision in estimating the size of the heart.

Auscultation should include the entire precordial area, a point of special importance on account of the frequency of pericarditis at this age. In some cases the back of the chest, and even the large arteries, should be auscultated. There are certain points, also, which may be emphasized as of particular interest.

If we are to follow Thayer, the occurrence of a third heart sound in the young and healthy is frequent. When they are examined lying on the left side or the back, it is, in fact, the rule rather than the exception. Such has not been the writer's experience, but of its existence there is no doubt. Associated with this third sound there may be a wave in the jugular tracing, and an early diastolic rise in the electro-cardiographic tracing. Hirschfelder attributed this third sound to sudden tension of the mitral valve following the swift dilatation of the left ventricle at the commencement of diastole. This third sound occurs at an interval

sufficiently long after the second not to be mistaken for a reduplication. There is no murmurous character in it, and as a rule it is audible at or close to the impulse.

Organic basal murmurs, systolic in time, are more frequent in childhood on account of the occurrence of congenital heart disease. Aortic diastolic murmurs are usually louder and more evident to the left of the middle line than over the third right costal cartilage. Multiple valvular lesions are frequent in their occurrence, and a to-and-fro mitral murmur is often met with, which is audible at the impulse and associated with mitral regurgitation, complicated sometimes with an adherent pericardium. The cantoring rhythm so frequent in adult renal disease is comparatively rare in childhood.

Lastly, we are compelled to be exceedingly careful in the examination of the child's heart, because it is then that we so frequently meet with the earliest signs of organic disease, and these are often fleeting and comparatively unobtrusive.

Modern Methods of studying Heart Disease.—The most important and suggestive method of studying heart disease in childhood is by experimental study of the pathological processes in animals. Already this method has put on sterner ground the exact pathological changes in rheumatic carditis, and in heart disease associated with chorea; the relation, also, of simple to malignant endocarditis; and the nature of myocardial changes in rheumatism, diphtheria, and other infections.

The method is one that brings with it promise of future advances both in prophylaxis and treatment. Here its services will be apparent, not only in the headings devoted to the pathology, but in the fact that the arrangement of this article has been modelled largely upon the results of its influence.

There are other modern methods which have attracted great attention, and which have helped and will help in the future to make the subject more precise.

There are two instruments which are all-important in these investigations—the *clinical polygraph* and the *electro-cardiograph*. The remarkable investigations of James Mackenzie with the former instrument, and the valuable work of Thomas Lewis with the latter, have enhanced the literature of English medicine. Two references will suffice to give the reader a clear insight into this field—Mackenzie's monograph upon "Diseases of the Heart," second edition, and Lewis's upon the "Mechanism of the Heart Beat." In these will be found also numerous allusions to the work of Continental authorities. The writer is indebted to Lewis for the electro-cardiograms that illustrate this article, and the last three of these are kindly lent by him from the illustrations of a forthcoming monograph upon electro-cardiography. To those who are unacquainted with these tracings a few explanatory details may be helpful. Kölliker and Müller demonstrated the presence of two definite electrical changes in the contracting ventricle of the frog's heart, and later Waller applied the same method to the human heart, and showed these changes with the capillary galvanometer. Bayliss and Starling registered the curves, and Einthoven by his string galvanometer greatly increased the accuracy and delicacy of the records. In the three tracings (Fig. 25, I, II, and III) there are seen three elevations from left to right, P, R, and T. P is the galvanometric index of the auricular contraction. R and T are connected with the ventricular. In No. III, immediately before R there is a dip below the basal or iso-electric line. This is called Q by Einthoven. The dip, best seen in No. I following R, is called S. Neither of these is lettered in these tracings. Q, R, S, and T are all connected with the ventricular systole.

These methods have not as yet the scope in the heart disease of childhood that they have in the adult, for they concern themselves chiefly with disturbances of rhythm, which are of more serious importance in the adult; but it would be premature to attempt to limit the future possibilities of research in this direction.

Another line of investigation, hardly modern, but one which has received a great impetus in recent years, is the study of the blood-pressure with the *sphygmomanometer*. This again, has less scope in childhood when the diseases associated with high blood-pressure are rare.

This method has value in training the fingers to accuracy in studying the pulse, and gives permanent records of the two great facts—a high and low blood-pressure—but the writer is of opinion that the subject of the variations and meanings of the blood-pressure is one that is far too complex to be sketched by methods which at the best are very primitive.

Ecdiography has an increasing value in the study of heart disease in childhood on account of the steady improvement in technique. It affords great assistance

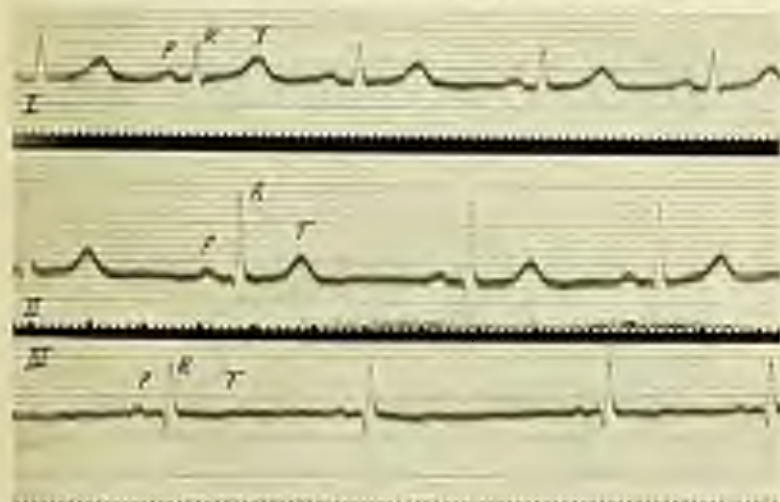


FIG. 23.—ELECTRO-CARDIOGRAMS FROM THE NORMAL HEART, ILLUSTRATING THE THREE CHIEF LEADS. (LOWIS.)

Lead No. I. is from right arm to left arm. Lead No. II. is from right arm to left leg.
Lead No. III. is from left arm to left leg.

in the most urgent and difficult problems that arise in connection with pericardial effusions, whether purulent, sero-fibrinous, or serous. It is also of assistance in the diagnosis of congenital heart disease and rare cases of intrathoracic aneurism, and new growth implicating the heart or pericardium.

General Symptoms of Heart Disease.—It can hardly be too much insisted that symptoms in childhood are frequently conspicuous by their absence or slight degree of severity, and that if we rely upon them as an index of commencing heart disease we are very liable to go astray. Even in acute pericarditis pain and distress may be wanting, and pyo-pericardium is generally overlooked even when considerable care is exerted. There is one great assistance in childhood, and that is *oedema*, which is an exceedingly valuable warning of morbus cordis. There is much to be learnt from the

facial aspect and general behaviour of the child. The cyanosis of congenital heart disease, the congestion of mitral disease, the pallor of aortic disease, and the pinched features and grey colour of virulent endocarditis, are cardinal types. Again, we must notice the curious taciturnity in cardiac weakness with dyspepsia, and the feebleness and apathy in dangerous myocardial diseases. Among other important symptoms may be mentioned shortness of breath on exertion, muscular weakness, anæmia, and wasting. Fainting attacks are not very uncommon, but they must be distinguished from *petit mal*, and at puberty they are usually a sign of functional disturbance rather than of organic disease. Nervousness is frequent, particularly when there is an aortic lesion; but if parents and medical men are tactful, it is exceptional to meet with that mental distress and lack of confidence which are such prominent features in many cases later in life. Probably because cardiac failure in the young is so often associated with active endocarditis rather than mechanical disability, and on account of the healthy vessel walls, great oedema is less frequent in childhood, although sudden swelling of the face is curiously common. Precordial sensations, angina, and dyspeptic disturbances, are less prominent in childhood. On the other hand, active rheumatic manifestations are exceedingly frequent. Symptoms of the utmost gravity are livid pallor, restlessness, vomiting, and a subnormal temperature. The liability of children to the generalization of infective processes in their tissues leads to an increased tendency to heart complications in pneumococcal and tuberculous diseases, as compared with a similar occurrence in adult life.

No difference is more striking in a comparison of child and adult than the absence of the distressing paroxysms of breathlessness at night, which are so frequent in the adult with arterial degeneration, for it is only in cases of exceptional rarity that we see the uræmic cardiac manifestations of chronic renal disease. On the other hand, the clubbed fingers and the facies of congenital morbus cordis are equally distinctive of heart disease in childhood, for though well known in adult life, they are exceptional.

Among other points of interest in the study of heart disease in childhood are the frequency of pericardial adhesion and of great cardiac hypertrophy. To these may be added the combination of congenital and acquired heart disease, the rarity of malignant endocarditis, the frequency of multiple valvular lesion and of subcutaneous nodules with heart disease. The recuperative power of the child's heart is considerable, but the liability to renewed cardiac infections is greater than in adult life. Lastly, when the circulatory disturbances commence early in life and are severe, there may be a striking arrest of growth and general development.

CONGENITAL HEART DISEASE.

Under this heading the most frequently occurring symptoms and signs and the chief varieties of congenital heart disease will be considered, and reference should be made to monographs upon the subject by those who desire to go more deeply into the question.

As a necessary introduction, some details will be given of the embryology of the heart and bloodvessels, and also of the fetal circulation and of the changes that occur in it after birth.

The Development of the Fetal Heart.—The embryonic heart commences to develop in the third week of fetal life, by the fusion of the median parts of the

two primitive anterior ventral aorta. Upon the single tube thus formed four dilations appear, called respectively the "sinus venosus," the "auricle," the "ventricle," and the "bulbus cordis." At first the sinus venosus is separated from the auricle by a valve consisting of a right and left segment, but when, as eventually happens, this chamber merges into the right auricle and coronary sinus of the complete heart, the right segment of this valve is incorporated with the Eustachian valve, and the left with the interauricular septum. The four primitive chambers of the heart lie originally in a straight line, but later two curves develop, one lateral and V-shaped, the other antero-posterior. As a result the ventricular part of the primitive tube moves forward and to the right, the auricular backward and to the left.

The further development is concerned with the formation of various septa, first of which may be mentioned the auriculo-ventricular division. The passage between the auricle and ventricle elongates into an auriculo-ventricular canal, which is then divided by the projection into its lumen of two cushions from the anterior and posterior walls respectively. By their fusion are formed the right and left auriculo-ventricular openings.

The interventricular septum, according to the most recent investigations, is formed by downgrowths of the primitive tube, a septum being left between these as they develop. It is thus formed from above downwards, and not from below upwards, as generally described. The fused endocardial cushions become the pars membranacea septi.

The interauricular septum is a complex development. There first appears a partition—the septum primum—which springs from the back of the primitive auricle, and descends towards the endocardial cushions, but in its descent this separates from the roof of the auricle, leaving an open space above its upper border. To the right of this opening there now appears a second partition—the septum secundum—which, springing from the roof and back of the auricle, extends down to the centre of the cavity, leaving between itself and the septum primum an opening—the foramen ovale—which is often imperfectly closed in cases of congenital heart disease.

Keith has directed particular attention to the importance of the bulbus cordis in the history of congenital cardiac affections. This, the fourth primitive chamber, apparently becomes incorporated with the infundibulum of the right ventricle, and it is to errors in the process that he would attribute some of the commoner malformations. Among such we have subdivision of the right ventricle into two compartments, arrest of expansion of the infundibulum, presence of the infundibulum and arrest of the right ventricle, and subaortic stenosis due to failure in the natural atrophy of the bulbus upon the left side of the heart. He would also explain transposition of the great vessels as due to reversal of the usual changes that take place in the bulbus. Normally the left wall of the chamber atrophies, and the right side expands, in forming the infundibulum; but should the right side atrophy and the left side expand, the aorta will occupy the position of the pulmonary artery, and vice versa.

This brief description will serve to show how probable it must be that some defect in the perfect structure of the heart will arise should intra-uterine life be disturbed by disease or other adverse occurrence.

The Vascular System of the Fetus.—The fetal heart when fully developed shows certain peculiarities. The foramen ovale is open and the Eustachian valve is

large; the auricular part of the heart exceeds the ventricular, and the thickness of both ventricles is about equal. The arterial system is also remarkable, in the communication of the pulmonary artery with the aorta by a short vessel, the *ductus arteriosus*, and in the continuation of the internal iliac vessels to the placenta as the hypogastric arteries.

In the venous system there are communications between the placenta, liver, and portal vein, and another between the umbilical vein and the inferior vena cava by means of the *ductus venosus*.

The Fetal Circulation.—Tracing this from the placenta, it takes the following course. Leaving by the umbilical vein, the arterialized blood enters the abdomen at the umbilicus. The umbilical vein then gives off two or three branches to the liver, and at the transverse fissure divides into two parts; the larger is joined by the portal vein, and enters the right lobe of the liver; the smaller passes on as the *ductus venosus*, and is joined by the left hepatic vein as it opens into the inferior vena cava. In the caval vein the arterialized blood meets that returning from the lower extremities, and, entering the right auricle, is directed by the Eustachian valve into the left, where it meets a small quantity of blood issuing from the rudimentary lungs. Thence it passes into the left ventricle, and by the aorta almost entirely to the head and upper extremities. Returning by the superior vena cava, the current flows into the right auricle and over the Eustachian valve into the right ventricle, thence into the pulmonary artery. A small quantity is directed into the lungs, the bulk passing along the *ductus arteriosus* into the aorta beyond the orifice of the left subclavian, mingling now with such little blood as is passing along the aorta to the lower extremities. The circulation finally ends in the placenta by passing along the hypogastric arteries. Shortly after birth the foramen ovale closes, and the *ductus arteriosus*, umbilical vein, and *ductus venosus*, atrophy and become fibrous cords.

Causation of Congenital Heart Disease.

Imperfect Development.—First among the factors in causation must be put *imperfect development*. Arrested or imperfect development depends in great part upon agencies which we cannot accurately analyse, and we are driven to support the reality of the occurrence by its association with similar defects in other parts of the body. Literature confirms this evidence. Thus, Viennet found 80 examples in 700 cases. Abbott in her collection found an average of 20 per cent. There were six examples among the records of fifty-six cases under the writer's observation. Three of these showed well-marked Mongolian deficiency, and one of these club-feet also. One had symmetrical deformities of the little fingers, one congenital pigmentation of the abdomen, one congenital familial oedema of the feet.

The most interesting association pointed out by A. E. Garrod is that of Mongolian deficiency and congenital heart disease.

Intra-uterine Infections.—There is much difficulty in ascertaining the relative importance of intra-uterine infection as a cause of congenital heart disease, but it would seem probable that gross lesions produced by these agencies are uncommon.

Various infections have been claimed, and among them the tuberculous, the typhoid, rheumatic, influenzal, pneumococcal, and syphilitic. One of the most conclusive examples of such an occurrence was furnished by a case of the writer's, in which a woman suffering from active rheumatic fever in the late months of

pregnancy gave birth to a child who was cyanosed and the obvious victim of heart disease. Death ensued in forty-eight hours, and numerous recent vegetations were found upon the mitral valve, from which strepto-diplococci were isolated in pure culture. There are some cases on record in which active rheumatism has attacked a mother during pregnancy, and a cardiac lesion has been detected on auscultation of the foetal heart, the child being born later with heart disease. An analysis of fifty-six cases, with a view to ascertaining the part taken by infections, gave the following results: In forty-two there was no clue; in nine there was rheumatism in the family, and two of the mothers had suffered from rheumatic fever during pregnancy; in three, shock, worry and severe vomiting during pregnancy were respectively blamed. In one the mother had a congenital lesion also; in one case five sisters of the mother were all said to have had "blue babies."

From this record it would be difficult to claim that infective processes take an important place in causation, but the question is not decided, for we hardly yet realize the possibilities of these processes.

The influence of alcoholism is uncertain.

Varieties of Congenital Heart Disease.

The varieties of congenital heart lesions are numerous, and may be classified as follows:

Alterations in the heart as a whole:

- (1) Displacements.
- (2) Congenital hypertrophy.

In the pericardium—e.g., absence.

In the septa:

- (1) Defects of the interauricular septum.
- (2) Defects of the interventricular septum.
- (3) Combined defects of the septa.
- (4) Defects of the aortic septum:
 - (a) Absence.
 - (b) Deviation.

Valvular defects and lesions:

- (1) Pulmonary stenosis and dilatation.
- (2) Aortic stenosis and anomalous valves.
- (3) Auriculo-ventricular stenosis and anomalies of the cusps.

Changes in the aorta:

- (1) Contraction or narrowing.
- (2) Hypoplasia.
- (3) Anomalies.

Changes in the pulmonary artery:

- (1) Atresia.
- (2) Dilatation.

Patent ductus arteriosus.

Myocardial weakness.

Anomalies of the veins:

- (1) Systemic.
- (2) Pulmonary.

Congenital lesions are often complex, as an analysis of the following 50 fatal cases illustrates :

- In 36 the interventricular septum was patent, and in 2 absent.
- In 19 the interauricular septum was grossly patent.
- In 5 the aortic septum was absent.
- In 25 the heart was enlarged.
- In 10 the ductus arteriosus was patent.
- In 11 there was pulmonary stenosis.
- In 3 pulmonary atresia.
- In 12 stenosis of the tricuspid orifice.
- In 5 stenosis of the mitral orifice.
- In 4 stenosis of the aortic orifice.
- In 5 the aorta came off astride the ventricle.

GENERAL SYMPTOMATOLOGY.—Considered quite apart from the lesions, the most important symptoms of congenital heart disease are—

- Cyanosis.
- Clubbing of the extremities.
- Dyspnoea and paroxysms of disordered respiration.
- Systolic and epileptiform attacks.
- Cardiac pain.
- Wasting and arrest of growth.
- Mental deficiency, instability, and outbursts of passion.
- Sensitiveness to cold and heat.
- Hæmorrhages, polycythæmia.

Cyanosis is the most frequent symptom, and was particularly noted in forty of the writer's fifty-six cases. It is not invariable, and may be slight and easily overlooked, occurring only during attacks of passion or in some respiratory illness of slight severity. This is a point of practical moment, for such cases may be entirely overlooked and treated for wasting, the result of intestinal or other troubles. On the other hand, in the winter months cyanosis may reach an extreme degree. There is no direct relation between the degree of cyanosis and the danger to life, for cases that show it to a high degree may survive to adult life, while, on the other hand, sallow pallor is of much graver prognosis.

Some children who are hardly cyanosed at birth and in infancy may, at the age of four or five years, when there is a greater call upon the heart, become very blue. Again, cyanosis may be paroxysmal, as is the case sometimes also with the dyspnoea, and Vaquer has recorded examples of this paroxysmal cyanosis with a normal colour between the attacks associated with a patent interventricular septum. Death, too, may occur in these attacks, and a murmur sometimes makes its appearance in the last days of life.

Much speculation has arisen as to the exact cause of this cyanosis, and it would seem that attempts have not uncommonly been made to arrive at too simple conclusions. We have apparently conflicting facts to consider. Thus, for example, in Brechet's case the left subclavian came off the pulmonary artery, and yet the left arm was the same colour as the right ; while, on the other hand, in advanced emphysema without congenital heart disease there may be extreme cyanosis. Now, in the first instance there is gross admixture of arterial and venous blood, but

to cyanosis, and in the second much cyanosis, but no gross admixture. Again, in some cases in which the heart has two auricles and one ventricle, there may be a good colour until shortly before death, and then extreme cyanosis may develop. Polycythæmia may reach a high degree when there is cyanosis, but at high altitudes in the normal person polycythæmia occurs without necessarily any alteration in colour. It would appear from clinical facts that deficient oxygenation of the blood is the essential cause of the blueness, and that this in turn is dependent upon various factors. Thus, there may be gross admixture of blood and a failing circulation, or an imperfect pulmonary circulation the result of pulmonary stenosis, or an overgorged pulmonary circulation with retarded flow in the systemic capillaries, as when, for example, the blood is deflected into the pulmonary artery and the aorta comparatively starved. It has been proved, also, that in the extremities and in the skin and viscera there may be an increased amount of connective tissue around the distended capillaries. Here, again, may be a factor in deficient oxygenation, and in the lungs this is particularly likely to result if the viscosity of the blood is increased by polycythæmia. In these various ways deficient oxygenation may be produced, and the apparently erratic occurrence of cyanosis be explained by the multiplicity of factors in its causation.

Clubbing of Extremities.—The pathology of this is dealt with elsewhere (Chapter XVII., p. 946). Here it will be sufficient to point out that it is a gradual development, and is preceded by a shiny appearance of the skin and tenderness of the nails. It is not peculiar to congenital heart disease, and may also occur in acquired valvular disease, when there is pulmonary fibrosis, and in some chronic alimentary affections; and it reaches a higher degree in bronchiectasis apart from heart disease.

Dyspnoea is of more practical moment than the foregoing symptom, and may be continuous or paroxysmal. It is increased on exertion, and this aggravation on movement affords some test of the reserve power of the heart. The paroxysmal form is closely associated with outbursts of tachycardia.

Syncope and *epileptiform attacks* are grave events, and the syncope attacks may be of extraordinary severity and prolonged duration. They may come on without clear causation, or as a result of over-exertion or abrupt changes in temperature, of passion, or digestive disturbances. In the worst attacks the child lies propped up semi-conscious and livid. The pulse cannot be counted, and the entire precordial area flutters with the turbulent action of the heart. Such attacks may prove fatal, or recovery find the heart and general state of the patient at a lower level of resistance. Following on such attacks, the writer has seen and verified the occurrence of cerebral thrombosis.

Pain over the heart is not very frequent, but it may be severe and anginal in character.

Marasmus in infancy is a frequent result, and a general arrest of development may produce a condition of *infantilism*.

Mental deficiency of the Mongolian type is sometimes present, but the most constant mental change is instability of the emotions with loss of control.

Hæmorrhages may occur from the mucous membranes in exceptional cases. In other cases they take the form of small subcutaneous extravasations.

The most important changes in the blood are polycythæmia, macrocythæmia, and increase in the viscosity. Bach records the case of a child, aged seven weeks, in which the red blood-count was 11,400,000 per cubic millimetre, and the specific gravity 1026 to 1080.

The temperature is often subnormal.

GENERAL REVIEW OF PHYSICAL SIGNS.—Physical examination of the heart is of the greatest assistance, because the most frequent congenital affections are connected with the pulmonary region and the undivided space, both of which are basal rather than apical, and situated to the left of the sternum. There may be much hypertrophy of the right ventricle, but in young children enlargement of the heart is frequently not very striking. Thrills are often felt, and are sometimes accurately localized, as in cases of patent ductus arteriosus, or, again, they may be felt over a wide area, as in pulmonary stenosis, with a powerfully-acting right ventricle. Less commonly thrills are presystolic or diastolic in time. The murmurs that are met with are usually systolic in time, though presystolic and diastolic ones also occur, and sometimes there is a continuous souffle throughout the cardiac cycle. In a series of fifty-six cases, in forty-eight the bruit was systolic; in two no bruits were detected; in two there were double mitral murmurs; in three systolic and diastolic murmurs occupied the cardiac cycle; in one there were systolic basal and presystolic and systolic mitral bruits.

The character varies. Often harsh, and loud, and audible over the entire precordial area, they may, on the other hand, be most difficult to detect on account of a peculiar puffing character closely resembling the rapid feeble breath sounds of infancy.

The most remarkable are the continuous roaring murmurs occupying the entire cardiac cycle, and usually attributed to a patent ductus arteriosus.

The intensity of the murmurs generally diminishes toward the apex. Sometimes they can be traced along the large arteries. Or, again, the maximum may be in the middle of the left axilla, or they may be audible at the back over a comparatively localized area. They may vary with posture.

Although these murmurs are of immense assistance in the general diagnosis when we come to consider the particular lesion we are confronted by such a diversity capable of producing the systolic bruit that we are left in the greatest uncertainty. Fortunately for practical purposes, it is of much greater importance to recognize the symptoms that point to grave disability of the circulation than it is to differentiate the exact nature of the defect.

GENERAL DIAGNOSIS.—There are other conditions in childhood which, although not dependent upon primary cardiac disease, may present some of the symptoms of the congenital affections; and in addition to this it is important to differentiate between congenital and acquired lesions.

A continuous cyanosis may occur in chronic bronchitis and emphysema, and in some cases of cretinism there may be extreme cyanosis of the extremities. Paroxysmal cyanosis with dyspnoea may occur in epilepsy, asthma, enlargements of the thyroid, and some laryngeal affections. Each of these conditions must be differentiated by a review of all the symptoms and the absence of any decisive evidence of heart disease. Profound anemia, as in splenic anemia infantum, may give rise to murmurs which are indistinguishable from those occurring in congenital lesions, and hectic basal murmurs occur at all ages in childhood.

Acquired heart disease may be exceedingly difficult to differentiate, particularly when it occurs in very early life and with little evidence as to its origin. Reliance must be placed upon a detailed study of the history, physical signs, and symptoms. Basal murmurs in the pulmonary region, with definite thrills on palpation and distinct hypertrophy of the right heart, favour congenital lesions,

as also do much cyanosis or considerable clubbing of the fingers. A loud basal murmur with very slight alteration in the size of the heart points to a congenital lesion. Difficulties arise when an acquired lesion is complicated by retraction of the left lung from the pulmonary cone, for in such cases there may be a singularly loud systolic pulmonary murmur; or, again, when with mitral stenosis there is also unusual fibrosis of the lungs, producing much cyanosis and some clubbing of the fingers. Congenital systolic aortic murmurs may be at their maximum intensity above the third right costal cartilage, acquired ones over this cartilage. Much hypertrophy of the left ventricle favours acquired heart disease. It must be remembered that asthma and deformities of the chest wall may complicate acquired heart disease, and that then systolic basal murmurs of considerable intensity may appear.

Lastly, a combination of congenital and acquired heart disease is not exceedingly rare, and each such case must be dealt with on its own merits, and the symptoms, signs, and course of the affection, duly weighed.

There will remain a certain number in which it is almost, if not quite, impossible to determine whether there is a slight congenital lesion or a functional cardiac bruit, and further allusion to this will be made under functional affections. If in such cases symptoms are absent, the distinction is not of great importance.

GENERAL PROGNOSIS.—The outlook in congenital heart disease is very serious, as will be seen from an analysis of fifty fatal cases.

Of these, forty-four proved fatal in the first year, one in the second, two in the third, one in the eighth, and one in the ninth.

It is clear from these cases that many are examples of complicated lesions which are either incompatible with any but the briefest existence, or favour an early death from some infection. Thus, in these 50—

- 13 died directly from the heart disease.
- 10 from general feebleness.
- 8 from tuberculosis.
- 6 from broncho-pneumonia.
- 4 from convulsions.
- 1 from cerebral thrombosis.
- 1 from cerebral abscess.
- 1 from jaundice.
- 2 after operations upon other malformations.

Such lesions as atresia of the aorta or pulmonary artery, transposition of the great vessels, or the origin of both vessels from a common trunk, are as a rule incompatible with life beyond infancy. On the other hand, pulmonary stenosis of a moderate degree with a patent interventricular or interauricular septum; a patent ductus arteriosus or patent interauricular or interventricular septum only; or stenosis of the aortic or mitral orifice of mild degree, are compatible with survival to adolescence, or even adult life.

A certain number of patients fall victims to a secondary malignant endocarditis, and some few die from a cardiac failure comparable to the asystole of acquired heart disease. In such cases dropsy, albuminuria, and pulmonary congestion with an enlarged liver, make their appearance. Many succumb to secondary infections, such as tuberculosis or pneumonia, which may be a consequence of one of the exanthemata or of whooping-cough,

GENERAL MANAGEMENT.—It is impossible that medicines can exert any directly curative effect, and the most that can be hoped for is that nature may come to the rescue during the course of development, and correct a small defect or produce a sufficient compensatory hypertrophy. In spite of these limitations, some useful assistance can be given to the parents. These children must be carefully protected against cold and against extremes of heat, and they should, if possible, live in a warm and equable climate. From the first they should be trained to avoid excitement, and to be content with a plain diet in which, if it can be digested, fat should have a prominent place. In the early days every effort should be made to control emotional outbursts by gentle discipline, but should the child have obtained the upper hand, it will become a matter of judgment as to whether it may not be absolutely necessary to give way. When the malformation is slight in degree, we must by cautious steps try to enlarge the child's activities both mental and physical, and on no account be led by fond parents to advise a life of lethargy. By quiet and patient observation we may be pleasantly astonished to find how much such children can do with their lives. Hot, stuffy rooms and exciting games and scenes are particularly injurious.

Among outdoor pursuits, such occupations as light gardening and botany may be useful. Some thoroughly trained to self-discipline can manage cycling and a suitable round of golf. Walking is the chief resource. Professions are difficult to choose, but that of a briefless barrister is, for those who can afford it, one of the best for giving an object for intellectual discipline. Public school life is seldom permissible, although some boys have done well when residing in the school-doctor's house under close observation. In the poorer classes special schools are indicated, and light employments must be chosen. One such patient at the age of forty-five was, for example, an admirable valet.

The dangers of exanthemata and pulmonary affections should be explained to the parents, and special care directed to their avoidance.

Particular Types of Congenital Heart Disease.

Pulmonary Stenosis usually associated with a Patent Interventricular Septum.—

Some degree of pulmonary stenosis is the most frequent and most practically important form of congenital heart disease. Keith found that in 70 per cent. of 185 specimens in various hospital museums there existed some abnormality in the pulmonary region. For the varieties of pulmonary stenosis, including complete atresia of the vessel, and for the associated lesions and the theories connected with their explanation, reference must be made to monographs. Here two main types may be mentioned, one arising early in fetal life from some error in the formation of the pulmonary conus, the other of later date, due to inflammatory changes in the pulmonary valves.

SYMPTOMATOLOGY.—The usual symptoms are cyanosis, clubbing of the fingers, and dyspnoea, paroxysmal or persistent. In the most severe cases any of the symptoms given earlier in this section may be present.

Cyanosis may be extreme, and this group furnishes the best examples of the *morbus cordis*.

PHYSICAL SIGNS.—There is enlargement of the right side of the heart, and sometimes precordial bulging. A systolic thrill may or may not be present, and the characteristic murmur is systolic in time and harsh in character, with a maximum

intensity in the second left intercostal space. This murmur diminishes in intensity toward the apex, and is audible in the left infra-axillary region, when there is an associated septal defect. In several cases under the writer's observation there have been two distinct murmurs, both systolic, one rasping and local in position, the other shaking and audible immediately behind the sternum, at the level of the third intercostal space. The latter was attributed to patency of the inter-ventricular septum. When the septum is open, a systolic murmur may be audible in the large arteries of the neck and upper limbs.

The characters and direction of conduction of the systolic murmur vary in different cases, and, though this is rare, pulmonary stenosis has been recorded with no murmur. The pulmonary second sound is usually weak, and must be absent when there is atresia; but, as Pearcek pointed out, in such cases the large aorta, encroaching upon the position of the atrophied pulmonary artery, produces an accentuated aortic second sound, which may be mistaken for a true pulmonary second.

The right ventricle may be greatly hypertrophied. In Fig. 36 hypertrophy of the right ventricle is indicated by the height of *R* in No. III. and its small size in No. I. Also by the depth of *S* in No. I. When the left ventricle is hypertrophied, *R* is tall in lead I, and reduced in lead III. *S* on the contrary is deepest in lead III.

DIAGNOSIS.—The first step is to decide that the lesion is congenital, and then to take into consideration marked cyanosis, clubbing, basal systolic murmur and thrill, right heart hypertrophy, and weakness of the pulmonary second sound. Considerable increase in the transverse extent of the cardiac dulness favours the diagnosis of a patent septum.

PROGNOSIS.—It would appear that when pulmonary stenosis is the only lesion middle age may be reached, but that if there is in addition a patent inter-ventricular septum, death almost always occurs before the age of twenty.

Pulmonary atresia with a closed septum is incompatible with life beyond a few months, but if the septum is open life may be prolonged two or three years.

Patency of the Ductus Arteriosus.—This gives a different clinical picture to the preceding. The ductus arteriosus, which in the foetus is about $\frac{1}{4}$ inch in length, extends from the left branch of the pulmonary artery to the under-side of the arch of the aorta, just beyond the left subclavian artery. By the second or third week of life the lumen of this tube is completely closed, and after some months is converted into a ligamentous cord. The usual explanations that are given for its persistence after birth are the existence of conditions which keep the blood-pressure high in the canal, and disease of its walls preventing the normal collapse that occurs after birth. When the duct remains patent, blood is forced from the aorta



FIG. 36.—ELECTRO-CARDIOGRAM FROM A CASE OF PULMONARY STENOSIS. ILLUSTRATES HYPERTROPHY OF THE RIGHT VENTRICLE. (LEWIS.)

into the pulmonary artery, and a funnel-shaped passage results, having its base directed toward the aorta.

The symptoms are few. Cyanosis is absent, or delayed to the last stage of life, and instead of it there may be unusual pallor. There is absence of clubbing, but dyspnea, anginal attacks, and severe paroxysms of tachycardia, may occur, and in these death may result. On the other hand there may be a gradual failure of compensation.

The physical signs, when characteristic, are—

1. Gerhardt's band-like area of dullness, which is about $\frac{1}{2}$ inch in breadth, and extends from the second costal cartilage on the left side, immediately to the left of the sternum.

2. A cap-like pulsating shadow on radiographic examination, situated upon the top of the cardiac shadow, and caused by the dilated pulmonary artery.

3. A systolic thrill in the second left intercostal space.

4. Hypertrophy of the right side of the heart, with a loud pulmonary second.

5. A remarkable murmur, rumbling or churning in character, occupying almost the entire cardiac cycle, and with a maximum in the second intercostal space on the left side. This murmur rises and falls with respiration, and this character may even be detected posteriorly at the level of the third and fourth dorsal spines, just to the left of the middle line. Carpenter held that the murmur is systolic in childhood, and develops the diastolic element in later life. The writer has noticed, as has Williams, the reverse occurrence in the disappearance of the diastolic element of a continuous murmur with advance in age. There seems no doubt that a purely systolic murmur is compatible with the existence of a patent ductus arteriosus, and fatal cases in childhood that have given the murmur occupying the whole cardiac cycle appear to be very rare.

6. The pulse paradoxus. Walker describes also capillary pulsation.

DIAGNOSIS.—This is not easy, although when all the signs are present the conclusion can generally be arrived at with confidence. The question arises as to whether an exocardial condition with anemia may produce a similar murmur. The writer has met with some remarkable cases in childhood in which there has been a history of influenza, after which a murmur of the continuous rumbling type has been apparent. In some of these previous to the illness there had been no suspicion of congenital heart disease. This murmur must be sharply distinguished from the venous hum that is sometimes met with in delicate children. With this there is no thrill or cardiac hypertrophy, Gerhardt's band, or cap-like shadow; and, further, this bruit may be much altered by flexion, extension or rotation of the neck.

PROGNOSIS.—This is uncertain, but on the whole good. Hale White describes a case that proved fatal at the age of fifty three years.

Patency of the Interventricular Septum.—There may be all degrees of this condition, from an almost complete absence to a pinhole aperture in the undivided space. The majority of the examples of this condition are included in the first group, in which the lesion is combined with pulmonary stenosis.

When the malformation occurs as an isolated event, cyanosis is often absent, but is very liable to appear should any pulmonary complications arise. There is a decided tendency to palpitation and some degree of dyspnea. In some cases, however, there are no symptoms at all.

In pronounced examples there are precordial bulging and visible cardiac pulsation, with a thrill which is systolic and often diffuse. The characteristic murmur is systolic, with its maximum at the inner end of the third left intercostal space, and with no special line of conduction. It is not modified by respiration. In some cases in which there is practically no septum there may be great cyanosis and no murmur.

A small opening is compatible with long life, and the prognosis must be largely dependent upon the particular symptoms presented by the individual case.

Patency of the Interventricular Septum.—The chief importance of this lesion is as a complication of other congenital defects. Sometimes it is an adverse factor in the course of a case of pulmonary disease. The isolated defect frequently gives rise neither to symptoms nor physical signs, but there may be a basal bruit which differs in character in different cases. Thus it may be systolic or presystolic or to-and-fro; it is not transmitted along the pulmonary artery.

Tricuspid Stenosis.—This is a not infrequent congenital lesion, occurring in twelve out of fifty fatal cases analysed by the author. The diagnosis during life is not clear, and as a rule death occurs within the first year of life.

Coarctation of the Aorta.—This lesion deserves a short mention, if only on account of the remarkable clinical picture it may produce. The term usually means a pathological narrowing of the lumen of the aorta in the region of the isthmus, that portion of aorta which lies between the left subclavian artery and the origin of the ductus arteriosus. Following Boerhaave, two types are recognized: a diffuse or infantile type, and a localized stricture or adult type.

It is the adult type that is of special interest, and according to Skoda it is the result of the extension of the peculiar tissue of the ductus arteriosus into the aorta at the point of insertion. This tissue, contracting after birth, produces the stricture.

The great features of these cases are the uneven supply of blood to the upper and lower segments of the body, and the remarkable collateral arterial circulation that is developed in the effort to balance this inequality. The principal anastomoses, according to Abbott, are by means of the superior intercostal, internal mammary, and posterior scapular branches of the transverse colli arteries above, with the first four aortic intercostal, phrenic, and superficial and deep epigastric arteries below the stenosis.

There may be no symptoms. On the other hand, there may be evidence of active congestion of the head and upper extremities, and advance in development of the upper as compared with the lower part of the body. There may also be obscure pains in the abdomen and lower extremities. The radial pulses may be unequal and incompressible, whereas the femoral pulses are weak. Masses of tortuous arteries may be present in the axilla, on either side of the sternum, or posteriorly in the suprascapular fossae. A rasping systolic murmur may be heard over the precordia, along the dilated arteries, and down the back and the heart itself may be hypertrophied and forcible in its action.

The outlook is varioux. Some die of other diseases, and the coarctation is only discovered at the necropsy. In other cases death has been sudden; in others cardiac systole with dropsy has resulted.

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 ROCHOWSKY: *Defekte der Scheidewand des Herzens*. Vienna, 1935.
 TRENT: *Lohr'sch und Orlitzky's Epochenw. Abth. I.*, 1902, p. 583.

For a consideration of the bearings of the developmental anatomy of the heart upon congenital heart disease:

- KERN: *Lectures on Malformations of the Heart*, *Lancet*, 1909, ii, 359, 432 and 519.

ACQUIRED HEART DISEASE.

RHEUMATIC HEART DISEASE.

This will be considered in detail, because it is the most frequent form of heart disease, and rheumatism attacks all parts of the heart. Similar lesions have been experimentally produced, and almost the entire field of cardiac pathology resulting from an infection can, accordingly, be studied from a *ensu* basis. Detail in this section will greatly prevent repetition hereafter, and so enable attention to be directed later to the peculiarities of the other infections.

ETIOLOGY.—The exciting cause of rheumatic morbus cordis will be considered by the writer to be a diplococcus belonging to the streptococcal group. If this explanation cannot be accepted, the only alternative at present is some hypothetical infection of unknown nature.

The predisposing causes are heredity and sudden changes of temperature—in particular raw cold after prolonged heat; in all probability, also, insufficient clothing, neglected throat affections, and all agencies that tend to produce inflammations of the throat. Females are more susceptible than males. A daybed with insufficient drainage and damp surroundings aggravates the course of the disease and favours its development, as many clinical observations of the writer's clearly support. Lastly, mal-sanitation is a predisposing factor. Rheumatic heart disease is rare under three years of age, and not frequent under five. From this age toward to twelve years it becomes increasingly frequent. There occur waves of acute rheumatism, which in some years may almost be termed "epidemic." Some of these various points in the etiology may be illustrated by the following analysis. Of 500 consecutive cases, 181 were males, 319 females.

In 200 cases in which a careful inquiry was made into the probable causes—

- In 50 there was a sore throat at the onset.
- In 31 there was clear evidence of a damp dwelling-house.
- In 23 there was clear evidence of getting wet through and chilled.
- In 2 the house was insanitary.
- 13 directly followed scarlet fever:
- 2 " " erysipelas.
- 2 " " diphtheria.
- 2 " " otitis media.
- 5 " " measles.

Of the 70 remaining cases, 32 gave a family history only, and in 38 no cause was forthcoming.

These statistics are from the poorer classes, who cannot be expected to watch their children as carefully as the well-to-do. When analysing, for example, the late Dr. Cheadle's casebooks for his articles on rheumatism in childhood, the writer found an heredity taint in 80 per cent. Admitting this difficulty, it is apparent that sore throats, cold, and droup, were prominent in 104 out of these 230 cases.

MORBED ANATOMY.—The most certain channel of infection is the inflamed tonsil, but it cannot be denied that diseased conditions of the nose and teeth and possibly some alimentary disorders, may also be avenues of infection.



FIG. 37.—SECTION THROUGH THE CRYPT OF A TONSIL FROM A PATIENT SUBJECT TO TONSILLITIS AND RHEUMATISM. (PONTON AND PAINK.)

A, Inflammatory cell; B, diplococci.

The results that follow the invasion of the diplococcus will depend, as in other infections, upon its virulence and the resistance of the tissues. Accordingly, we find fulminating cases of heart disease or single or again relapsing types. As a rule the heart affection is associated with other rheumatic manifestations, but it must be insisted that it may for all practical purposes be a solitary lesion—that is, be a *primary cardiac rheumatism*. In 19 out of 350 cases this appeared to be undoubtedly the correct explanation.

The most frequent associates of rheumatic heart disease are sore throat and arthritis, or chorea.

All parts of the heart may be affected by rheumatism, and the most characteristic lesion in severe cases is carditis. An analysis of 150 fatal cases shows the relative frequency of the gross lesions found in the severe infections to be as follows:

In 75 per cent. the pericardium was more or less adherent, and in only 3 of the 150 was the pericardium stated to be healthy. The mitral valve was damaged in 149 cases. The aortic valve was damaged in 51 cases. The tricuspid valve was damaged in 34 cases. The pulmonary valve was damaged in 4 cases.

The frequency of myocardial lesions could not be estimated from lack of notes upon the microscopy. In spite of this, gross changes were found in 34 of these cases.

Pericarditis.—The first step is the circulation of the diplococci in the pericardial capillaries, both visceral and parietal. Upon their escape from the vessels they



FIG. 38.—THE FIRST STAGE OF RHEUMATIC PERICARDITIS. (PORTER AND PAIVL.)

A part of the parietal pericardium of a rabbit dead on the fifth day from pericarditis and endocarditis produced by intravenous inoculation of a strepto-diplococcus isolated by Pasteur from a case of rheumatic pericarditis.

A, Blood-capillary; B, branch of A, with a cellular exudation around it. C, D, Diplococci creeping into the respective tissues.

produce localized inflammatory changes and swelling of the connective tissues. In virulent cases capillary hemorrhages result. Over the foci of inflammation the lining endothelium is damaged or destroyed, and exudation of fluid takes place into the pericardial sac.

In very exceptional cases the amount of fluid may be large, reaching almost a pint; but in only 12 of 150 fatal cases was it stated definitely that more than 2 ounces of fluid were found in the sac. This fluid may be hemorrhagic or sero-fibrinous. When sero-fibrinous, the exudation gives a shaggy appearance to the opposing pericardial surfaces, this being largely due to numberless local

foci of subendothelial inflammation, with corresponding endothelial necrosis. Extensive deposits are frequently found on the posterior surface of the heart. It is the writer's experience to find the displacements in films of the exudation when semi-fibrinous. In subacute cases of severity great swelling of the pericardial tissues themselves will occur, and nodular deposits are far from rare, and will be alluded to again under the myocardial lesions. In such cases the inflammatory process may implicate the external surface of the pericardium, adjacent mediastina, and pleura.

When the pericarditis is acute, but of slight virulence, the exudation may be serous, and the endothelium of the pericardium be but little damaged.

The curative processes are essentially of the nature of a fibrosis wherever there has been necrosis of the tissues. The mild cases may recover with practically no damage; the more severe lead to pericardial adhesions, which may be internal or external, or both combined. One other point must not be lost sight of; the healing may be incomplete, and foci of unhealed disease left behind, which probably explain the relapsing character of some cases of rheumatic pericarditis.

From the description that has been given, it is apparent that the changes resulting from the rheumatic infection are akin in nature to those which occur in other forms of infective pericarditis. The distinction that has always been insisted upon is the absence of suppuration; but the writer maintains that there is a rheumatic suppuration differing from other suppurations in this detail only, that the digestive and liquefying actions of the poisons in this disease are far less powerful.

Endocarditis.—In fatal cases of rheumatism this is almost invariable.

Clinical observation of slighter cases leads to the belief that the valves may escape even when there is obvious cardiac dilatation, but the actual proof is a matter of great difficulty.

There is some difference of opinion as to how the micrococci attack the valves. The writer's observations lead him to believe that this occurs by means of the coronary circulation—a view also supported by Carey Coombs.

The earliest changes point to a process arising in the subendothelial tissue. Here there is a swelling of connective tissue, leucocyte infiltration, and necrosis, with the formation of minute projections representing the early vegetations. This is explained their firm attachments, which merge seamlessly into the healthy tissue of the valve. Upon them fibrin may or may not be deposited from the bloodstream. In severe cases similar processes may be traced under the endothelium of the auricular or ventricular walls. In childhood it is rare for the vegetations to be of large size, but this point will be again considered in the section on Malignant Endocarditis (*q.v.*). When the active disease subsides, a process



FIG. 29.—A SECTION THROUGH A RHEUMATIC ENDOCARDIAL VEGETATION; MURAL. (POYNTER AND PEARL.)

Below is seen the fibrous structure of the valve; above, necrotic tissue; between, an area of cellular infiltration.

of scarring takes place, which may produce thickening or distortion of the valves, chordæ tendineæ, and muscular papillares.

A remarkable rheumatic process occurring in all parts of the heart, but particularly well exemplified in the mitral valve, is a smouldering inflammation which recalls to mind that occurring in fibrinoid phthisis. This produces mitral, and more rarely aortic or tricuspid, stenosis. It is of interest, also, because it may be apparently a solitary rheumatic lesion, although it is most frequently associated with relapsing chorea. In childhood the stenosis may reach a high degree, but this is exceptional.

The valves on the left side are the most frequently affected in rheumatism, not on account of any difference in the blood on the two sides, but because the



FIG. 45.—ACUTE RHEUMATIC MITRAL ENDOCARDITIS.

The left side of the heart is opened, and shows the mitral segments fringed with small vegetations.

(From the Museum of University College Hospital.)

mitral is the largest valve, and the aortic lies in very close proximity. The frequency of valvular lesions stands in this order:

1. The mitral only.
2. The mitral and aortic.
3. The mitral, tricuspid, and aortic.
4. The aortic only.
5. All four valves.

The frequency of multiple valvular lesion in childhood must always be remembered, and is illustrated by the following numbers from 150 fatal cases:

- In 98 the mitral only was affected.
- In 32 the mitral, aortic, and tricuspid.
- In 15 the mitral and aortic.
- In 4 all the valves.
- In 1 no lesion was detected.

The pathological changes in malignant rheumatic endocarditis are described in the section on Malignant Endocarditis.

Myocardial Changes.—The pathology becomes more complex than that of pericarditis and endocarditis, for in addition to the changes in the connective tissue there is the ventricular damage. The connective-tissue changes in the framework of the myocardium consist essentially in the production of multiple foci of inflammation in the neighbourhood of the minute bloodvessels. When there is pericarditis, these lesions are most evident in the connective tissue immediately beneath it; but they are also scattered in foci far removed from the



FIG. 41.—RHEUMATIC MYOCARDITIS: A SECTION THROUGH A PART OF THE HEART WALL, SHOWING—A, PERIVASCULAR EXTENSION; HUMAN.

In these areas, under high magnification, occur the subillary nodules of Aschoff and Carey Coombs.

surface, even when there is no pericarditis. Aschoff, Tawara, and Carey Coombs describe specific cells in the foci, and Coombs finds these cells also in the pericardial and endocardial lesions. Following Carey Coombs, these subillary nodules consist of firm areas formed by large spindle-shaped cells lying in the intermyocardial trabeculae of connective tissue. These cells are larger, but otherwise resemble fibroblasts, and are often multinuclear. Around their periphery are found plasma cells and mononuclear leucocytes. These nodules are generally replaced slowly by connective tissue. The writer is indebted to H. G. Butterfield for showing him an example of a rheumatic subillary nodule invading the atrio-ventricular bundle, in a case of acute rheumatic carditis. Clinical investigation does not support the view that extensive cardiac fibrosis occurs in childhood, except in rare cases.

The cardiac muscle is damaged severely in virulent carditis, and the rapidity of the process is well illustrated by experimental investigation. Extensive fatty degeneration is found scattered in patches through the myocardium in the fatal carditis both of man and animals.



FIG. 42.—RHEUMATIC MYOCARDITIS, SHOWING EXTENSIVE FATTY DEGENERATION.—HICMAN.



FIG. 43.—EXPERIMENTAL MYOCARDITIS.
(PAYTON AND VERNON SHAW.)

Showing fatty degeneration in the myocardium of a monkey dead of carditis and multiple arthritis, the point of intravenous injection of the diptheria isolated from a case of acute rheumatism.

Judging from the frequency of rheumatic dilatation, it is reasonable to suppose that in cases of minor severity some definite though transitory damage must be done to the cardiac muscle and cardiac nerves. The particular property of the muscle that seems to suffer most is that of tone.

Two results that may follow acute rheumatism, which are both interesting and important, are adherent pericardium and multiple serositis. The first of these—adherent pericardium—is a passive condition, in which secondary results follow from embarrassment of the heart's action. When the adhesions are internal, they produce little or no effect, but when conjoined with mediastinal and pleuritic adhesions, they are an important factor in the production of asystole.

The results of passive pericardial adhesion will be described under Chronic Heart Disease (vide p. 483).

Multiple serositis, which merges almost insensibly into the former condition, is distinguished by the occurrence of outbreaks of subacute inflammation in various serous membranes combined with adhesion of the pericardium. This condition is more frequent in cardiac tuberculosis and is described in that section.

SYMPTOMATOLOGY.—Before describing the various symptoms, the following classification will assist in making clear the lesions that may result from the rheumatic cardiac infection:

1. Acute carditis:
 - Acute dilatation.
 - Acute pericarditis.
 - Acute endocarditis:
 - (a) Simple.
 - (b) Malignant.
2. Chronic carditis:
 - Chronic rheumatic dilatation.
 - Chronic relapsing pericarditis.
 - Chronic endocarditis:
 - (a) Simple—e.g., mitral stenosis.
 - (b) Malignant.
3. Multiple sclerosis.
4. Chronic heart disease the result of heart scars:
 - Fibroid heart.
 - Adherent pericardium.
 - Chronic valvular disease.

The frequency with which rheumatism affects the heart in childhood will always remain a matter of controversy, because there are a number of cases in which some observers will hold there is a dilatation sufficient to justify the claim of cardiac involvement, which others will not accept. All, however, are agreed on the one essential point—that every case of suspected rheumatism in childhood needs a most careful examination of the heart. The writer's figures on this point, taken from a series of 500 out-patients, gave 287 examples of heart disease, and 63 of dilatation, making 350 in all. This clearly underestates the danger, for in many cases this was a solitary observation made previous to admission of the patient to the hospital.

Acute Carditis.

This is a result of the more severe rheumatic infections, and, though this is fortunately a comparatively rare occurrence, it may prove fatal in the first attack. The most acute example under the writer's care ran its course in seventeen days.

In these cases the onset is generally acute, with sore throat and sometimes epistaxis, and there may be fever, vomiting, and diarrhoea, with rapid development of breathlessness, anæmia, and pain over the heart. Signs of dilatation, endocarditis, and pericarditis, rapidly appear. Other rheumatic manifestations, including nodules, may accompany the cardiac infection, and arthritis and muscular pains generally precede the symptoms due to the carditis. If it is borne in mind that this complex lesion is the most characteristic in rheumatism, it will be less difficult to describe the acute lesions under the three main divisions of—

- Myocardial damage (dilatation).
- Endocarditis.
- Pericarditis.

It will, then, be readily understood that in acute carditis all these lesions may appear and be equally severe, or that one or other may be the more prominent, and of these acute endocarditis is generally recognized most easily, though dilatation is more frequent.

Dilatation is the most frequent of the acute cardiac affections of rheumatism, for it may occur uncomplicated, and, in addition, is always present in some degree when there is endocarditis or pericarditis. In the entire range of children's diseases, there is probably no one event of greater importance than this dilatation; for though it may subside without giving any trouble, and is very rarely fatal in itself, it may prove to be a forerunner of the gravest heart disease.

The symptoms at first are few and easily missed. If the child has not been put to bed, there are pallor with some breathlessness and a rise of temperature, the result of the infection. The physical signs are as follows: An increase in the frequency of the pulse, with a fall in the blood-pressure. The impulse of the heart moves outward and becomes feeble, and the deep cardiac dullness is increased transversely. The first sound is short, and the pulmonary second sound at the base accentuated. In many cases there develops a soft systolic murmur at, or internal to, the left nipple. It is impossible to be certain that there is not also a slight mitral endocarditis when the case is seen in this phase, but clinical experience has shown that in many instances the heart resumes its normal condition, and no trace of disease is left behind.

In the rare cases, such as those described by West, in which dilatation becomes extreme and the immediate cause of death, there develop symptoms of extreme breathlessness and orthopnea, together with syncope attacks and remarkable muscular feebleness. The physical signs are those of great cardiac dilatation.

A peculiar feature of rheumatic dilatation is the rapidity of its development, even when the child is at rest, coincident with an increasing wave of infection.

Acute Endocarditis.—Mitral Endocarditis.—In many cases the early signs are those of acute dilatation, but the systolic murmur, which is soft and blowing in character, can soon be traced outward from the nipple line toward the axilla, and later may become audible at the back of the chest. In some cases the murmur fades away in the axilla, to become audible again posteriorly at the inferior angle of the left scapula. In severe cases of mitral endocarditis there develop other physical signs: The dilatation is great and the impulse feeble; the systolic murmur almost appears to obliterate the first sound, and is loud, long, and blowing; in addition a blowing diastolic murmur at the impulse may make its appearance, giving rise to a to-and-fro bruit. This in an adult would strongly suggest double aortic disease, but it is frequent in childhood as an evidence of a severe mitral lesion. The impression is sometimes given that this to-and-fro mitral murmur is a result of prolonged and repeated rheumatism; it may, however, develop in a week or two in cases of severe heart disease, and must not be looked upon as an indication of severe mitral stenosis.

There is usually some degree of fever in acute endocarditis, and this may be very persistent. So long as there are fever and an excited action of the heart, we cannot feel secure that the lesion is not advancing; but it is necessary to remember that some other manifestations of the rheumatic state may have arisen and be responsible for the pyrexia. Among these cases of endocarditis with prolonged fever are to be found the links between simple and rheumatic malignant endocarditis.

Aortic Endocarditis.—This lesion requires very careful examination of the heart, and it is not rare. In the record of 500 cases of rheumatism, the writer has notes upon 22 cases, and has seen a considerable number more than this. Aortic endocarditis alone is, however, comparatively rare, and this has an important

bearing upon the explanations given of the secondary results produced by the lesion in heart disease. Much has been written upon secondary mitral regurgitation consequent on relative incompetence of the valve; but in many if not in the great majority of cases in childhood the mitral incompetence is dependent upon an actual mitral lesion—in some cases the aortic, in others the mitral, predominating. As a rule aortic disease develops later than mitral, either in the same rheumatic attack or more frequently in a subsequent one. It is the lesion that is most frequently overlooked, and its occurrence may be associated with no apparent illness. A slight rise of temperature may be the only warning.

Aortic regurgitation is the usual result of acute aortic endocarditis, and aortic stenosis will be considered with mitral stenosis under the chronic forms of rheumatic carditis.

The physical signs are the following: The aortic second sound at the base becomes faint, and a soft systolic bruit may appear. This usually disappears, and a diastolic murmur, soft, blowing, and short in duration, becomes audible. This murmur may be missed on account of its softness, or because the fact is overlooked that it may be frequently heard more plainly in the third left space or behind the sternum, or even over the xiphisternum. When the lesion is severe, capillary pulsation and a collapsing pulse with facial pallor may become obvious in a fortnight. There are, undoubtedly, lesions of the aortic valve so minute that they give rise to no physical signs, and on the other hand, as in some cases of mitral regurgitation, a slight damage, giving rise at first to physical signs, may eventually disappear.

If there is decided aortic regurgitation, as the heart recovers from the acute process, lengthening and hypertrophy of the left ventricle and a forcible impulse will be apparent.

As has been already stated, in most cases we shall find the double lesion of aortic and mitral incompetence, and this group of cases is one of great importance and gravity in the heart disease of childhood. Another group of cases of much interest are those in which with the aortic disease there has been also serious myocardial damage, for it is these which sometimes develop repeated attacks of angina pectoris, even at an early age.

The development of acute tricuspid or pulmonary endocarditis in rheumatism is not sufficiently definite to give rise to obvious signs, and the latter is extremely rare.

Lastly, if there is one point more than another that stress may be laid upon in this section upon acute endocarditis, it is the excited action of the heart as an indication of active damage.

Pericarditis.—The occurrence of pericarditis in a first attack of acute rheumatism, with but few exceptions, points to a severe infection. It is, however, a lesion which is more likely to occur when the heart has already been damaged by previous rheumatic attacks, and is then the most frequent cause of death.

SYMPTOMATOLOGY.—The violence of the symptoms varies most remarkably. There may be no pain and but little dyspnoea, and the pericarditis may come as a revelation to the physician when examining the heart in the usual routine. On the other hand, there may be great precordial pain, and dyspnoea is frequent. There is a moderate rise of temperature, the average being about 100° to 102° F. The aspect is usually pale, and the expression anxious. Delirium is

stressed, and should arouse suspicion of the supervention of acute chorea, or possibly salicylate-poisoning. The attitude of the patients differs. In the worst cases the child may lie flat on the back, but more usually there is some degree of orthopnea, and sometimes, although this is more usual in the more chronic cases, the child turns on his side or leans forward upon a bed-rest.

The respiration may be much hurried and suggest the presence of pneumonia. Occasionally a most harassing cough causes much distress, as may also pleuritic pain. In virulent cases ghastly pallor develops, and with the approach of asystole, restlessness, vomiting, and intense dyspnoea supervene.

PHYSICAL SIGNS.—The pulse is rapid and compressible, and in the later stages of fatal cases becomes irregular or running. The blood-pressure is low. Pericardial friction is usually present, although in exceptional cases of anterior adhesion and posterior inflammation it may be missed, or, again, occasionally it may be as soft in character and evanescent in duration as to be overlooked.

The friction murmur is usually to-and-fro, but may be systolic only in time. As a rule it is first recognized at the base of the heart, then rapidly becomes general, and is modified by slight pressure with the stethoscope. A cantering rhythm may precede the appearance of the friction or accompany it. The excited action of the heart attracts attention, as does also a rapid increase in the precordial area of dullness, an increase almost entirely due to a rapid dilatation of the heart, and not to a sudden and copious pericardial effusion. When more fluid than usual is poured out, some muffling of the heart sounds becomes apparent, and in exceptional cases there may be all the important signs of a considerable pericardial effusion (q.v. under *Pyro-pericardium*, p. 465).

When, as a result of weakening of the heart wall, asystole commences to show itself, the liver becomes enlarged and tender, the bases of the lungs congested, the ankles oedematous, and the urine albuminous and scanty in quantity. Endocarditis is almost invariable in rheumatic pericarditis.

In fatal cases death is often quite sudden, or may be preceded by all the signs of asystole; vomiting, livid pulset, and restlessness, with a running pulse, in particular being sinister symptoms. The temperature may fall to subnormal.

With recovery the pulse and respiration rate slow, the dilatation subsides, the colour improves, and the temperature steadies. If there has been a considerable amount of fluid exuded, friction may sometimes reappear for several days, when the roughened surfaces come once more in close contact. The duration is various; death may occur in a fortnight, or clear signs of recovery be evident in the same time; the illness is, however, always a matter of weeks, and sometimes of months.

When an attack of acute pericarditis occurs in a child already the subject of previous cardiac rheumatism, the symptoms are of the same order, but in a considerable number of cases there may be difficulty in deciding at first as to the actual development of pericarditis. The excited heart and breathlessness may make such an onset suspicious, but it may happen that a previous attack of carditis has obliterated the pericardial cavity anteriorly, and the recent inflammation be restricted to the posterior surface. It is seldom that the friction rub is audible at the back, and thus the occurrence of the pericarditis may remain doubtful. Fortunately, if due importance be attached to the rapid beat, the dilatation, the fever, and dyspnoea, the treatment is in no way influenced by this uncertainty.

Chronic Rheumatic Heart Disease.

The morbid anatomy of acute rheumatism shows that, although the acute lesions may be repaired, usually with the formation of scar tissue, there remain many cases in which the healing processes are imperfect. In these cases, amid the fibrous tissue formed in the processes of healing, whether in the pericardium or valves, there remain areas of necrotic tissue not invaded by fibro-blasts, which must be looked upon as foci of danger. This point is well shown in chronic pericarditis, when not uncommonly the anterior part of the heart is found firmly adherent to the pericardium, while on the posterior aspect there is active disease. Clinical experience also makes it clear that in some cases the cardiac muscle is damaged to such an extent that a condition of chronic myocardial weakness results.

Accordingly we meet with an important class of cases in which the carditis smoulders on, breaking out from time to time, and sometimes lasting for many months, or even years.

If the processes are not only persistent but also severe, there arise conditions such as malignant pericarditis and malignant rheumatic endocarditis. When the processes are less severe but persistent, there develops a slow adhesive pericarditis, with possibly indurative mediastinitis, and in the valves there develop the various forms of stenosis—mitral, aortic, and tricuspid.

This group of cases links up acute carditis from a first attack of rheumatism with the cases of acute carditis that supervene upon a previous attack, which has completely quieted down before the new infection arose.

Chronic Rheumatic Myocardial Disease.—These cases are not very common, for endocarditis is almost always present in addition.

They are characterized by some irregular fever, with anæmia, nervousness, general debility, breathlessness, and sometimes syncopal attacks.

The physical signs are—A pulse which is rapid, easily excited, and often irregular. The cardiac impulse is displaced outwards, and is feeble and diffuse. There is no evidence of endocarditis or of active pericarditis or an adherent pericardium.

They are cases which run a very tedious course, and are likely to suffer severely from any overstrain. The symptoms, contrary to the usual rule in childhood, appear to be more serious than the physical examination of the heart would suggest, and there is a decided danger that the real nature of the case may be overlooked, or that the condition may be mistaken for a purely functional disturbance.

Chronic Endocarditis.—The malignant type is considered under Malignant Endocarditis. The simple type is typified by mitral stenosis.

Mitral Stenosis although it seldom reaches a high degree in childhood, is a lesion of great importance, both on account of its frequency and the permanency of the changes.

It may be practically a pure stenosis in which the orifice, button or funnel shaped, is surrounded by the thickened and welded valve segments and the shortened chordæ tendineæ. Experimental investigation has produced the inflammatory changes in the musculi papillares and chordæ, which no doubt antedate their

stenosis. The process is so slow that it is often impossible to be sure when it is advancing or when it is quiescent, although there is no doubt that after reaching a certain degree in early life it may remain stationary for many years. Lastly, although sometimes the stenosis is from the first post-cumant, the majority of cases are examples of mitral regurgitation with some degree of narrowing.

Considerable doubt has arisen as to the causation of pure mitral stenosis arising in early life, and it is noteworthy that the patients often give no history of any illness to guide us. Some have thought congenital syphilis, others tuberculosis, and others rheumatism, to be the agent, and the writer believes the last to be the most frequent. The more closely rheumatism is studied, the fewer cases of mysterious mitral stenosis are discovered.

An Analysis of Fifty Cases of Mitral Stenosis under Personal Observation.—In every case chosen for this purpose there was a presystolic thrill, rumbling murmur, short first sound and accentuated second pulmonary sound. Thirty-six were in females, fourteen in males.

One was undoubtedly congenital, in a male of seven months, who died of broncho-pneumonia, the necropsy showing extreme mitral stenosis.

One, a double mitral case, also in a male, was almost certainly congenital.

One patient, a female aged eleven, showed marked infantilism, and the lesion was possibly congenital.

In these three cases the only point in the histories was the occurrence of tuberculosis in the father of the third patient.

Fourteen of the cases appeared to be examples of pure mitral stenosis.

In seven of these there were histories of prolonged and repeated chorea.

In six there were histories of prolonged rheumatism.

In one there was no history.

The remaining thirty-three were cases with double mitral lesions.

In twenty-two there were histories of prolonged or repeated chorea.

In nine there was a history of prolonged rheumatism, but in two of these of pains in the limbs only.

In two no history was obtained.

Thus, of fifty cases, in only six was there no history of rheumatism, and three of these were probably of congenital origin.

It is upon this evidence that the writer inclines to the rheumatic origin of most cases of mitral stenosis in childhood.

In the great majority of these the lesion was observed to be slowly developing after many months or some years from the first note that was made upon the condition of the heart. The first clear evidences of stenosis may commence at a very early age—for example, in the third, fourth, or fifth year. In the early stages there are often no cardiac symptoms except when there are outbreaks of rheumatism, when the result of the active infection is to favour the appearance of some signs of systole. In others there is from the first a remarkable tendency to tachycardia and irregularity of action, and with these nervousness and palpitation.

In all advanced cases the symptoms are much alike. A congested aspect, with high-coloured cheeks and full lips, some cyanosis, palpitation, dyspnoea, tachycardia, heart pain, and a tendency to repeated bronchitis.

In exceptional cases, quite early in the progress of the disease there may be such evidences of infarction, as hæmoptysis, hemiplegia, hæmaturia, and pain over the spleen, but as a rule such events occur when the age of childhood is passed.

PHYSICAL SIGNS.—The signs of mitral stenosis vary a good deal in their development. When the stenosis is pure and there has been no definite antecedent history, there is found a presystolic localized thrill, a long rumbling presystolic murmur, a short sharp first sound and an accentuated pulmonary second sound.

Much more frequently the first sign is a systolic murmur, followed by the mid-diastolic sound that gives rise to the well-known "lib-tig-tat"; then later a presystolic thrill and murmur may follow, or the murmur may be mid-diastolic or diastolic, but rumbling in character.

Then, again, there are cases in which previous to the development of any presystolic murmur the original systolic bruit disappears, and leaves a short first sound at the impulse, with an accentuated pulmonary second sound at the base. In such cases one is very liable to overlook these details, and to think that with the disappearance of the systolic murmur a cure is resulting. When the stenosis is severe the pulse has a small wave, and the radial artery feels tense, although the rhythm is usually regular until the evidences of auricular fibrillation begin to make their appearance.

Aortic Stenosis.—This is rare in childhood, only two cases being met with in a series of 500 cases of heart disease. One was in a male, the other in a female. In both the symptoms were slight, but a tendency to syncope and some heart pain, with breathlessness, was mentioned by the parents.

The pulses were diminished in rate and volume.

There was a loud, harsh systolic aortic murmur conducted into the vessels of the neck, with a systolic thrill over the aortic cartilage. The left ventricle was hypertrophied and the impulse heaving.

In both these cases there was a history of protracted rheumatism.

Tricuspid Stenosis is associated with mitral stenosis, and in childhood is hardly possible of diagnosis, for at this age the presystolic mitral murmur may sometimes be clearly audible in the tricuspid area.

Chronic Rheumatic Pericarditis.—The malignant type can be most easily illustrated by a characteristic case.

A boy aged seven years had a severe attack of enditis a year before, and had never been well since. Three weeks before coming under observation he had been ill with multiple arthritis and precordial pain. There was severe mitral disease and many nodules appeared. His illness lasted eight and a half months, during which time the temperature ranged from 104° F. to normal. He grew more anæmic and short of breath, and at intervals pericardial friction appeared and then faded away. Arrhythmia gradually developed. The necropsy showed former mitral and tricuspid endocarditis. There was great thickening of the pericardium, and many recent adhesions were also present.

The more chronic forms show no violent symptoms, but great persistence. The leading features are—Slight fever, precordial pains, and localized pericardial friction, which may last for weeks and then disappear for a while. Partial recovery may occur, with signs of indurative mediastinitis, or, more rarely, of multiple serositis, or death may finally result from a gradual asystole. After death great thickening of the pericardium is found, and scattered amid the fibrous adhesions areas of recent disease.

In all these prolonged and intractable cases, which constitute a clinical group of themselves, there is a marked tendency to the appearance of subcutaneous nodules.

Chronic Adhesive Pericarditis in "Still's Disease."—This lesion is perhaps most conveniently mentioned here. In some of the fatal cases, at the necropsy, a complete adhesion of the pericardium has been found, the adhesions being delicate and forming a film attaching the two layers. There are apparently no conclusive clinical signs that point to its supervention in these cases.

In some cases of hemophilia a pericarditis resembling the rheumatic form may occur and help to produce death.

Chorea and Heart Disease.

It is only out of respect to tradition that the writer makes this a separate section, and until the view is obsolete that chorea is a cause of heart disease it will serve to recall the frequency with which these two symptoms of rheumatism are associated. Allowing that all chorea is not necessarily of rheumatic origin, we err on the side of probability if we claim all cases of chorea with heart disease as rheumatic.

The irregular action of the heart that is often met with in chorea is very possibly part of the general nervous instability, and to this extent chorea may in itself be a cause of cardiac disturbance, but this irregularity is so transient and apparently harmless that it can be hardly claimed to be a disease. This irregularity is intimately associated with respiratory irregularity.

Some figures from the writer's notebooks upon chorea, heart disease, and rheumatism, are instructive.

Taking 500 consecutive cases of rheumatism, heart disease, and chorea, 23 came with chorea as the leading symptom. Of these, 122 had obvious organic heart disease; 38 more had dilatation of the heart, rheumatic arthritis, and pains; 10 followed immediately upon a sore throat; 20 more gave a decided family history of rheumatism; 15 were attributed to fright and shock, 2 of them, to the writer's knowledge, developing heart disease later; 15 gave no clue as to causation, one of these succumbing three years later to carditis, and one being hopelessly crippled from the same cause.

These numbers confirm once again the close association of chorea with rheumatism and heart disease.

There is no necessity to recapitulate the history of heart disease with chorea, for it has been already dealt with under Rheumatism. One or two points of interest only need emphasis.

Chorea and heart disease are more frequent in female children. Two hundred and eighty-four consecutive cases of chorea were made up of 202 females and 82 males. The form of heart disease particularly associated with chorea is mitral stenosis, and it is in female children that the early development of this condition is best studied. In the writer's opinion, the explanation for this predisposition of the female must be sought in some mysterious difference in the metabolism of the female which alters the course of the infection. This is also suggested by the fact that acutely fatal carditis is, on the other hand, more frequent in the male. In 500 cases of rheumatism 26 were fatal. Of these 500, 313 were females, with 14 deaths; 181 were males, with 22 deaths—that is, 4.4 per cent. in the female, as against 12.1 per cent. in the male.

The association of severe carditis and severe chorea is a most dangerous one, and the supervention of a severe chorea upon an attack of carditis produces imminent danger, the muscular exertion rapidly telling upon the recently damaged heart.

When a child who suffers from repeated relapses of chorea is very young at the time that the first attack commences, a slowly progressive mitral stenosis is an exceedingly likely occurrence. So slow is this process of valvular change, and so latent the symptoms, that it may not be possible to prove the presence of an active lesion in the heart, but the clinical evidence of its obstinate rests upon the fact that each year in which there is a relapse of chorea the condition of stenosis will be found to have advanced.

Lastly, there may be remarkable tachycardia of a very persistent type in cases of chorea. This tachycardia always suggests the supervention of a mitral stenosis; but it is certainly not always dependent upon the stenosis, for it may date from the first illness, and persist for months before there is any reason to suppose that there is evidence of contraction of the valve. The pathology of the persistent tachycardia has not been explained, but it is associated with considerable nervous instability, and its occurrence early in the history of a child with rheumatic heart disease adds much to the gravity of the outlook as regards a good recovery.

Rheumatic Multiple Sepsis.

It is singular how rare this is in rheumatism when it is remembered how frequently the pericardium is damaged, and how often rheumatic pleurisy is associated with recurrent pericarditis.

The probable explanation is that in order to get the true picture of this disease an exceedingly slow and dense form of pericardial adhesion is needed. Such a firm of adhesion strangles the heart, which may at the necropsy be found smaller than usual. In rheumatism, though pericardial adhesion is frequent, it is not as a rule dense, and is usually associated, not with a small, but with a large and hypertrophied heart. The symptoms of the condition are described in the section upon Cardiac Tuberculosis (p. 473), with which the most characteristic cases in childhood are associated.

DIAGNOSIS OF RHEUMATIC HEART DISEASE.—In many cases, owing to the concurrence of other rheumatic manifestations, diagnosis is easy; in some it is only a question of probability, and in the remainder it is both difficult and important.

It is the last group that needs consideration, and the chief questions that arise are the following:

1. Is the condition organic or functional?
2. If it is organic, is it the result of congenital or acquired disease?
3. If it is the result of acquired disease, is this rheumatic or due to some other infection?

1. The differential diagnosis between rheumatic heart disease and functional affections is likely to be difficult when the rheumatic lesion is chiefly myocardial.

All facts bearing upon the case must be collected, and the schema given under Functional Affections of the Heart (p. 489) will be found useful.

The occurrence of an attack of rheumatism is always suspicious, and it is essential to remember that functional disturbance may complicate organic myocardial disease. The persistent appearance of dyspnoea on moderate exertion, with irregularity of action and a poor cardiac first sound, are greatly in favour of organic disease, for in functional affections it is repeatedly noticed that exertion does not seem to embarrass the child. Edema of the ankles and enlargement of the liver, with some obvious dilatation, are signs which, whether dependent upon strain of

infection, demand like treatment; but if, in spite of this, the heart refuses to maintain its strength, this fact favours organic myocardial disease. Fever always suggests an organic cause.

The occurrence of signs of mitral stenosis or of aortic regurgitation is sure proof of organic disease. On the other hand, irregularity of action, whether respiratory or dependent upon change of posture, is compatible with a normal heart.

Fainting attacks may occur both in organic and functional affections, and also as a reflex disturbance in some nervous children quite apart from any heart affection. There is no way of coming to a correct conclusion short of a general review of the entire case.

It is well known that a great number of so-called "fainting attacks" are not cardiac, but epileptic, in nature, and the differential diagnosis is of the utmost importance. As a rule the momentary loss of consciousness, the abrupt onset and recovery, and the absence of all other cardiac symptoms, make the distinction easy. A few cases remain in which it is almost impossible to decide the point. For example, epilepsy and organic heart disease may be associated, or the attack of "fainting" may be preceded by an aura which is most suggestive of a primary cardiac event, on account of the intense sensation of sinking, nausea, pallor, and occurrence of cold sweats.

2. The main distinctions between congenital and acquired heart disease have been considered under the section of Congenital Heart Disease (p. 428).

3. The question as to whether a case of acquired heart disease is rheumatic or due to some other infection is sometimes exceedingly difficult. There will be found many points bearing upon this under the sections of this chapter dealing with the various infections.

It is useful to remember that acute carditis is pre-eminently the type of a rheumatic lesion.

When myocardial damage is prominent, diphtheria and influenza are particularly likely to come into the question of diagnosis.

In influenza, fever, muscular pains and sore throat are frequent, but there is very rarely actual arthritis, there are no nodules, there is no chorea. The thickly coated tongue is suggestive, and the general absence of valvular or pericardial lesions and presence of intense depression favour this diagnosis.

In diphtheria, the history, the result of examination of the throat, the absence of endocardial and pericardial lesions, and presence of signs of paralysis, are of special importance.

Where pericarditis occurs with friction, rheumatism is the most probable cause, but tuberculosis, pneumococcal affections, and lympho-sarcoma, may each produce pericardial friction. The differential diagnosis then depends upon a study of every detail in the particular case.

When there is pericarditis without friction, the diagnosis becomes more difficult, but proceeds upon the same lines.

When endocarditis is the chief feature, the question arises as to whether this is simple—that is, healing—or progressive and malignant.

The distinctions are given under the Symptomatology of Malignant Endocarditis (p. 455). Bacteriological examination is valuable, but a warning is necessary upon this point. On several occasions Paine has isolated the rheumatic streptococcus from the blood-stream in cases which necropsy later proved to be

examples of simple endocarditis, and, on the other hand, cultures may be negative when the case is undoubtedly malignant. There is a great danger in these days of trusting to one bacteriological sign of disease, just as formerly mistakes arose from reliance upon one so-called "pathognomonic" clinical sign in medicine. Undoubtedly the isolation of a micrococcus from the blood-stream in cases of endocarditis is a most valuable aid in the diagnosis of malignant endocarditis; but it should be coupled with the other clinical signs of this affection, and should not in itself be looked upon as a proof of malignant endocarditis in childhood. It may also be added that if the endocarditis is malignant this is no proof that it is not rheumatic.

Prognosis.—The prognosis involves two distinct problems:

The first is the outlook for the present illness; the second, the outlook for the future. Unfortunately, the second of these contains an element of great uncertainty, is the liability to further acute attacks.

In general terms, the prognosis as to recovery in a first attack is good.

Pericarditis in a first attack is grave, and those cases which begin acutely with vomiting and diarrhoea, and many rheumatic manifestations in very young subjects, are serious indeed. The rapid development of anæmia, general pericarditis, and much dilatation, with outbursts of nodules, should they be accompanied by restlessness, vomiting, and lividity, point to a fatal termination.

The superposition of pericarditis after severe chorea is an exceedingly grave complication.

Another group of cases of less immediate gravity, but in which the future is gloomy, are those of carditis in which the damage is so great that compensation is never satisfactorily established, or in which repeated outbursts of pericarditis occur with no true intermission, and in which nodules are also frequently appearing.

On the other hand, slight cases of pericarditis undoubtedly occur in which there is practically complete recovery. They are easily recognised by their evanescent course and the quick return of the heart to its normal dimensions. There was active pericarditis in all the 26 fatal cases of a series of 500 examples of rheumatism.

Myocardial damage requires even now more clinical observation before we can estimate its exact prognosis. There are some cases occurring in soft, flabby, anæmic subjects in which the heart appears to remain permanently enfeebled. In other cases there is a slow progress to recovery which is easily set back by impudence; yet after a year or two the recovery seems to be complete.

Of nodular lesions, severe combined aortic and mitral regurgitation is very serious, and so also is severe aortic regurgitation alone.

The rapid development of severe mitral stenosis in the very young is another valvular lesion in which the outlook is bad, and this is also true of severe mitral regurgitation with great loss of tone in the cardiac muscle.

On the other hand, many cases of slight mitral endocarditis make an excellent, and some few undoubtedly a perfect, recovery. This is also true, in the writer's opinion, of some cases of aortic disease.

Mitral lesions may even, if permanent, be perfectly compensated, and, if no further rheumatic attack occurs, are compatible with a long, full, and useful life. A moderate degree of mitral stenosis is well known to remain quiescent for many years, although it must be admitted that this lesion, if definite in childhood, carries with it considerable uncertainties in connection with the child-bearing period of life.

Puberty in itself has not seemed to the writer to influence these organic lesions adversely, for in most instances this process is so gradual and natural that the heart readily adapts itself to the change. When, however, anemia follows, symptoms are likely to arise, and malignant endocarditis becomes more frequent.

In many cases the occupation after childhood alters the prognosis for the poorer classes, and in any case a renewed rheumatic infection may nullify the most hopeful opinions for the future.

TREATMENT.—The treatment of rheumatic carditis in childhood is unsatisfactory, and the salicylates appear to the writer to have very little, if any, effect, whether they are given in small, moderate, or large doses; but, out of regard for the opinions of others, the details will be given of the "specific" salicylate treatment. The most encouraging feature of the present time is the increased stimulus to prevention which must follow the demonstration of the infective nature of the disease, and the possibility that new methods may be evolved for dealing with this urgent problem.

GENERAL MANAGEMENT.—In severe cases the path is clear, but in the mild there is a great practical difficulty dependent upon the insidious character of many cases of early heart disease. The absence of symptoms leads both parents and friends to reject advice, either as alarmist or even as made with an ulterior object.

Rest is the first essential, and these children should be protected from draughts. Rest will not prevent the development of pericarditis, as was illustrated by a patient who, though he had been kept in a Phelps's box for over a year with suspected spinal caries, developed two attacks of acute rheumatic pericarditis during that period of time.

The nightdress should be of all-wool flannel, and, although sweating is so feature of the disease in children, they lie comfortably and are safer between blankets. In the bad cases skilled nurses are valuable, for much nursing is required. Food should be liquid, and consist of milk suitably diluted, or made more digestible by adding citrate of soda (gr. i. in $\frac{3}{4}$ x). It may be necessary to have recourse to peptonized milk, or even ass's milk, in the worst cases. Provided that the kidneys are sound, there is no objection to some beef or chicken tea. The popular and widespread idea that meat foods are bad for the rheumatic appears to be fallacious so far as children are concerned. Alcohol is not usually required, but it is of value when the vitality is low, and the notion that it damages the heart and is detrimental is contrary to clinical experience, provided that it is given carefully and for a definite purpose. Half an ounce of brandy in twenty-four hours is an average quantity for a child of seven years.

When the attack lessens in severity the diet should be carefully increased, for children do not do well when kept for a long time on a liquid diet, and, provided the digestion is good, there is no apparent need for such a restriction.

Vomiting, when severe, may require all food by the mouth to be stopped for twenty-four hours, and rectal feeding employed.

LOCAL MEASURES.—These have been chiefly advised for pericarditis, but some have believed that mild blisters applied to the cutaneous branches of the first four dorsal nerves have a deterrent action upon endocarditis. To the writer, in view of the pathology of the disease, such a result seems unlikely, but they are distinctly helpful in obtaining the necessary rest.

In pericarditis with acute pain, warm fomentations and antiphlogistics or kaeoplasts are comforting, and suit nervous, weakly subjects.

When the pain is severe, nothing relieves it better than an ice-bag or leeches. Loos believes the ice-bag possesses in addition some curative action. If it is used careful nursing is required, and it is not suitable for the asthenic patient with a low temperature.

Constant application is most serviceable, and for the first twelve hours it is advisable to take the temperature every two hours. Strict orders should be given as to its use, and the area of application indicated. Two bags are advisable, the one to replace the other. Only the finest layer of gauze should lie between the chest wall and the ice-bag, and care should be taken to see that the bag does not leak. When there is much precordial tenderness, the bag should be hung from a cradle, so as to bear lightly upon the chest.

One of the best ways of fixing the bag is to pass the neck of it through a hole made in the vest, and fasten it by a flannel band passing round the neck. The lower border of the vest is fixed to a binder. In all cases hot-water bottles should be placed near the lower extremities.

Four leeches applied to the precordia will also relieve pain in most instances, but in childhood the most important indication for their use is the occurrence of an urgent dilatation of the heart. The face in such cases becomes livid, the cardiac dulness to the right of the sternum is much enlarged, the impulse of the right ventricle forcible and struggling, and the radial artery small and compressible. There are dyspnoea and restlessness.

The routine use of blisters is not advisable, and it is doubtful whether they have real power in assisting the absorption of a pericardial effusion.

INTERNAL TREATMENT.—At the outset it is generally advisable to open the bowels with a dose of calomel (gr. i. ss.), followed by magnesium or other saline in the evening. If there is painful arthritis, salicylate of soda (gr. x. for a child of seven years) may be combined with bicarbonate of soda. It is a matter of opinion as to whether double the dose of the bicarbonate is indicated, and sometimes oil volatile or *hera vomica* is preferable. In restlessness, opium and sodium bromide are valuable; thus,

Solui brevesch	gr. x.
Nepenthe	ss.
Tinct. Iapudi	℥ss.
Aqua chloroformi	℥ss.

for a child of seven years, can be given at night. Chloralhydrate (gr. x.), if there is only restlessness, may be effectual.

When the attack is a mild one, and there are no special indications, a simple diaphoretic mixture can be given. Digitalis is well known to be disappointing in acute carditis, and, if the digestion is giving trouble, is better avoided; but it has some power in steadying the action of the heart, and is well borne by children, either as the tincture or as digalen. When there is a tendency to collapse and coldness of the extremities, strychnine is of value, and for sudden heart failure ammonia and ether mixture, or strychnine hypodermically, or intramuscular injections of camphor (gr. ss.) in sterile olive-oil.

A child of seven years can be given 5 minims of the tincture of digitalis or of digalen. Strychnine can be prescribed in a 1 in 100 solution, and 4 minims of this can be given hypodermically.

Oxygen is helpful for lividity and dyspnoea, and inhalation of nitrite of amyl may relieve an acute anginal attack or paroxysm of urgent pain and breathlessness.

Digestive disturbances require prompt treatment. Inability to take food and loss of sleep are two of the worst symptoms of acute heart disease in childhood.

It is unusual for a rheumatic pericardial effusion to require surgical interference in childhood. Should the necessity arise, and the clinical diagnosis be strongly supported by a good radiogram, it is advisable to open the pericardium by removal of a portion of the fifth costal cartilage on the left side. By this means the fibrinous exudation can be dealt with most satisfactorily.

In all cases of acute carditis a watch must be kept for the occurrence of such complications as pleurisy with effusion, delirium which should suggest the onset of chorea, or possibly the toxic effect of salicylates. Severe chorea with carditis is a very serious complication. For quieting the movements in such cases the writer advises the combination of chloral and the bromides (chloral, gr. v., for a child of seven years), followed by a suitable dose of brandy. Others prefer trional in 5-grain doses, or chlorotone (gr. iv.). In any case the compromise has to be made between an endeavour to quiet the movements and yet to prevent the cardiac circulation from being depressed by the use of sedative drugs.

TREATMENT WITH ANTI-RHEUMATIC SERUM AND VACCINES.—This is a method under trial, but unfortunately is based upon an insecure footing. None of the anti-streptococcal sera at present in use are satisfactory. "Vaccines" have achieved extraordinary popularity, but when it is borne in mind that these "vaccines" are obtained by growing the infective agents on culture media very different from the living human tissues, there seems good reason to suspend judgment upon them for some years.

The method adopted by the writer has been to give the anti-rheumatic serum during the acute attack in 10 c.c. doses—at first every other day, and then, according to the course of the disease, at longer or shorter intervals. The vaccine treatment is commenced when the acute attack has subsided, with a view to strengthen the resistance against relapses. The stock vaccine is one obtained during life from rheumatic arthritis or from the blood in acute carditis. Unfortunately, it is very difficult to obtain these cultures owing to the rapid destruction of this micro-organism in the local lesions, and it is a stock vaccine on that account which has usually to be employed. The initial dose is 1,000,000. This method is mentioned here because it must occur to all physicians that, if rheumatism is a streptococcal infection, and if vaccines have a real value in treatment, relapsing rheumatic carditis is a condition in which such a method should be of value; but the question is entirely *sub judice*, and needs prolonged and careful observation. The writer is convinced that in the present state of knowledge the greatest care is needed in using this method in acute carditis.

THE USE OF SALICYLATES AS SPECIFIC REMEDIES.—If the salicylates are used as specific remedies, the following doses have been recommended by D. R. Lees: For a child of six to ten years, 10 grains of salicylate of soda and 20 grains of bicarbonate of soda are given every two hours during the day and every four hours at night. In two days these doses are raised to 15 and 30 grains respectively, and later even to 20 and 40 grains. This treatment is persisted in until the active symptoms are overcome, and then the doses cautiously diminished. If vomiting occurs, the drug must be stopped for twenty-four hours.

If this method is followed, it must be realized that salicylate of soda has a

toxic action, quite apart from any specific effect. Ten years ago the writer pointed out that violent vomiting, great depression, a slow pulse, air-hunger, and fatal coma, might follow, and Langmead entirely confirmed these observations. The depression, in the writer's opinion, is unlike the result of the rheumatic infection, and is in great part due to an intolerable nausea. That there is an abscynsory is undoubted, but there is no guide as to when this is to be expected.

It seems to the writer certain that the delicate, high-strung rheumatic child will not endure this treatment, and he is sure that relapses may rapidly occur, even after this method has been pushed to its utmost limits, for examples of such an occurrence have come under his personal observation.

The Convalescent Stage of Acute Rheumatic Carditis.—This is probably the most important from the point of view of the physician, for he alone can realize the time that is needed for a pericardial exudation to be absorbed, myocardial damage to be repaired, and endocarditis to heal.

No definite time can be laid down, for this convalescence will depend upon the severity of the attack and the recuperative power of the patient. Three weeks after the fall of the temperature in a mild case of endocarditis is a general guide as to the period before an extra pillow is allowed. Observations on fatal general pericarditis have proved to the writer that general recent adhesion may be found three months after the acute attack, but this does not necessarily imply that the all-important myocardium has recovered its tone. Each step onward will be tested by the temperature, the pulse-rate, and the size of the heart. Fever and an excited heart prohibit any advance, and if while the child is at rest the area of cardiac dulness is diminishing, no change should be made; or if, after an endocarditis of severity, hypertrophy is shown, by the forcible impulse, to be developing, this must not be interfered with by added strain. A steady pulse and normal temperature are very encouraging, but the character of the pulse will often need to be estimated by the nurse in attendance, for the doctor himself may by his visit excite the pulse in a nervous rheumatic child. In the summer it may be possible to carry out the rest in the open air, provided that the child is kept quiet.

When progress towards recovery has commenced, the various resources at our command should be brought forward cautiously, for it is almost always time that is being struggled for. Aimless resting is bad for the child, for it means, among other drawbacks, the probability of a sudden revolt by the parents, and then a reaction from all caution. The extra pillow, sitting up, playing with toys, sitting in a chair, walking along the flat, and then up gentle inclines, can be used as proofs of gradual progress. Massage is useful, and sometimes passive movements are helpful. Six months of careful supervision is not a moment too long after an attack of acute carditis, yet this supervision can be quite well adapted to the finances of the patient. Skilled massage and passive movements are not essential, and the loyal execution of simple orders will not need repeated visits. As the realization that rheumatic heart disease is the result of bacterial poisons becomes more general, there will be more ready acceptance by the public of the necessity for time in its treatment.

Quinine, arsenic, the hypophosphites, cod-liver-oil, and some iron preparations, are all of service in convalescence, and a bracing, dry climate is indicated when the final stage is reached. When the limit of recovery has been reached, enlarged unhealthy tonsils, chronic nasal discharges, and decayed teeth, should be appropriately treated.

Prophylaxis.—The medical inspection of school-children in this country will probably do much in this direction, and there is no doubt that in the future these cases will be detected and treated earlier.

There is another precaution which the writer hopes will in the future be feasible, and that is the establishment of some convalescent homes especially designed for cases of recovering heart disease, to which children can be transferred for suitable rest, from the great hospitals which necessarily have to deal with acute disease. Ordinary convalescent homes are not suited to these cases.

Directions printed or written are useful to the parents of the children in whom rheumatism has occurred, or who inherit a tendency to its development.

The following simple directions are of value:

1. Rheumatism is a disease which often runs in families, and attention should be paid to "growing pains" and repeated sore throats, for these are often the first indications of an attack.

2. Undue nervousness, night terrors, and fitful movements, must be recognized, for they may be warnings of St. Vitus's dance or chorea, which is a sign of rheumatism.

3. Rheumatism is the chief cause of heart disease in childhood, and it may occur without pain. Breathlessness, a bad colour, wasting, and fainting attacks, should be attended to promptly, for they may point to the occurrence of heart disease.

4. Rheumatic children must be warmly clothed and their extremities protected.

5. Cold, damp rooms and neighbourhoods are particularly injurious.

6. Autumn, winter, and early spring, are the most dangerous times of year for rheumatic children.

7. Heart disease requires very patient treatment.

MALIGNANT, ULCERATIVE, OR PROGRESSIVE ENDOCARDITIS.

Malignant endocarditis is decidedly a rare event in childhood, though its rarity is not easy of explanation. It was, however, more difficult still when rheumatic endocarditis was looked upon as simple or non-infective. The usual explanation given then, and often given now, was that this form arose as the result of an infection attacking valves that had been previously damaged. If this were so, it should often occur in childhood, when valvular damage is frequent and the liability to infection at its highest. At the present time we are a step nearer the truth, for it is undoubted that rheumatic endocarditis is an infective process, and rheumatism, which is the most frequent cause of valvular disease, does not in early life tend to a local malignancy, but to widespread infection. This is at once apparent when a series of fatal cases are examined after death, for multiple acute lesions are almost invariable. The same tendency to widespread involvement of organs is also found in the acute pneumococcal and tuberculous infections in childhood. These data point to the view that infective processes in childhood show far less tendency to assume a local malignancy than in adult life.

Of sixteen cases of this form of endocarditis below the age of twelve years under the writer's observation, in seven there was a previous history of rheumatic fever, four were pneumococcal, two were staphylococcal, and three of doubtful origin.

Pathology.—The malignancy of an endocarditis does not depend upon the size of the vegetations, although some of the events in the illness—*e.g.*, embolism—

are favoured by their magnitude. The most malignant forms are those in which the vegetations are comparatively small, but the bacteria in them exceedingly numerous and virulent; for then they are not destroyed by natural processes, but, multiplying in the lesions, are scattered by the blood-stream in every direction. The more chronic forms, such as are some of the rheumatic, show large partially-healed vegetations in which many of the micrococci are destroyed. These vegetations may spread far from the valves on to the surfaces of the auricles or ventricles, and by ulceration produce rupture of the valves, or even of the cardiac muscle.

This form of heart disease is always infective, and may result from any agent capable of attacking the cardiac valves, and may also, in all probability, be the result of a mixed infection.

The Rheumatic Form of Malignant Endocarditis.—The evidence brought forward to support the existence of this form, as published by Paine and the writer



FIG. 44.—A SECTION THROUGH A VEGETATION FROM A CASE OF MALIGNANT ENDOCARDITIS IN A CHILD THE SUBJECT OF REPEATED RHEUMATIC ATTACKS. (POLYPUS AND PAINE.)

The necrotic tissue shows a dark fringe, which under higher magnification was seen to consist of masses of strepto-diplococci. Contrast the vegetation is "simple" rheumatic endocarditis, Fig. 39.

in 1901, is clinical, pathological, bacteriological, and experimental. Stated briefly in tabular form, it is to this effect:

1. This form of endocarditis may occur in rheumatic children, associated with signs of active rheumatism, indistinguishable, except in so far as the endocarditis is concerned, from their previous attacks of rheumatism.
2. The post-mortem lesions, except for the severity of the endocarditis, are indistinguishable from those in severe simple acute rheumatism.
3. A strepto-diplococcus, indistinguishable from that obtained from simple rheumatism, can be isolated from the valves.
4. This micrococcus reproduces in animals on intravenous injection malignant endocarditis, and also acute rheumatism.
5. The micrococcus obtained from simple acute rheumatism, on the other hand, produces acute rheumatism, and also malignant endocarditis, in animals.

There is one point in the pathology of malignant endocarditis upon which it seems too little stress has been laid, and that is, the frequency with which small

areas of necrotic tissue remain in simple vegetations that are believed to be healed. It is these areas that are of such vital importance in any theory as to the origin of malignant endocarditis, for it is most probable that they contain within them bacteria that are capable of attaining fresh virulence. If such is the case, it may well be that the malignant endocarditis may sometimes be due to them, and not to a fresh infection from without.

A short clinical history of one of the cases from which we verified our conclusions will illustrate the rheumatic group of cases. A boy aged eleven had suffered from rheumatic fever when three and a half years old; his mother had also suffered from the same disease. Five weeks previous to his coming under observation there had been multiple arthritis, pains in the chest and abdomen, and pro-



FIG. 65.—MALIGNANT ENDOCARDITIS OF THE AORTIC VALVE, SHOWING A LARGE FUNGATING VEGETATION.

(From the Museum of University College Hospital.)

gressive anæmia, with fever. There was severe mitral disease, and both liver and spleen were enlarged. Later, blood and albumin appeared in the urine, and splenic infarction followed. Purpura, vomiting, progressive anæmia, wasting, irregular fever, and sweating, with an intercurrent attack of pericarditis, were other persistent features. The necropsy showed recent sero-fibrinous pericarditis, extensive malignant endocarditis of the mitral valve with ulceration, and numerous infarcts in spleen and kidneys. The diplococci was isolated in pure culture.

It is the rheumatic cases that give the most characteristic examples of malignant endocarditis, with all its symptoms well developed.

The pneumococcal cases may be characteristic, but more often are so acute that the symptoms of the endocarditis are lost among more obstructive signs of empyema, pneumonia, or suppurative meningitis.

The pyæmic foci resulting from such lesions as middle-ear disease, jugular thrombosis, or septic periostitis, are as a rule in no way characteristic, and merge into the general septicæmia.

There remain some mysterious cases of uncertain origin which may produce symptoms that dominate the clinical course of the disease. Some of these are cases of great severity, and, according to the particular symptoms that predominate, have been called "typhoidal" or "meningeal."

The following is an example:

A girl aged nineteen months had been feverish for three weeks, and moaning with pains in her limbs. No cause for this was found, and the family history was of no assistance. The infant was slightly yellow in colour, the abdomen distended, and lower limbs flexed. The skin over the left knee-joint was shiny in appearance, the temperature 102.8° F. Pulse 160 to the minute, and respirations 80.

Later the temperature rose to 106° F., and then remained at about 104° F. The heart was extremely rapid. Osteomyelitis, pneumonia, and general tuberculosis, were all suspected in turn, and seven days later three purpuric patches developed upon the left cheek, dorsum of the left foot, and end of the nose. A loud systolic murmur also developed, of maximum intensity over the impulse, and there were the faintest signs of pericardial friction.

Death occurred on the ninth day, and the necropsy showed very early pericarditis and ulcerative endocarditis of the mitral valve. There was an infarct in each lung, and subpleural and subpericardial petechiæ. There was also enlargement of the spleen and intense fatty degeneration of the kidneys. No signs of pneumonia, typhoid, tubercle, or osteomyelitis, were discovered. A culture from the pericardial fluid gave a pure growth of strepto-diplococci, and films from the vegetations showed their presence in enormous numbers. The micrococci produced multiple non-suppurative arthritis, but, curiously enough, no endocarditis.

The leading symptoms that point to the malignant nature of an endocarditis may be divided into (1) constitutional and (2) local. Additional evidence is also sometimes obtained by (3) bacterial investigation of the blood.

1. The constitutional symptoms are—

- (1) Irregular fever.
- (2) Rigors (rare).
- (3) Sweating and wasting.
- (4) Vomiting and diarrhoea.
- (5) Progressive anæmia.
- (6) Delirium.

2. The chief local symptoms are—

- (1) A persistently excited action of the heart.
- (2) Variability in the character of the cardiac murmurs.
- (3) Evidences of induration, or great enlargement of the spleen without induration.
- (4) Purpura, and sometimes profuse epistaxis.
- (5) *Nodosités cutanées éphémères*. These are minute spots, slightly tender, raised, and red, with a white point in their centres. They last from some hours to a day or two, and are situated generally near the tips of the fingers.

3. The isolation of micro-organisms from the blood-stream is very important evidence of the condition, but the failure to obtain them is of little value, and their presence is not conclusive, for the diplococcus can be isolated from the blood-stream in a severe rheumatic carditis which shows no evidence of malignant valvular disease.

As a rule malignant endocarditis is a fatal disease, and no remedies are as yet proven to have any control over its course.

It is the most frequent cause of severe cerebral hæmorrhage in childhood. In such cases an embolus infects a vessel, such, for example, as the middle cerebral, and this, giving way under the blood-pressure, may produce rapid death or a severe hemiplegia. A cerebral abscess occasionally follows the lodgment of septic emboli in the brain, and aneurysms of the arteries of the extremities may also result from embolic processes.

The course of the illness varies much—from a matter of days on the one hand, to months on the other. In some cases, after a long illness in which there has been infatuation, wasting, anæmia, and active heart disease, the condition may quiet down and the patient recover with a damaged heart. Such recoveries have been attributed, on quite insufficient evidence at present, to the use of sera or vaccines; for it is well known that such cases occurred occasionally before such methods were known.

TREATMENT.—This is very unsatisfactory. At the present time, if the condition is proved to be due to a streptococcal or other known infection, anti-sera are administered or vaccines given. With both methods recoveries have been recorded, but the general rule is complete failure. Apart from these special methods, the management of such cases is conducted on general lines, and then occasionally an unexpected recovery will occur.

The writer would emphasize the great importance of the possibility of preventing some cases of the rheumatic type by a more patient endeavour to produce a cure of simple endocarditis. There is a certain amount of evidence to show that the existence of an imperfectly-treated endocarditis may lead to the graver illness, particularly should there be anæmia or unhealthy surroundings.

HEART DISEASE DUE TO OTHER INFECTIONS.

Scarlet Fever.—Heart disease not infrequently dates from scarlet fever, and some forty examples have been under the writer's observation. The cause of these heart affections is disputed, and there is evidence to show that there may be more than one explanation of their occurrence.

We may put aside examples of heart affections the result of nephritis of scarlatinal origin, for these are considered later, and confine to this section the cases of carditis directly associated with the disease.

The problem that first presents itself is this: Some patients develop affections of the heart during scarlet fever, and others—the majority—after the early stages of the disease are over. Are we to interpret the cardiac lesions as the direct result of the exanthem, or are any of them of this nature?

There can be no doubt that scarlet fever has a remarkable effect in producing rapidity of the heart's action, and in a considerable number of cases this rapid action is a feature which may persist over a considerable period.

When, however, we turn to the cases of pericarditis and endocarditis, we find that there are other probable causes, and an analysis of twelve fatal cases illustrates this point.

1. A female, aged eight years, with mild scarlet fever, developed pleurisy eighteen days later, and died on the twentieth day, when pericarditis and pneumo-pneumonia were found.

2. A male, aged five years, developed pericarditis in the first week, and died nine days later; there was no valvular disease.

3. A male, aged four years, died in the tenth week. Suppurative pericarditis, empyema, and nephritis, were found.

4. A female, aged four years, fourteen days after the rash appeared, developed a valvular murmur and died. The post-mortem examination showed malignant aortic endocarditis and an empyema.

5. A female, aged four, died on the ninth day after the rash, of pneumonia and early pericarditis.

6. A male, aged fifteen months, died on the thirteenth day, of empyema and suppurative pericarditis.

7. A male, aged two years, died on the fourteenth day, of pleurisy and pericarditis.

8. A female, aged six years, died on the nineteenth day, of nephritis with early mitral endocarditis.

9. A female, aged nine years, developed chorea, and then scarlet fever, followed in three days by arthritis. Death ensued from diphtheria. The post-mortem examination showed mitral endocarditis.

10. A male, aged two years, two months after scarlet fever, developed an empyema and suppurative pericarditis.

11. A male, aged four and a half years, three weeks after scarlet fever, developed renal disease and pleuro-pneumonia, with mitral beading.

12. A female, aged six years, died in the fourteenth week, of pericarditis.

Are we to call these cases of scarlatinal heart disease? The answer is clearly impossible until we understand the real nature of this infectious disease. The writer inclines to the view that these lesions are of various origin, the result of secondary infections invading a patient who is suffering from the result of the primary disorder—scarlet fever.

This view is supported by a clinical study of cases of heart disease dating from an attack of scarlet fever which are not fatal. The first point that is evident in the history of such cases is that they are usually associated with signs of "scarlatinal rheumatism." Thus, in twenty-five examples of scarlatinal rheumatism, sixteen showed cardiac affections.

Again, these cases of heart disease as a rule show no peculiarity by which they can be distinguished from the rheumatic, and, further, may undergo relapses, with other manifestations, long after the occurrence of the scarlet fever. Chorea, repeated arthritis, pains, and nodules, may each of them occur over a period of years. In one case an organism indistinguishable from the diplococcus of rheumatism, both in culture and from experiment, was isolated from a fatal pericarditis of this kind.

Another very important consideration in this problem is the part taken by the streptococcus described by Mervyn Gordon in severe scarlet fever, and the relation that the micrococcus bears to the rheumatic organism.

In conclusion, it would appear to the writer that heart disease may result in scarlet fever from Gordon's streptococcus, from the diplococcus of rheumatism,

from the pneumococci, or from other micro-organisms of the septic type; further, that the probable channel of infection is in most cases the secondary sore throat; and, lastly, that the scarlet fever virus may have a direct effect in producing tachycardia, and an indirect effect in lowering the resistance of the constitution to secondary infection.

PATHOLOGY.—There will be no necessity to recapitulate the details of the pericardial and endocardial lesions of the rheumatic type. The suppurative forms of pericarditis are dealt with under the head of pro-pericarditis and the malignant forms of endocarditis in their corresponding section. There remain some cases of sudden death in scarlet fever in which it would appear that the neuro-vascular apparatus has been particularly damaged. Gorget and Decharux have described such cases without any lesion of the myocardium. Weill and Mouriquand, on the other hand, have recorded a case, fatal on the fourteenth day, in which there was severe myocarditis. The cardiac muscle showed much fatty degeneration and a segmentation of the fibres, and the fibrous tissue in the region of the minute bloodvessels was infiltrated with leucocytes, mostly mononuclear.

SYMPTOMATOLOGY.—The myocardial cases of the severe type show all the signs of cardiac asthete. There is a rapid and even running pulse, which may be irregular with coupled beats or interrupted by intervals of retardation. There is moderate dilatation, with feeble, irregular heart sounds. There is muscular prostration, with dyspnoea and lividity, and death may result from syncope and convulsions. Popischill attaches considerable importance to a condition of the heart that is sometimes met with in the early days of scarlet fever. In some of these cases there develops a blowing, apical systolic murmur, with an accentuated pulmonary second sound and irregular action; in others a loud, grating systolic murmur becomes evident, with its maximum to the left of the sternum, at the level of the third costal cartilage.

The symptoms of pericarditis and valvular disease are given under the rheumatic and suppurative infections. It remains here to emphasize the methods of development of these lesions. They may be placed in three classes:

1. Acute, rapidly fatal cases of pericarditis associated with the acute scarlet fever.
2. Cases developing at or after the appearance of such a lesion as pneumonia, or pleurisy and empyema.
3. Cases developing in the stage of convalescence. These are usually rheumatic, and the initial symptom may be a multiple arthritis or a chorea—this latter is frequent; or, again, it may be a primary carditis.

It cannot be too much emphasized that these cases of *antrix cordis*, far from being in any way of a milder type than those of ordinary acute rheumatism, are identical in symptoms and prognosis.

The *Diagnosis* of these forms of heart disease is considered under Rheumatism, Malignant Endocarditis, and Pyo-pericardium. The *Prognosis* falls under similar headings, as also does the *Treatment*.

REFERENCES.

The following references to recent observations upon heart disease in scarlet fever may be consulted:

- NEUBOGER: *La Clinique*, March 25, 1909.
POPISCHILL AND WEISS: *Zeitschr. f. ärztl. Fortbildung*, 1911, p. 68.
WEILL AND MOURIQUAND: *Presse Médicale*, 1911, xix, 11.

Pneumococcal Infection—Pyo-pericardium.—This is an important group, because it includes the great majority of cases of pyo-pericardium, a condition of great gravity and of extreme difficulty of diagnosis. For this reason it is a convenient place in which to detail the signs of pericardial effusion in childhood.

In common with other infections, the pneumococcus may damage all parts of the heart, but suppurative pericarditis is the outstanding lesion. Endocarditis, when it occurs, tends to be malignant in type, although by some a simple endocarditis is considered to be more frequent than is generally believed to be the case. An analysis of 100 fatal cases of pneumonia does not, however, support the latter view, for in none of these was there any sign of endocarditis; and in 100 fatal cases of pyo-pericardium, the vast majority of them apparently of pneumococcal origin, only one instance of endocarditis was found. It is the writer's experience that pneumococcal malignant endocarditis, just as the rheumatic malignant form, is more frequent in adult life.

Myocarditis would also seem to be rare, and the slow pulse that is sometimes so striking a feature after the crisis of a pneumonia cannot be looked upon as an evidence of a gross myocardial affection, seeing that it is so transitory and recovery so complete.

In severe cases of pyo-pericardium there is some fatty change in the muscular fibres, with perivascular cell exudation, and in some cases of malignant endocarditis there may be abscesses in the cardiac wall, but these occurrences are exceptional.

EMPHYSEMA.—It will be convenient here to deal with the whole question of pyo-pericardium, which, though as a rule pneumococcal, may also result from infection with other pus-forming organisms.

An analysis of 100 fatal cases brings out some points of much practical interest. In this list the chief associated conditions were as follows:

1. Pneumococcal infections, of which 55 per cent. were associated with empyema on one or both sides, 31 per cent. with acute pneumonia and pleurisy. In 1 per cent. pericarditis was the outstanding lesion. This form is most frequent under four years.
2. Osteomyelitis, usually staphylococcal in origin. This is more frequent in children over four years of age.
3. Middle-ear disease and septic broncho-pneumonia associated with mixed infections.
4. Suppurating wounds.
5. Acute streptococcal infection of unknown origin.
6. Virulent scarlet fever, usually associated with empyema.
7. Tuberculosis complicated by secondary infections.
8. Measles and hooping-cough complicated with pneumonia.
9. In rare cases the infection may arise from a subphrenic abscess.

The ages at which pyo-pericardium is most frequent are remarkable: 84 per cent. occurred under four years, 66 per cent. between one and three years, and it is apparent that the pneumococcal form is mainly a disease of infancy and very early childhood.

PATHOLOGICAL ANATOMY.—The pathological processes vary with the virulence of the infection. In some cases there are only a few flakes of fibrino-plastic exudation; in others there is intense pericardial infection and a blood-stained exudation; in others a large purulent collection distending the sac; and in some rare instances

great thickening of the pericardium, with partial organization of the inflammatory exudation. The pneumococcus can be isolated from the purulent effusions with ease.

How does the pericarditis arise? It has often been stated that it does so by direct extension from an empyema, and the general impression is left that this process of direct extension is the rule. The truth of this is not, however, established, and it is far more likely, from clinical and pathological investigation, that the pericarditis is the result of an independent infection. It certainly arises when an empyema is present yet situated far from the pericardium, and in very severe cases there may be a pericarditis and pleurisy which, from the dates of the illness, must have clearly originated asynchronously. In the writer's opinion, it is in accord



FIG. 46.—*LOBAR PNEUMONIA OF THE UPPER LOBE OF THE LEFT LUNG, WITH SUPPURATIVE PERICARDITIS.*

A, Red hepatization of upper lobe of left lung; *B*, suppurative pericarditis and heart compressed by exudation; *C*, distended pericardial cavity; *D*, collapsed area of left upper lobe; *E*, healthy lung of left lower lobe; *F*, visceral pleura.

(From the Museum of University College Hospital.)

with experience of cardiac infections that pyo-pericardium arises as an independent result of an infected coronary circulation much more frequently than as a direct extension from an adjacent focus.

The figure above illustrates the point at issue. In the case from which the specimen was taken there was acute pneumonia of the left upper lobe, and pyo-pericardium, but there was no empyema. The interest lay in the fact that the pleura bounding the right border of the left upper lobe, in spite of the pneumonia, was not inflamed; there were no flakes of exudation upon its cardiac surface, no reddening, and not even thickening of this membrane. Yet there was severe pericarditis.

Such a specimen cannot prove, but it strongly supports, the view that there were two local infections, dependent not upon contact, but upon the invasion from the general blood-stream.

The difference of opinion upon this point is valuable, because it enables one to emphasize a fact which it is essential to realize: that, owing to the extreme difficulty of the diagnosis of pyo-pericardium, it becomes often impossible to date the moment at which the pericarditis arises, and so the life-history of the condition is as yet but imperfectly understood. There are few who would like to express an opinion upon the length of life that is compatible with the existence of a pyo-pericardium, and when reference is made to records to elucidate this point, we are generally driven to infer that duration, not from some definite event such as pericardial friction or heart pain, but from the length of the illness with which it is associated, and with the lesions that are found in the post-mortem room.

An analysis of these illnesses in the 106 cases showed that in 20 the duration was about four weeks; in 50 from four weeks to six months; in 17 from six months to over a year. Of the remainder, some were not pneumococcal, and of the others there were insufficient data.

If we add that in some cases great pericardial thickening and considerable though imperfect organization were present, it is reasonable to believe that suppurative pericarditis may exist for some considerable time before it causes death.

SYMPTOMATOLOGY.—The commencement of the illness may be an attack of measles or whooping-cough complicated by broncho-pneumonia, or an acute lobar pneumonia or pleuro-pneumonia. In the more chronic cases pleural pain and pleurisy, cough and wasting, are early symptoms, and vomiting and diarrhoea are also met with. In the insidious forms gradual wasting and failure of health may be the only signs.

The detection of the pericarditis is most difficult in young children, and not always easy in the older ones, and for this there are two particular reasons:

1. The frequent absence of any pericardial friction.
2. The frequent presence of serious disease of the respiratory organs, which not only distracts attention from the heart, but also complicates the already obscure indications of the pericardial inflammation.

It is probable that some soft transient pericardial friction is more frequently present than clinical records would seem to show, and emphasis must be laid upon the importance of examining the patient, not only in the recumbent position, but also sitting up and leaning forward, for by this procedure any friction at the extreme base may be brought into evidence. The most probable explanation of the ill-marked friction in these cases is the greater digestive action of the poisons formed by the suppurative processes, which thus produce a rapid softening of the exudation as compared with the lesser peptic action of the rheumatic processes.

If there is only a limited effusion without friction, the diagnosis is practically impossible, although a knowledge of the possibility of pyo-pericardium, coupled with evidence of continued but unexplained fever and general illness, with an excited and rapid action of the heart, may arouse suspicion.

Pericardial Effusion.—When the effusion is considerable, the most valuable indication is a gradual muffling of the heart sounds coincident with an increase in the area of precordial dulness. Another sign of value is the discovery that the

cardiac impulse does not coincide with the left lower border of the cardiac dulness. When the effusion is large, the pulse may be notably rapid, small in volume, and easily compressible, yet, on the other hand, it may be unusually slow. There may also be fulness of the jugular veins and distressing dyspnea on movement. Sometimes, but by no means always, orthopnea is present; or the child leans forward, or even assumes a kneeling position. There may be turgescence of the face and some edema of the lower extremities. In rare cases there is difficulty in swallowing. A rapid upward extension of the cardiac dulness to the left of the sternum is another valuable sign, and the upper lobe of the left lung in front may then be so compressed that impairment of the note extends to the left clavicle.

Bulging of the precardial intercostal spaces and an undulatory wave transmitted by the heart may be present, and the note over the precordia be intensely dull.



FIG. 47.—SUPPURATIVE PERICARDITIS, WITH EXTENSIVE EFFUSION. (HARSHBARGER.)

The darker shadow of the heart is visible within the effusion.

The outline of the cardiac dulness is pear-shaped, with the stalk upward, and there is loss of resonance in the fifth right intercostal space. Posteriorly there may be tubular breathing in the subscapular region about the middle of the lower lobe of the left lung.

The X-ray picture may show decisively the presence of an effusion, as illustrated in Fig. 47.

Extraordinary though it may seem, in spite of these various indications we are not infrequently humiliated by the discovery of considerable yet unsuspected pericardial effusions after death.

When, as sometimes happens, the effusion is retro-pericardial, all

these physical signs may be very ill-marked and the X-ray picture inconclusive, as has been the experience of the writer. Extreme dilatation may also be most difficult to distinguish, even by radiography.

As regards the SYMPTOMS, some importance must be attached to the occurrence, in young children suffering from empyema or pneumonia, of sudden unexplained rises of temperature, great pallor, extreme illness, paroxysmal attacks of dyspnea, syncopeal attacks, and occasional convulsions. Death is often sudden after some slight exertion or after taking food.

Death may also result from suppurative meningitis (15 per cent.) or the illness may be complicated by suppurative peritonitis (10 per cent.).

DIAGNOSIS.—The differential diagnosis of pyo-pericardium is most difficult in the pneumococcal cases, because it is pre-eminently in these that we meet with associated lesions of the respiratory system, such as empyema, pneumonia, and mediastinitis.

Apart from entirely overlooking the lesion, tuberculosis is probably the most frequent diagnosis given in these cases, on account of the fever, respiratory signs, and wasting. Reliance must be placed on careful physical examination of the heart, including radiography, coupled with examination of the blood and ven

Pirquet's cutaneous test for tuberculosis. An effusion into the pleura on the left side may be extremely difficult to distinguish, particularly if this comes across in front of the heart or if the two lesions are associated. The differentiation rests upon minute physical examination of the precordia and left chest, coupled with X-ray examination and a careful weighing of the evidence thus obtained with the history and symptoms of the illness.

Dilatation of the heart may be mistaken for pericardial effusion, and, vice versa; the differentiation of dilatation of the heart and pericardial effusion is seldom very difficult in childhood, but in the exceptional cases it puzzles the most experienced. As a rule the history is a guide, and against the signs just given as evidence of effusion we can place the clearness of the heart sounds, the absence of any striking upward extension of dullness, the presence of a definite if weak impulse, and absence of massive cardiac dullness and intercostal bulging. Radiography will also give assistance.

The difficult cases are those in which there is great dilatation of the heart and also much thickening of the pericardium from old and recent inflammation. We must bear in mind that well-marked friction, though much rarer often as evidence of a slight effusion, is also compatible with a large one; and, on the other hand, that the cardiac dullness may extend upwards both to the right and left of the sternum quite as high in these complicated cases of cardiac dilatation as in a case of pericardial effusion.

Muffled heart sounds are also met with when the pericardium is much thickened. Much importance then attaches to the history. Repeated attacks of rheumatism point to dilatation and an adherent thickened pericardium. A pneumococcal infection, on the other hand, will suggest a large effusion, as also will clear evidence of tuberculosis or lympho-sarcoma. Radiography will assist to some extent in these cases if the apparatus is an exceedingly good one.

The Prognosis is very grave, and it is doubtful whether recovery can occur without surgical interference. Even if the pericardium is opened, the extreme illness of the patient and the associated lesions frequently preclude any good result. In so far as very young and cachectic children are concerned, it seems unlikely that, apart from some potent serum, any method of treatment could be successful. When the child is over six years of age the outlook after operation is decidedly more hopeful.

Cyril Ogle and Allingham's incision would appear to be the most satisfactory for dealing with considerable effusion. In this operation an incision is made in the epigastrium at the lower level of the left seventh rib cartilage, and the pericardium is reached by working through the costal attachment of the diaphragm.

Diphtheria.—No infective disease is so liable to produce sudden and fatal heart failure as diphtheria. In some cases the arrest of such a tragedy would seem to be entirely beyond our control, but it is undoubtedly that when the utmost care and caution are taken in supervising the convalescence much may be done to prevent such a calamity.

The clinical warnings of heart failure in this disease are unobtrusive and easily overlooked by those who are cautious over the management of convalescents; on the other hand, though unobtrusive, they are almost invariably capable of detection, and unexpected death becomes progressively rarer with increasing vigilance. In a hospital ward devoted to the disease, with every possible advantage

at hand, cases of diphtheria admitted early in the illness rarely succumb from heart failure, but death may not be prevented when they come some weeks after the onset, with signs of progressive and severe paralysis.

PATHOLOGICAL CHANGES.—In many cases there is myocardial damage from the direct action of the extracellular toxins upon the cardiac muscle. In others, as C. Bolton, J. J. Thomas, Vincent, and Bocaz, have shown, there are degenerative changes in the *medulla oblongata*, with no obvious cardiac lesion. Such a condition as this has also been produced experimentally by Le Ferré and Verger. In yet other cases advanced fatty degeneration has been found in the fibres of the *parasympathetic* nerve.

The damage to the heart wall is twofold—parenchymatous and interstitial. In the fibres the first change is a fatty one, which may progress to a complete destruction of the sarcomic elements. A section of such tissue will show in some places fatty degeneration; in others, swollen and varicose fibres; in others, again, hyaline masses and complete destruction. The interstitial changes which spread from the regions of the bloodvessels take the form of minute hemorrhages and focal deposits of leucocytes. Later the damaged muscular fibres may be replaced by connective-tissue proliferation and a varying degree of interstitial myocarditis. A lesion of the *curculo-ventricular bundle*, with consequent heart-block, has been demonstrated by Fleming, Kennedy, and others in cases of bradycardia due to this disease.

There is also sometimes considerable dilatation of the heart, and with this ante-mortem thrombosis in the cardiac chambers; and Klumoff has demonstrated degenerative changes in the intra-cardiac ganglia, which have also been experimentally produced in animals by Née.

A recognition of the various ways in which diphtheria damages the heart is of practical importance. It is apparent that the acute bulbar lesions may produce syncope with alarming rapidity and but little warning. On the other hand, the subacute myocardial lesions give clear indications, and the after-effects may be of considerable duration. Pericarditis and endocarditis are rare, and probably result from secondary infections.

Emphasis must be laid upon the fact that the final event which frequently turns the scale against the feeble heart is paralysis of the diaphragm.

An *Analysis of Fifty Fatal Cases of diphtheritic paralysis* will serve to illustrate some valuable points; 30 were males and 20 females. The condition is most dangerous between the second and fifth years, and it is noteworthy that in 11 cases there was absence of any convincing evidence of the initial diphtheria. This clearly introduces the danger, which occurred in all these cases, that the nature of the illness may be overlooked until the onset of the paralysis.

The onset of diphtheritic paralysis, and with it the possibility of cardiac involvement, commenced at any period from the first week, when the membrane was still present, to the eighth week after the commencement of the *sore throat*. In 39 cases in which the onset of the diphtheria was accurately ascertained, in 28 the symptoms of paralysis commenced between the second and fifth weeks.

In no less than 30 of the 50 cases the terminal event was supervention of paralysis of the diaphragm, and in 13 of these the clinical notes expressly stated the satisfactory state of the heart some days before this occurrence.

In 37 cases there was clinical evidence of damage to the heart, and the most

important signs were rapidity and irregularity of the pulse, shortness of the first sound, and dilatation. In some cases dilatation was considerable, but cardiac murmurs were exceptional. In no instance was there pericarditis, and in only one some thickening of the mitral valve was described. Anteriorly thrombosis was mentioned in some instances, but asked eye signs of disease of the heart wall were conspicuous by their absence.

SYMPTOMATOLOGY.—The general condition when there is cardiac weakness is one of listlessness and muscular prostration. Fainting attacks may occur. The pulse is often a valuable guide, and undue rapidity and irregularity with a small wave and low tension are the most frequent indications of danger, particularly if at the same time there is a normal or subnormal temperature. A slow pulse is not so frequent, but may, as Fleming and Kennedy have shown, be dependent upon a true heart-block.

A somewhat different but very serious group of cases are those in which there is much slowing of the pulse, with vomiting and great prostration, and sometimes syncopal or epileptiform attacks. These cases may show well-marked signs of diphtherial paralysis.

Damage to the myocardium is evidenced by the feeble impulse, irregular action, dilatation, which is usually of moderate extent, and the short first sound and loud pulmonary second. A short systolic mitral murmur may or may not be present.

The dates of the appearance of the cardiac affection vary considerably, but in general terms they may be grouped under two headings—those in which the symptoms appear during the acute attack or just as it is subsiding, and those in which they appear about the fourth to eighth week, as a rule synchronously with the appearance of signs of diphtheritic paralysis.

A fatal case in the first group runs a course of the following kind. From the onset the child is seen to be severely poisoned, and, when the membrane has cleared away, lies prostrate, pale, and listless. Then vomiting—always an ill-omened occurrence—commences, the pulse becomes rapid, feeble, and irregular, and death occurs from a sudden syncope.

A fatal case in the second group shows a different clinical course. The active disease is now over, and the child may seem practically convalescent. Then the knee-jerks disappear and the limbs become weak, ocular and palatal palsies follow, and the heart is found to be weak and possibly dilated. Difficulty in swallowing and paralysis of the diaphragm supervene, and death is usually the result of the combined effect of massive collapse of the lungs and cardiac astyole.

When in the course of the illness cerebral or pulmonary embolism occurs, a thrombus forming in one of the chambers of the heart should be suspected. Auché has recorded emboli in the arteries of the limbs, and Eschrich renal emboli from the same cause.

In milder cases the cardiac feebleness may be quite transitory, but there still remains a group in which irregularity of the pulse and feebleness of the heart, with a tendency to syncope, may persist for many months. Such cases are much aggravated by any undue strain resulting from incautious over-exertion. It would seem probable that these chronic cases are the result of a definite myocarditis.

DIAGNOSIS.—The chief danger is that of overlooking the early signs of myocardial weakness, and when once suspicion is aroused the question usually resolves itself into one of determining the cause of a moderate degree of dilatation of the

heart. Among these causes, influenza, rheumatism, and some cases of primary renal disease, are the most difficult to differentiate. The history must be investigated, bacteriological examination of the throat undertaken, and other evidences of diphtheritic paradyaia looked for.

PROGNOSIS.—If the case is early under observation, and the attack mild, the outlook is good. Clear evidence of damage to the heart in the first ten days is serious. Pharyngeal and respiratory paralysis, severe vomiting, great pallor, restlessness, and syncopeal attacks, are all bad signs.

A rapid, ill-sustained, irregular pulse or a markedly slow pulse is grave, and close approximation of the two sounds of the heart is a sign of great danger.

The usual course is for the heart to make a gradual but complete recovery, but when chronic myocarditis follows, irregularity of action, breathlessness, and some degree of dilatation, may remain for months, and in some instances appear to leave a permanent weakness. Over-exertion when the heart is weakened by the diphtheritic poison may cause sudden death, or delay complete recovery for twelve months or more.

TREATMENT.—Good nursing is a valuable aid in severe cases, for a thoroughly trained nurse accustomed to these cases realizes the great importance of absolute rest in the recumbent position. In addition, when there is widespread paralysis, skilful feeding by the nasal or oesophageal tube will be needed. In such cases a warning must be given against too rapid feeding or the giving of too large quantities. Antitoxin will be injected when the case is seen in the early stage, and it is hard to believe that an agent such as this, which hastens the process of recovery, does not also diminish the time during which the manufacture of the poisons that produce paralysis is proceeding. L. Dudgeon has, in fact, put forward some experimental evidence in favor of the view that antitoxin given early has a preventive effect in antagonizing the myocardial poisons.

As regards the length of rest, each case must be decided upon its own merits, but we have as a guide the knowledge that the period at which signs of cardiac weakness show themselves most frequently is in the fourth week. And we also know that there is no necessary relation between the severity of the initial attack and the subsequent paralysis. Consequently it is certain that all cases should be kept at rest for at least three weeks after the attack of diphtheria is over, and that after this, again, each step forward should be gradual and watchful.

When the heart is affected, if convalescence has commenced, no delay should be made in a return to absolute rest. Among the drugs most used are strychnine and atropine, and to these may be added camphor, and possibly adrenalin given hypodermically. Lees advocates the combination of atropine and strychnine, and pushing the atropine until there are early signs of intoxication. Adrenalin may be given hypodermically in 1 in 1,000 solution in doses of 3 to 5 minims repeated every four hours. Camphor may be dissolved in olive-oil (in the proportion of 1 part to 9 parts of olive-oil), and injected into the muscles in doses of 1 to 2 grains. On the Continent camphor in 1-grain doses is combined with 17 minims of ether, and put up in sterile capsules for intramuscular injection when there is sudden heart failure. Stimulants are indicated, and oxygen also when there is lividity. For mild cases or for those recovering, the combination of iron and quinine is a useful tonic.

No violent exertion should be allowed for six months after an attack of diphtheria

in which there has been a suspicion of cardiac damage, for it is impossible to ascertain with accuracy the degree of that damage, and it is certain that premature indulgence in violent exercise will produce an intractable cardiac dilatation and irregularity.

Tuberculosis.—It would appear from Continental literature that in this country, where rheumatism is so rife, there may be some danger of underestimating the frequency of cardiac tuberculosis, and there is no doubt that this infection may attack all parts of the heart, and sometimes also the larger bloodvessels. For the clinician the pericardial lesions are of most importance, and on this account they will be considered first.

PATHOLOGY.—The pathological processes are strictly comparable to those that occur in tuberculosis of other organs—that is, there may be acute or chronic lesions, miliary deposits, or caseating areas, and in some instances much development of fibrous tissue.

An analysis of 30 fatal cases in which there was tuberculosis of the heart shows that in the majority of instances these lesions are of no clinical importance, but are merely incidents of a general infection. Four of these, however, showed that a chronic and progressive multiple serositis may result from this infection. Of these 30 cases, 19 were males and 11 females. The ages ranged from four months to ten years. Tuberculous infection was noted in the pericardium on 22 occasions; in the mitral valve on 4; and in the myocardium on 9. The pericarditis consisted of a few miliary deposits with inflammation around the base of the large vessels in 15 instances; in 4 there was a multiple serositis with general pericardial adhesion; in 3 an acute general pericarditis with considerable thickening of the pericardium. In one case the endocarditis was vegetative in type; in another there were calcareous deposits in the valve rings. The myocardial damage consisted as a rule of miliary deposits, but there were four examples in which there were caseous areas reaching the size of a split pea in one or other ventricular wall.

The majority of these cases proved fatal from acute and general tuberculosis. Even the distinctive examples of multiple serositis died eventually of a miliary outburst in the cerebral meninges.

Tuberculous Pericarditis.—The lesions may be acute or chronic, general or localized. In the latter case the deposits are usually at the base of the heart.

The acute lesions may be associated with the exudation of a large quantity of fluid into the pericardium, which may be clear or blood-stained, and possibly arise from a primary infection through the coronary bloodvessels, though there may also be a direct extension of the tuberculous process from pulmonary lesions or caseating bronchial glands. In these acute cases there is a rise of temperature, with the symptoms of pericarditis, and possibly some pericardial friction. Later all the signs of a large pericardial effusion develop, which may seed parametosis, and may recur after withdrawal of the fluid. Purulent pericardial effusions may also occur in the tuberculous, but in such cases it is most probable that there has been a secondary infection by micrococci complicating the tuberculosis.

Tuberculous pericarditis may also be plastic in character, and then tends to become chronic and to relapse. This dry form gives rise to pericardial friction, and the condition is very likely to be looked upon at first as rheumatic in nature. When it is relapsing in type, much fibrosis results, and the clinical course that is

seen then is described below under Multiple Sarcoid. The symptoms are not obstructive, but there may be some fever and precordial pain, with dyspnoea and a rapid pulse. Dilatation of the heart is not a striking feature.

The **DIAGNOSIS** is difficult, and although the history, and possibly the use of one of the tuberculin tests—such as, for example, von Pirquet's—may lead to a very probable surmise as to the cause of the effusion, the actual demonstration will probably require experimental inoculation of a guinea-pig with some of the fluid. Cultures alone have repeatedly proved sterile. Absence of valvular disease and the presence of other tuberculous lesions favour the diagnosis of tuberculosis.

The **PROGNOSIS** is uncertain, and certainly grave. Although in some cases the inflammation quiets down and the heart recovers, more often the extensive nature of the tuberculous lesions in the other viscera and the acute nature of the infection preclude recovery.

Tuberculous Endocarditis.—Tuberculous endocarditis may be divided into an acute and chronic form.

Among the acute cases, we can at once dismiss as of no practical importance those in which minute miliary deposits are found after death, usually upon the mitral valve. The investigations of Meek, Teissier, Landouzy, and Gougerot and Barbiere, have advanced our knowledge of tuberculous endocarditis and proved the power of the infection to produce a vegetative endocarditis. Such vegetations need not be of large size, but they may show all the changes of a tuberculous lesion. Foci of necrosis and caseation occur with giant cell formation. Numerous lymphocytes are found in these areas, and tubercle bacilli may be present in considerable numbers. In some cases tubercle bacilli have been isolated from the general blood-stream, but it more frequently happens that the bacteria cannot be demonstrated in the tissues, although fragments of the vegetation have produced tuberculosis in guinea-pigs. More than one valve may be affected.

The clinical history of these cases is in no way remarkable; they appear as examples of acute endocarditis in which the active cause is usually a matter of dispute. When the acute process subsides, there remains a case of chronic valvular disease of an origin usually unsuspected. In other instances the course of the disease is malignant in type.

Durozier, and later Teissier, are of opinion that mitral stenosis may be a result of tuberculosis, believing that in such cases the condition is produced by a toxæmia rather than a direct bacterial invasion of the valve. Following this view, Landouzy and Barbiere would explain in this way some cases of early and mysterious mitral stenosis in childhood. Without denying the possibility of this, the writer believes he is expressing the opinion more generally received in this country when he states that such cases are usually the result of a mild and persistent, but overlooked, rheumatic process.

As with the pericarditis, so also with the valvular lesions. It must not be forgotten that such may arise in tuberculosis as a result of secondary infections.

Tuberculous Myocardial Lesions.—These take the form of miliary deposits or larger caseous nodules, which are usually met with in the wall of the left ventricle. They are not of clinical importance.

Allusion must here be made to the occurrence of a rapid pulse and excited action of the heart in some cases of pulmonary tuberculosis in childhood. Some

observers have found these so constant as to constitute a diagnostic symptom of some value. However this may be, there can be no doubt as to the reality of such a condition, which is presumably a result of the toxæmia, and it may lead us astray by focussing our attention upon the heart and leading us to overlook the pulmonary affection. This rapid action of the heart may be persistent and independent of any rise of temperature, as has been the case in some very striking examples under the writer's care.

On the other hand, there is a converse difficulty to be remembered in the occurrence of pulmonary congestion in severe rheumatic mitral stenosis. The resulting physical signs in the lungs are often exceedingly suggestive of tuberculosis, and the mistake of diagnosing tuberculosis and mitral stenosis under these circumstances is one which is not very infrequently made.

The occurrence of tuberculosis of the lungs in mitral stenosis is certainly very rare in childhood, but the combination undoubtedly exists.

Tuberculosis Multiple Serositis.—This term is not altogether satisfactory, and the condition which is under consideration now is rather a phase of infection than a definite disease.

The essential points to realize are easily explained. We recognize both the occurrence of acute pericarditis and the fact that an infection may produce with this acute pericarditis an acute pleurisy or peritonitis or meningitis. We also recognize that a severe serous inflammation may completely quiet down, and yet leave traces of its presence in adhesions, which by purely mechanical means may embarrass vital organs. In multiple serositis we have a condition between the two extremes. On the one hand occur outbursts of active serous inflammation, subacute in character, and on the other there occur adhesions the result of the quieting down of the previous attacks of the inflammation. There are, then, in this condition outbreaks of subacute inflammation of serous membranes, at one time pericarditis, at another mediastinitis, or pleurisy, or peritonitis. As a result of the pericarditis dense adhesions form, which may be so extreme as partially to strangle the heart and gravely embarrass its function. The pleural inflammation may be plastic or associated with considerable effusion. Ascites, a very constant feature, may be the result of a chronic perisplenitis and perihepatitis, or be caused by the embarrassed cardiac action, or both agencies combined.

A description of the chief lesions found at a post-mortem examination of one of these cases will show the final results of this process.

The autopsy showed that there was pericardial thickening of great density, with mediastinitis and some caseous deposits between the pericardial layers. The heart was small, and there was some thickening of the mitral valve. Both pleura were densely adherent, and the lungs were contracted, congested, and studded with miliary tubercles. There was much fluid in the abdomen, with extreme hepatic and moderate splenic enlargement. Perisplenitis and perihepatitis were present, and there was also recent tuberculous meningitis. The illness had extended over six years.

"Multiple serositis" is not a good term, for it is obvious that many instances of inflammation of several serous membranes occur without the classical symptoms of the condition under consideration. The particular serous inflammation that dominates these cases is the chronic pericarditis with mediastinitis leading to dense and practically complete adhesion.

Such a result may follow rheumatism, but tuberculosis, with its tendency to

run a chronic and relapsing course and to the production of much fibrosis, in particular favours the development of multiple serositis.

SYMPTOMATOLOGY.—The symptoms may be conveniently classed into three periods: those of the original illness; of the developed disease; and the terminal phase.

The original illness is a pericarditis, usually of the dry kind; there are pericardial friction, fever, and other symptoms of this condition. This phase of the illness may not come under observation, the disturbance of health being overlooked; but when it does the diagnosis usually inclines to that of rheumatism, particularly if there should be at the same time some endocarditis. Suspicion will, however, be aroused if there are no other symptoms or no previous history of rheumatism, and if, on the other hand, there are clear indications of tuberculosis. The pericarditis may be prolonged in duration, though subacute in character.

The second stage varies greatly in duration, and the usual history is that the child disappears from observation, and comes back with the symptoms well developed. Should the course be unusually rapid, the next event is probably a left-sided pleurisy, which may closely follow, or even accompany, the pericarditis, and be equally obstinate. The area of cardiac dulness is now found to be increased, and the percussion note unusually dull. In particular, attention should be directed to the upward increase of the cardiac dulness.

The heart sounds are muffled and rapid.

The liver is greatly enlarged, and ascites, and later dropsy, appear. The large veins in the neck are distended, and there is well-marked oedema. The striking feature of this period is the recurrent ascites.

The last stage is one of active tuberculosis. Headache, convulsions, coma, and vomiting, the result of a terminal tuberculous meningitis, may close the illness. The temperature chart shows no striking features in these cases, and there may be a subnormal variation of some extent for months before a terminal rise due to the acute exacerbation of tuberculosis.

When the course of the illness is more chronic, there may be long bouts of moderate fever coincident with outbreaks of local inflammation. Thus, in one case there were three attacks of pericarditis, many attacks of pleurisy, and some outbreaks of abdominal pain suggestive of local peritonitis.

The course of the illness, when once thoroughly developed, is steadily, though often slowly, downhill, and the most striking symptom is a recurrent ascites, which requires repeated tapping. Gradually the child becomes emaciated, with a large abdomen, increasing dyspnoea, and general weakness. Death may result from tuberculous meningitis, which has been the cause in every fatal instance under the writer's observation.

DIAGNOSIS.—This is difficult for three chief reasons: The rarity of the complaint; the difficulty of getting an accurate history of the commencement of the illness; and the outstanding enlargement of the liver.

It is very necessary in making the diagnosis to keep before one's mind the cardinal features of multiple serositis—viz.: (1) The evidences of adherent pericarditis; (2) the striking enlargement of the liver and the ascites; (3) the recurrent exacerbations of local inflammation.

Simple adherent pericardium, then, will be excluded by the evidence of recurrent signs of active pericarditis and pleurisy, and guided by the three cardinal signs we are more likely to escape from the danger of mistaking the enlarged liver either

for a primary disease of that organ or for some tumour arising in the neighbourhood and pushing the liver forward.

Congenital visceral syphilis, with cirrhosis of the liver and ascites, may be suspected, but in these cases there will be no evidence of an adherent pericardium, and there may be well-marked jaundice. Banti's disease will be differentiated by the absence of signs of pericardial adhesion, recurrent pericarditis, or pleurisy.

The **PROGNOSIS** is grave. It seems possible that the condition may stop short of the declared disease, but when the cardinal features are present the course of the illness is downhill, though variable in its duration. The shortest course under the writer's observation has been nine months from the initial pericarditis; the longest remained in the second stage for over five years.

TREATMENT.—This would appear to be entirely palliative. It is possible that tuberculin in the future may prove of some service, but at present there appears no evidence of its value. Brauer's operation for fusing the pericardium from external adhesion is not likely to be of service in young children with flexible chest walls. In a boy of sixteen operated upon by Trotter—a case which would seem to be of this nature—there followed distinct improvement, and at the time of writing, three years later, he is still able to lead a comfortable life.

In one case a Talma-Morison operation was done, but this was not a tuberculous case, and no real improvement followed.

Typhoid Fever.—In general terms it may be asserted that the heart seldom suffers greatly in typhoid fever of childhood, and upon this depends one of the elements in the favourable prognosis that can usually be given in such cases. There are, however, notable exceptions, and death may result from a sudden or unexpected collapse with extreme cardiac failure, or from repeated attacks of acute dilatation, or from a condition of irritable weakness termed by Mousous the "ataxic-dynamic form," which Bernsdorff and Wullaume look upon as the direct result of the action of the typhoid poisons upon the nervous centres.

In an outbreak of typhoid fever under observation, when the first definite day of the disease could be accurately ascertained by the temperature chart, the writer noticed a remarkable feebleness of the heart for some days after the first evidence of the toxæmia. Both endocarditis and pericarditis may be a direct result of the typhoid infection, but they are rare occurrences and the pericarditis usually of mild degree. On the other hand, secondary infections may produce a fatal suppurative pericarditis.

The most important changes, however, are found in the myocardium, and take the form of fatty changes in the muscular fibres, with interstitial inflammation and cellular infiltration of the supporting framework.

SYMPTOMATOLOGY.—Sudden Collapse.—This is a rare occurrence in childhood, and most likely to occur after some unusually severe diarrhoea, or even after one very copious motion. The child's face turns ashen, respiration becomes hurried and sighing, the pulse fails at the wrist, and the heart sounds can hardly be distinguished. Death may occur almost at once, or the child lie collapsed and nearly pulseless for an hour or more, and then expire. Minor degrees of the condition recover with extreme ease in passing.

The *ataxic dynamic form* of Mousous is liable to occur in nervous children. About the twelfth day the pulse becomes much more rapid (130 to 150), but is

regular, and the cardiac sounds clear and rapid. With this there are restlessness and delirium. As the strength fails, collapse supervenes.

The myocardial form is to be suspected when the pulse and heart become unduly rapid for the stage and severity of the attack. This feeble action, which is accompanied by some dilatation and weak heart sounds, may remain for days an anxiety to the attendant, and may be complicated by sudden attacks of increased dilatation with collapse, in one of which death may occur.

In these there are general muscular prostration, rapid breathing, livid pallor, cold extremities, scanty urine, and pulmonary oedema. The liver is enlarged and slightly tender. Such collapses may be recovered from, and on more than one occasion.

In some cases the pulse remains rapid and irregular for weeks after an attack of typhoid fever, and Mollard has demonstrated in such a condition, where death had resulted from an intercurrent disorder, evidences of myocarditis. Sudden death during convalescence is exceedingly rare in childhood, and a slow, irregular pulse at this time, though at first alarming, is of good augury.

TREATMENT.—Much in treatment will depend upon skilful nurses, and they need not only to be skilful, but to be blessed with an intelligence that rises above the execution of mere hospital routine.

The fulminant cases would appear to react to no treatment; powerful stimulants, oxygen, digitalin, strychnine, adrenalin, and camphor, have all been tried without success. The nurses must be closely on the watch for any precursory warnings of such a condition, as shown by unusual collapse after movement or after the exertion of defecation. The writer believes that he was able to avert a catastrophe in a case of this kind. The patient, a little boy, had lost his sister from a sudden collapse, and he himself had shown serious heart failure after the exertion of a large motion. Constipation was produced by opium, and then, after free stimulation, the bowels were opened by cautiously given enemata. The child made a complete recovery.

The more common forms of dilatation of the heart will be dealt with by cardiac tonics and extreme care in the nursing. Tepid baths are recommended by Moussons and others, Moussons advising that the child should be kept in the bath for three or four hours at a stretch.

Measles.—There is considerable difficulty in defining the part that measles plays as a factor in heart disease. The writer has records of a considerable number of examples of valvular disease in which the only previous illness known was this exanthem, but these cases were all of the rheumatic type, in that they showed the usual tendency to other rheumatic manifestations and to relapses. It would seem reasonable to believe that during the sore throat of measles the rheumatic infection had gained access to the system. It is also indisputable that pyo-pericarditis may occur, but this is probably pneumococcal and associated with complicating bronchopneumonia or erysipela.

On the other hand, myocarditis has been described in fatal cases of measles, and it cannot be denied that neuro-muscular damage may sometimes result as the direct effect of the disease.

Clinical evidence certainly points to the occurrence of a remarkable tachycardia as an occasional event during the acute stage of the disease, lasting for some weeks in the convalescence.

Paroxysmal tachycardia has also been recorded as a sequela.

THE TREATMENT will consist in additional precautions during and after convalescence. In cases in which there is a question of a return to winter games of a violent form, due care should be taken that the heart has resumed its normal condition; otherwise there may follow troublesome dilatation and tachycardia.

Influenza.—Influenza resembles diphtheria in the profound damage that it may do to the neuro-muscular structures of the heart, while endocarditis and pericarditis would appear exceptional events.

THE PATHOLOGICAL CHANGES that have been observed are chiefly those of fatty degeneration of the cardiac muscle.

SYMPTOMATOLOGY.—In infancy there may be rapid cardiac failure, associated with severe influenzal toxæmia. Lividity, vomiting, cold extremities, and rapid panting breathing, occur with feeble, irregular heart sounds and some dilatation. Death may occur in a condition of profound collapse.

Even in later childhood this condition may occur, and death result from syncope. Recovery in these cases is slow, and relapses, which may even prove fatal, are probable. If undue exertion is indulged in, palpitation, precordial discomfort, and dyspnoea, reappear, and with these a recurrence of dilatation.

A relapse of influenzal dilatation of the heart, when it is severe, gives perhaps the most striking clinical picture of acute asystole in childhood. The temperature may not rise above the normal, but the pulse runs up to 100 or 120 to the minute and is small and easily compressible. There are orthopnoea, panting respiration, and heart pain. The cardiac impulse may be felt, tapping in character, in the anterior axillary line, and the cardiac dulness is much enlarged transversely. The first sound is short, and may be followed by a soft systolic murmur; the pulmonary second sound is accentuated. A very striking phenomenon is the rapid enlargement of the liver, which is a valuable index of the degree of cardiac failure. The face is livid and the expression anxious. Slight oedema of the ankles, oedema of the pulmonary bases, vomiting, restlessness, wandering delirium, and diminution in the output of urine, complete the signs of the acute heart failure.

A good many observers have directed attention to the development of a harsh basal murmur as a result of influenza. The explanation of this is very obscure. To the writer it seems possible that some cases of supposed patent ductus arteriosus are of this nature. In three children in which a curious harsh murmur, with a maximum at the level of the third left costal cartilage, was audible, this diagnosis had been made, but in each there had been previously no hint of congenital heart disease, and the cardiac symptoms had dated from an attack of influenza. In two of these cases some three or four years later the diastolic element of the murmur had been lost, but there was still some remnant of the harsh grating sound in systole. It must be admitted that the physical signs of a patent ductus arteriosus may also alter in the course of time, but it was a remarkable fact that in each of these cases influenza had been looked upon as the exciting cause of the heart affection, and that in one the doctor who had attended the child from birth was confident there had been no congenital lesion.

THE DIAGNOSIS is a difficult one unless there is clear evidence of an epidemic of influenza in the house. The pains in the limbs and sore throat are suggestive of rheumatism, and there is no doubt that a certain number of cases diagnosed as

influenzal are in reality rheumatic. The most suggestive signs are the absence of a valvular lesion and the presence of dilatation and cardiac weakness closely resembling that found in diphtheria associated with a strong influenzal history, and tendency to relapse.

PROGNOSIS.—In the fulminating cases of infancy it is almost hopeless. In the less severe cases recovery is slow, and often uncertain for some weeks owing to the tendency to relapse. In almost all cases recovery is slow, and, though eventually complete, needs patience and care.

TREATMENT.—In the fulminant cases oxygen, digitalis, strychnine, camphor, and stimulants, have all been tried, but the reaction to any remedy is of the slightest.

In the acute cases the treatment is conducted upon the lines of cardiac dilatation from any cause, but especial stress must be laid upon the mysterious tendency to relapse, for this emphasizes the extreme caution that is needed in relaxing any precautions for the avoidance of exertion. Should the heart return to its normal dimensions, absolute rest must be persevered with for at least a month, and each forward step carefully studied.

In the milder forms, which are sometimes overlooked, and then apt to be complicated by heart strain, great patience is required. If they are subjected to any overstrain, rapidity and irregularity of action, and some dilatation, will certainly follow. The child becomes breathless and nervous, and the recovery will be delayed, possibly for months. On the other hand, with rest and the use of mild tonics recovery generally occurs, and is eventually complete.

Congenital Syphilis.—There has been considerable divergence of opinion expressed as to the influence of congenital syphilis in the production of heart disease in childhood, and it is possible that its importance has been under-rated.

PATHOLOGY.—In 1899 the writer published two cases of acquired cardiac syphilis which illustrated the fact that acute changes may be present in the heart wall which are not apparent on naked-eye examination. These changes were both parenchymatous and interstitial, and he attributed the heart failure in these cases to the microscopic lesions rather than to the obvious and gross aortitis that was present. Since that date Mrazek, Adler and Wertheim have directed attention to the same feature in congenital syphilis, and, further, Wertheim has demonstrated the *Treponema pallidum* in these focal lesions.

The pathological changes are of three kinds:

1. Endarteritis obliterans of the smaller coronary vessels.
2. Fatty and other degenerative changes in the muscular fibres.
3. Cellular exudation between the muscular bundles and around the blood-capillaries.

These changes have been noted in the hearts of infants suffering from severe hereditary syphilis, who have sometimes died unexpectedly, and in whom no cardiac lesion had been suspected.

Upon these grounds it is claimed that more cases of irregular and feeble heart's action in childhood are the result of this infection than is generally suspected. It is also claimed, and apparently with some good evidence in its support, that congenital syphilis is one cause of congenital heart disease, either by producing arrest of development or focal endocarditis, or a combination of the two lesions.

It follows that, if in a case of congenital syphilis we are led to believe there are

signs of myocardial disease, mercurial treatment should be persisted with steadily and actively until there is real and continued improvement.

Meningococcal Infection.—This is an exceedingly rare event. Westendorp reports such a case in a child of one year suffering from the epidemic form of cerebro-spinal fever. Weichselbaum and Ghon describe another in a girl of nine years. In both instances there was a meningococcal septicæmia, with lesions in the respiratory tract as well as in the cerebral meninges and heart. The type of endocarditis was malignant, and the symptoms were lost in those of the general meningococcal septicæmia.

The prognosis in such cases must be practically hopeless, seeing the severity and multiplicity of the lesions.

The treatment consists in the early use of a serum such as Flexner's as soon as the diagnosis is made from blood-culture or from the cerebro-spinal fluid.

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CHRONIC HEART DISEASE.

There may at first sight seem to be little difference between chronic rheumatic or other forms of carditis and chronic heart disease; but there is considerable importance in the distinction, even if it only serves to emphasize one of the most striking of all differences between cardiac disease in childhood and adult life.

This is the susceptibility of childhood to infections, the result of which is that the course of chronic heart disease—that is, of mechanical embarrassment of that organ by the scars of a former active carditis—is continually altered and imperilled by new waves of active disease.

When this difference has been thoroughly realized, it becomes apparent that the chronic heart disease of childhood and adult life differ only in details.

The importance of active infection is well illustrated by the results of an analysis of 100 fatal cases of heart disease due to rheumatism under twelve years of age, in eighty-six of which there was evidence of active disease at the necropsy. It is this, as pointed out before, which makes the prognosis of this form of morbus cordis uncertain, for every attack of fresh infection tends to produce in a greater or less degree some measure of angiot. In adult life the tendency to active cardiac rheumatism diminishes, and we repeatedly meet with examples of chronic heart disease in which the final stages are the result of the failure of the heart to struggle against the burden it bears of mechanical imperfections produced by a disease of which the activity has died down years before. This is the condition meant here by "chronic heart disease."

The great majority of cases of chronic heart disease that prove fatal in childhood are examples of this condition complicated by renewed infection. Yet a number still remain in which there is no fresh attack of rheumatism, and it is with these, and with those due to other loaded infections, that this section is concerned.

Chronic Myocardial Disease.

This condition may follow various infections, notably rheumatism, diphtheria, and influenza.

SYMPTOMATOLOGY.—Whatever the cause, the results are much the same. There is usually pallor, and with it nervousness, lack of muscular power, dyspnoea on exertion, and a tendency to palpitation and syncope. The pulse is increased in rate, often irregular, and much affected by posture. The heart is enlarged, the impulse feeble and ill-defined, the cardiac first sound short, the pulmonary second sound accentuated, and a systolic murmur, with its maximum external to the nipple or at the base, may be audible.

The progress to recovery may be slow, and in some cases permanent weakness and instability of action remains. More frequently, after prolonged rest and avoidance of overstrain, the heart becomes stronger, and eventually recovers to all appearances completely. This is presumably a result of hypertrophy and increased efficiency of the healthy muscle. These cases are closely related to those which are described later under functional or neuro-muscular affections.

Chronic Valvular Disease.

Mitral Regurgitation may produce no obvious impairment of health or strength, and, except for the systolic bruit—over-attention to which has been the cause of much needless anxiety—may leave no evidence of cardiac damage.

Among these slight cases are a considerable number in which, in the course of years, the murmur may disappear and no trace of disease be left. It is remarkable, if sufficient care is taken in the original illness, what recuperative power the heart possesses in this condition.

If the lesion is a considerable one, there must necessarily be hypertrophy of the left ventricle, but the compensation may be complete, and all symptoms remain in abeyance. When, on the other hand, the pericardium and heart wall have been severely damaged, mitral regurgitation may be a serious condition, and follow a disastrous course. Compensation may then never be properly established, and only reach a degree sufficient to maintain an imperfect circulation when the child is leading the life of an invalid. The outlook in such cases is almost hopeless.

The aspect of the child is that characteristic of mitral disease, and orthopnoea is a frequent symptom. The pulse is rapid, and may be very irregular. The precordia projects from the pressure of the enlarged heart, the impulse of which is diffuse, though often forcible and felt as far out as the left axilla. There may also be signs suggestive of pericardial adhesions. The cardiac dulness is greatly increased, and there may be a systolic thrill, or, if the lesion is a combination of regurgitation and stenosis, a short presystolic thrill felt over the impulse. At the impulse, in the left axilla and at the back a loud, long systolic murmur is audible. Evidences of asystole are shown by the large cervical veins, enlarged and tender liver, congested pulmonary bases, and dropsy. The digestion is impaired by the chronic congestion of the gastric mucosa. The urine is scanty and albuminous. Attacks of severe dyspnoea with cyanosis occur not infrequently at night, due to the regurgitation of the right side of the heart. In many cases the labouring right ventricle produces a feeble epigastric pulsation. The illness may be protracted,

and partial improvement occur, only to fail again when the child is released from the skillful nursing and treatment.

It becomes apparent that the heart is too much injured to react to any remedies, and the end, though often delayed for many months, approaches inevitably either by complete asystole resulting from progressive weakness the result of prolonged suffering and inability to take food, or from an acute outbreak of enditis.

In all forms of failure of compensation general wasting is a common symptom.

Mitral Stenosis.—When mitral stenosis is the prominent lesion, the course is rather different. Tachycardia and palpitation are more common, anginal attacks, probably originating in the right heart, more likely. Repeated bronchitis is a

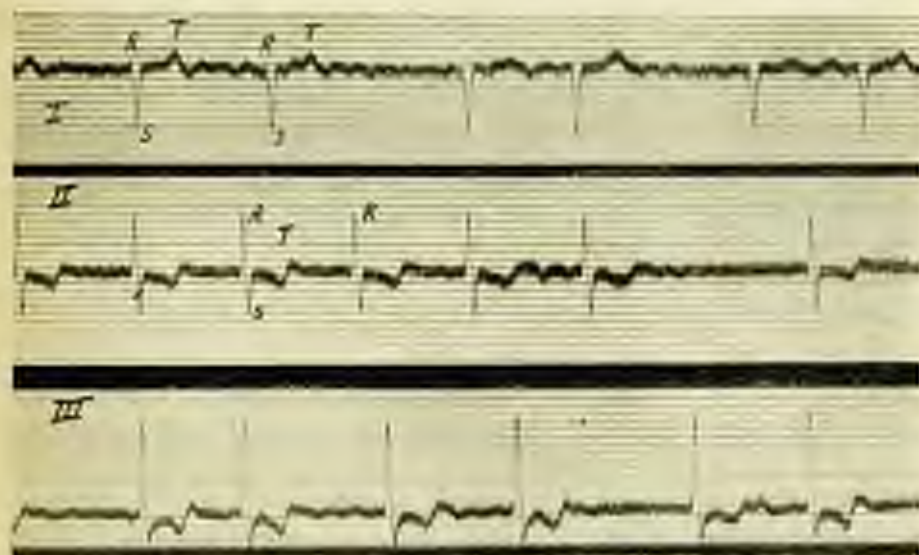


FIG. 48.—ELECTRO-CARDIOGRAM SHOWING ATRIAL FIBRILLATION IN MITRAL STENOSIS. (LEWIS.)

In these tracings it will be seen that (I) the auricular contraction is absent, and replaced by a series of undulations before (R).

striking symptom in some of these cases, and may be the starting-point of a complete breakdown. If pulmonary congestion is severe, hæmoptysis may occur. The great embarrassment of the right heart in failing mitral stenosis leads to antero-inferior thrombosis, and infarction may then result, producing hæmoptysis or hæmaturia, or enlargement of the spleen with pain, or, again, a sudden hemiplegia, which may either remain permanently or recover. Extreme irregularity of the pulse from the superposition of auricular fibrillation may develop, and in these cases the pulse is always of singularly small volume. With the superposition of asystole the presystolic murmur gradually disappears, and the pulmonary second sound becomes fainter.

Instead of the presystolic rumbling murmur, a diastolic blowing murmur may appear; but it must be insisted that a to-and-fro mitral murmur may occur in severe mitral regurgitation without any stenosis, and it would be an error to suppose

that in childhood this diastolic mitral murmur was necessarily an evidence of contraction of the mitral valves.

Ascites may anticipate other dropsical symptoms, and be associated with a much enlarged and hard liver. A rare event is paralysis of the left recurrent laryngeal nerve from pressure of an enlarged left auricle. Lastly, in some cases there is extraordinary vitality in spite of the failing circulation, and the terrible spectacle may be seen of a child with gangrenous fingers or toes still clinging to life.

Compensated Mitral Stenosis of moderate severity may show no obvious symptoms, but as compared with the milder mitral regurgitation is a more treacherous lesion. Growth is often stunted, the high-coloured cheeks and lips are frequently cyanosed, the child is more nervous, more liable to suffer from palpitation and dyspnoea upon exertion. Over-exertion may bring on dangerous tachycardia, and slight chills cause bronchitis.

It has already been mentioned that dropsy is not as frequent or severe in childhood as in adult life, and the explanation put forward is that heart failure in the former is not so often passive as the result of active disease. Experimental evidence of recent years has tended to cast doubt upon the influence of mechanical embarrassment in producing dropsy. The writer is, however, convinced from clinical evidence that it is an important factor, and this is borne out by an analysis of twenty-five cases of severe cardiac dropsy in childhood. The great feature in these cases was the occurrence of severe valvular lesions or mechanical embarrassment from great pericardial adhesions. If with these there is a strong heart rather than one damaged by virulent infection, dropsy will occur. Doubtless the healthy tissues and the young bloodvessels are agents in preventing an extreme degree of this dropsy.

In childhood we can readily differentiate a group of mitral cases in which dropsy and ascites are predominant features, and this not necessarily with any great engorgement of the bases of the lungs. In these cases we find the liver greatly enlarged and very hard, and there results a difficulty in the portal circulation favouring ascites. It is probably correct to suspect in many of them the complication of an adherent pericardium.

Aortic Regurgitation.—1. The solitary lesion is probably rare. Six cases under the writer's care in which there was no clinical evidence of mitral disease resembled one another very closely. Three were of mild degree and compensated, showing no symptoms except some pallor and breathlessness on active exertion. The condition of the heart and pulse were characteristic.

The patients were severely affected and anæmic and subject to attacks of giddiness, epistaxis, and actual syncope. They are also extremely nervous, and suffer from attacks of heart pain. Sudden death may occur. If the history of such cases is traced through adolescence to adult life, it will be found that some patients develop acute and repeated attacks of angina under thirty years of age, and may die in one of these attacks.

The pulse may be so collapsing in childhood as to disappear from the finger when the hand is raised above the head.

The aortic regurgitant murmur is particularly liable to be overlooked in childhood, apparently because it may be most audible to the left of the sternum, or sometimes is heard only in the epigastrium or immediately behind the sternum.

2. The combined mitral and aortic lesions (regurgitations) form the most important group of cases. The condition is a serious one. Of thirty-five patients under

the writer's observation, thirteen have died, the oldest at the age of fifteen years. The usual cause of death is an intercurrent attack of endocarditis, and it is the writer's experience that this lesion is more liable to be complicated in adult life by the superposition of malignant endocarditis than any other.

The symptoms of this group fall into two classes—those in which the aortic lesion is prominent, and those in which the mitral is the essential one. The former will be recognized by giddiness, epistaxis, nervousness, pallor, and other evidences of aortic regurgitation, and the heart and pulse will confirm this lesion. The latter will show mitral symptoms.

The **DIAGNOSIS** rests upon the physical signs pointing to the combined lesion, and the aortic regurgitant bruit to the left of the sternum must not be mistaken for a pulmonary regurgitation, which apart from a rare congenital condition, is practically unknown in childhood.

Aortic Stenosis.—This is a rare lesion at all ages, but more particularly so in childhood, because we do not meet with the syphilitic and degenerative diseases of the aortic valve associated with calcareous deposits.

As in the case with mitral stenosis, this lesion, which is almost invariably rheumatic, may be a pure stenosis or supervene upon a previous aortic regurgitation.

Symptoms are in abeyance when compensation is well established, but if the heart commences to fail, pain of an anginal type is complained of, and fainting attacks may occur, with dyspnoea.

The pulse is small and slow and the wave prolonged. There are marked hypertrophy of the left ventricle, and a forcible impulse.

The characteristic murmur is a harsh systolic bruit, and there may be a systolic thrill. The murmur is conducted into the large vessels of the neck. The lesion does not reach an advanced stage in childhood, and there is no report of a fatal case in the records of the Children's Hospital at Great Ormond Street.

Chronic valvular disease on the right side of the heart, as an acquired lesion, is so rare and ill-defined that it need not detain us further than to point out that tricuspid regurgitation due to *secondary dilatation* of the right ventricle and relative incompetence is a constant event in the failing heart resulting from mitral lesions, in severe dilatation of the heart, and is met with in some aortic cases and in adherent pericardium. When this relative incompetence supervenes, the liver is enlarged and becomes tender, the jugular veins are full, oedema appears, and the urine becomes scanty, high-coloured, and loaded with urates. The bases of the lungs are congested and the digestion impaired.

The recognition of the consequent dilatation of the right auricle, with increase in the cardiac dullness to the right of the sternum, the labouring right ventricle, and small radial pulse, are physical signs of the utmost importance in the treatment of such cases.

Adherent Pericardium.

The two infections that are most likely to produce this condition are rheumatism and tuberculosis, and of these the former is by far the more important. The tuberculous form tends to strangle the heart by the density of its adhesions; but the rheumatic is usually associated with considerable hypertrophy and dilatation. The ultimate result will largely depend upon two factors—the amount of myocardial damage and the degree of external pericarditis.

It is not too dogmatic an assertion to state that in most instances of pericardial adhesion the diagnosis is a question of probability rather than of actual certainty, and that it is impossible to detect a more obliteration of the pericardial cavity.

How possible it is that we may be right in our surmise rather by intuition than accurate diagnosis is borne out by the analysis of 150 fatal cases of cardiac.

In 113—that is, 75 per cent.—the pericardium was more or less adherent, and, of these 113, in 77 the adhesion was general.

The signs of chief importance are—1. Immobility of the impulse when the patient is turned from side to side. In childhood a very large heart and a narrow chest will produce the same result.

2. Increase in the cardiac dulness, with damping of the clearness of the cardiac sounds.

This sign is also present in pericardial effusion.

3. Fixity of the cardiac dulness with change of position, and an increased sense of resistance to the finger on percussion.

This sign may also be met with when a much enlarged heart is enclosed in narrow thorax.

4. Precordial bulging. This also occurs with an enlarged heart free of adhesions.

5. Systolic retraction of intercostal spaces over a wide area, diastolic recoil of the chest wall, systolic indrawing of the lower end of the sternum, and retraction of the lower intercostal spaces posteriorly on the left side, have all a certain value when taken together with other signs.

6. The pulsus paradoxus is a phenomenon which is also met with in respiratory embarrassment and sometimes when there is a patent ductus arteriosus.

7. Inspiratory distension of the jugular veins, from the presence of constricting adhesions around the large venous trunks made taut by the inspiratory movements, is an unusual sign.

8. A to-and-fro apical murmur and reduplication of the second sound without clear evidence of mitral stenosis.

The symptoms of an adherent pericardium are those of a gradual asystole when the condition is severe, or of an imperfect compensation when of lesser degree. Should the heart be able to cope with the added work entailed by adhesions, there will be no symptoms.

In the great majority of cases the attitude of the physician is to infer that, when the symptoms due to poor compensation are out of proportion to the apparent cardiac lesions, and when, in addition, there has been a previous pericarditis, the cause for the unfavourable course is an adherent pericardium. Much ascites and oedema also favour this diagnosis. The fallacy that is involved is the impossibility of ascertaining how much this failure of compensation is dependent, not upon the pericardium, but the damaged myocardium.

Cardiac Asystole.

This is a useful term to mark out the stage of failing heart in childhood, whether the result of valvular disease or other cause. It comprises all forms of cardiac failure, from a sudden fatal syncope to the slow but steady failure of a heart overburdened by mechanical disability. It may be of value to the reader if under this heading a brief allusion is made to some of the cases of sudden and unexpected asystole in childhood.

Myocardial lesions the result of diphtheria or of other infections may end abruptly, in some cases no cardiac lesion having been even suspected. Cases of viral and nortic disease, as also some rare cases of *un suspected pericarditis*, may end by sudden death, although such an occurrence is infrequent.

Epileptic attacks account for some cases of sudden death, and some of these are the result of asphyxia. In others, however, as the writer has seen, epileptic attacks have been associated with heart disease, either congenital or acquired, and in these the sudden death has been apparently of cardiac origin.

Another group is that in which sudden shock or *fright* has been the cause. It is remarkable, seeing how greatly a sudden shock may affect the cardiac action, that such cases are not more frequent, especially when the extreme nervousness of some of the modern children is remembered.

Another group may perhaps be classed as *toxic*. There are cases in which after some operation, usually abdominal and associated with suppuration, a child has appeared to be progressing favorably, and has then suddenly fallen back dead.

Acute suppurative hemorrhage and "rapidly fatal constitutional diseases" are perhaps best placed here. *Gastro-enteritis*, particularly the epidemic summer diarrhoea, may be associated in some cases with extraordinary heart failure. Death may occur abruptly in the acute attacks, or sometimes equally suddenly when the stage of convalescence has been entered upon.

During the convalescence from other acute infections, death sometimes occurs unexpectedly by sudden heart failure. Thus, during an attack of severe influenza, or even typhoid fever, rapid asystole may supervene.

The *status lymphaticus* has been credited with many cases of sudden death from circulatory failure, and many of the examples included in the preceding groups would be claimed by some as examples of this condition.

In sudden death associated with *obstetrical anaesthesia*, a subject of extreme interest, it is well known that death may occur at the outset of the anaesthesia, or during over-deep anaesthesia, or directly after the cessation of the anaesthesia.

Another group of importance is the sudden heart failure that may follow exploratory puncture of the chest.

The preceding groups include many of the best-known forms of abrupt asystole.

Acute asystole is much more frequent than sudden heart failure, and is well illustrated by the cardiac failure that occurs in a virulent pericarditis or severe infectious dilatation.

The symptoms are dyspnoea, pallor, lividity, restlessness, autonomic delirium, and vomiting. The physical signs are those of a failing heart with moderate enlargement of the liver, possibly with some slight oedema.

Chronic asystole, as in the last stage of chronic heart disease, has already been described. In childhood congestion of the bases of the lungs, extreme anasarca, prolonged mental anguish, or even mental derangement and persistent and severe albuminuria, are much less frequent than in adult life, but the general character of the signs are of the same order.

TREATMENT.—THE MANAGEMENT OF A CHILD WITH COMPENSATED HEART DISEASE.—There are some points of general importance to be considered.

First of all it is essential to estimate so far as possible the reserve power of the heart. This should not be done by any pseudo-scientific tests, but by careful observation of the results of ordinary exertion. The child has to live in the

ordinary workaday world, and the medical man and parents have to learn how much he can do.

Attention must also be directed to the nature of the lesion. Slight mitral lesions, for example, would be of no importance at all in some cases if we could throw away the stethoscope. Aortic lesions, however well compensated, demand more care.

When it is clear that the condition is such that chronic invalidism is out of the question, it remains to be decided how far it is permissible for the patient to receive an ordinary education and take part in the ordinary games. Lastly there is the choice of a career.

There will always be two difficulties to contend with: on the one hand, the heedlessness of youth; on the other hand, the danger of converting what might be a sane and healthy child into an introspective invalid.

If a medical man is convinced that there is a serious organic lesion, he can always give his reasons for that belief, and must not allow his judgment to be overwhelmed by the insistence of a parent who is what is called an "old sportsman." Such a man demands that his son should follow in his steps, and this parent may be most unreasonable because he sees that his son looks well when he is quiet. It is, however, a grave error to send a child who is breathless on moderate exertion among healthy, strong boys without the strictest supervision. However good compensation may be, it is not really wise, if there is considerable valvular disease, for a boy to enter into all competitive sports. He may succeed admirably, and the writer was acquainted with a first-class athlete who had a gross aortic lesion as a child, and yet rowing was among his accomplishments. At what a price these successes were won is clear when it is added that he died of rapid heart failure in the early prime of life, just when he had attained a good position and had a young family to support.

The intellectual side also calls for care. Children with aortic disease in particular are nervous, and if overpressed may break down badly.

When an ordinary life is out of the question, every attempt must be made to see that the modified one is as bright and broad in its outlook as possible. The advantage of occasional rests in bed when these children get irritable and thin is remarkable. A daily rest before the midday meal is also valuable. Warm clothing is needed, and during great cold and great heat these cases want special care. The diet should be simple, and no rigid exclusion of meat on account of rheumatic "tendencies" is advisable. Bright living-rooms and surroundings are a great assistance.

There is little scope for drugs. The general health must be maintained on ordinary principles, and probably the most valuable tonics are those which assist the digestion and act gently upon the bowels.

TREATMENT OF FAILING COMPENSATION (ASTHOLE).—The first step is an inquiry into the cause. Has there been a renewed *canthar* infection, or has there been over-exertion, physical or intellectual?

If there has been a renewed infection the patient will be treated for this, and, as it is usually rheumatic, the indications have been already given under that heading.

When there has been no evidence of renewed infection, we have to consider whether the condition is the result of some obvious indiscretion, or whether it is evidence that a heart which has barely maintained its efficiency with extreme care is beginning to give way. Such patients, accordingly, may come under our care

with an acute failure and urgent symptoms, of which the most important are severe dyspnoea, fainting attacks, and cardiac pain with auricular fibrillation, or with the more insidious warnings of progressive weakness and wasting, digestive disturbances, pallor, nervousness, and commencing dropsy.

When there is acute right heart failure, with a feeble pulse at the wrist, lividity, a struggling right ventricle, and dyspnoea, leeches should be applied over the precordia or liver, and oxygen administered. The bowels should be freely opened with calomel and salina, and digitalis, or a standardized tincture of digitalis, and strychnine given every four hours either by the mouth or hypodermically.

When there is dyspnoea with acute attacks of cardiac pain, the same measures may be needed at first. A capsule of nitrite of amyl inhaled by the mouth is sometimes valuable for the relief of pain. Atropine has also been recommended, pushed until dryness of the mouth is obtained. A prescription for a child of seven years is—

Liq. atropine sulphatis	℥i.
Liq. strychnine hydrochloratis	℥i.
Syrup. sciranti	℥ss.
Aqua chloroformi	℥i.
4ss. horis.					

It may be necessary to add liq. morphine hydrochloratis (℥ ss.) to this mixture, or even to give $\frac{1}{2}$ grain of morphia under the skin.

In all cases in which breathlessness is urgent, it must be remembered that the cause may not be wholly cardiac, but dependant more immediately upon the presence of a pleural effusion which needs aspiration. Rest and careful dieting are as needful as in acute carditis.

In the more chronic conditions due attention must be given to the particular cardiac lesion and the presence of any complications. It may be repeated that the simple measure of ordering a week in bed when these children are beginning to get thin and irritable has sometimes a remarkable effect in putting a stop to the first stages of a commencing failure of compensation.

Rest is specially beneficial to children with aortic regurgitation, and it frequently happens that mild sedatives, small doses of strychnine, and arsenic in alkaline solution, are useful drugs. Digitalis should always be given with caution to children with aortic disease.

In mitral stenosis bronchitis may be troublesome, and should be treated on general principles. When the irregularity of the pulse and absence of the rumbling murmur point to auricular fibrillation, digitalis may be serviceable in steadying the heart and controlling the rapidity of action. On the other hand, if the condition is one of nervousness and tachycardia, sedatives, such as bromides, may be added to the mixture, and the tincture of *Cactus grandiflorus* in 5-15 minim doses is sometimes useful.

Other mitral cases occur, comparable to those met with in adult life, in which there is much dropsy, orthopnoea, and a great decrease in the amount of urine passed. As a rule it will be found, when there is much ascites, that the liver is not only large, but unduly hard.

A drug doubtless uncertain, but of great value for increasing diuresis, is theocin-sodium-acetate, and it may be the only one that exerts any influence in this direction, as the writer has observed on several occasions. It needs, however, much care in its use, because it is peculiarly liable to upset the digestion, particularly

when combined with digitalis. So troublesome may this complication be that it may be necessary to give mouthful half an hour before the mixture, or to omit the drug for seven days, and then to give it only for two or three days in succession (gr. iii.-iv. is the average dose). When it is well borne, a rapid disappearance of ascites and dropsy may follow its use. Diastin in tablet form (gr. v.) is also of value, and digitalis itself may prove effectual without other assistance. Anacardum in tablet form (a combination of *Oxydendron arboreum*, *Sambucus canadensis*, and *Urginea scilla*) has been much praised, and is at present on trial.

The question may arise as to whether it is necessary to perform paracentesis in cases with severe ascites. When the heart is embarrassed by the abdominal distension, and there is little urine passed and much general discomfort, no progress will be made until this has been done.

Sleeplessness and general misery are indications for small doses of opiates, and *sepias* (℥iii. for a child of seven years) in a draught at night, with or without some bromide and tincture of *Lupulus* is very valuable. However apparently extraordinary, the position of greatest comfort in bed should be encouraged.

Vomiting must be promptly attended to by curtailing the diet, or even stopping all food by the mouth for twenty-four hours. Bismuth and alkalies, or cathartics and small doses of brandy, are all helpful. If the liver is engorged, calomel, grey powder, or small doses of emeryin, are indicated. A fancy diet is frequently needed when the appetite fails in these chronic cases.

When improvement commences, ample time must be given, and skilful massage may be helpful in restoring tone to the muscles and getting rid of the last traces of oedema. The forward steps are on the same lines as those indicated in the treatment of *carditis*, but we must be prepared to find that there is a limit to improvement in many cases, beyond which no care or skill can carry the child.

A dry and somewhat bracing climate is the most suitable, and sunshine is of valuable assistance. Extremes of heat and cold are injurious, and even more so are cold, damp, low-lying neighbourhoods. A quiet, regular life is of the utmost importance, and no excitements should be allowed in the evening before bed-time. High stairs throw much strain on the heart, a point which is sometimes overlooked by those who have the care of children with chronic morbus cordis.

FUNCTIONAL AFFECTIONS OF THE HEART.

The term "functional" is unsatisfactory, but difficult to replace, and the subject is one of much difficulty, and one also which at the present time may be justly described as being in a stage of active investigation, and therefore of considerable uncertainty. It is fortunate that in childhood the nervous element—using this term in the purely practical sense—which is so often prominent in the adult, is less pronounced. In spite of this, it must be insisted that much harm may result to intelligent children of nervous temperament from indiscreet remarks made in their presence by doctors or parents; for as a result a loss of confidence may follow which is comparable to that which so often aggravates any suspicion of a cardiac disorder in the adult.

Among the most important of the subjects that come to the front in this section are disturbances of rhythm, slight dilatations, cardiac bruits of obscure origin, epilepsy and cardiac disturbances, and heart strain.

INTRODUCTORY.—There are some general considerations which merit a few lines before entering upon details. First among these is the realization of the fact that there is no boundary line between organic and functional affections in clinical medicine. The natural tendency is to err in the direction of alarmist views where children's hearts are concerned, but it may be added that experience encourages us to be cheerful.

There is, however, one difficulty which it is always well to bear in mind when considering the question of heart strain in childhood, and that is the possibility of some overlooked infection, such as influenza, which has made the heart more susceptible to this danger. In discussions upon the subject, the writer has repeatedly heard the assertion that heart strain is a very rare occurrence in the healthy boy; and true though this is, we cannot lose sight of the fact that in a large public school a boy may have had some illness, such as influenza, which has not been suspected, and then the influence of severe games may make itself felt only too clearly.

Another point of importance is an ability to throw oneself into the aspect of life that appeals to the boy; to remember something of the keenness to risk everything to excel, and the valuelessness of preaching prudence; to preserve also some memory of the extraordinary deeds that this age is capable of attempting, though not necessarily with success. Lastly there are the very real emotional struggles that arise at puberty between the flesh and spirit, which need such sympathetic handling.

In these cases it is often the art of medicine, rather than the scientific instruments of precision, that will be needed, and every case must need special thought. It is always advisable to learn as much as possible from the parents or guardians, and to avoid leading questions and particular references about the heart or pulse in the presence of the child.

The examination of the patient requires particular care, and a scheme of this kind may prove of service:

A. The history of the present illness:

1. Duration.
2. Leading symptoms.
3. Possible infections, especially a history of repeated sore throat, of rheumatism, or of influenza.
4. Possible overstrain, in particular foot races and long runs.
5. A history of violent exercise combined with hard intellectual work.

B. Personal history of the child:

1. As to previous illnesses.
2. As to temperament.
3. As to rapid growth.

C. The family history.

D. The present state. This must be a general survey of all the systems.

Proceeding along these lines, though we may not entirely explain the case, we are guarded against serious error, provided that the first steps in treatment are cautious.

It will be justifiable also to devote here a few lines to the question of athletics, for, whether we approve or not, in this country they take a prominent part in school life. This being so, we make a serious error in stopping a boy's games

without excellent reasons, for we thereby foster introspectiveness and bad habits. On the other hand, to forbid some of the best open-air games, and allow equally severe indoor ones, is to bring one's judgment into certain ridicule among those who are concerned with public schools.

Writing from a personal experience of the subject, there is no doubt that the most trying forms of exercise are the competitive ones which depend upon endurance rather than skill. Long runs and racing of all kinds fall into this group. Even amongst normal boys there is a considerable difference in the ability to obtain "a second wind." In racing there is the danger that the greater leverage obtained by a tall boy is more than counterbalanced by the immaturity of his rapidly-growing tissues. Of winter games, Rugby football is the most trying, but experience shows that surgical injuries are more to be feared than heart strain. Boxing, squirts, and fives are all hard exercise. Lawn-tennis doubles are mild events except for experts. Swimming in cold water is admirable, but when the temperature is low the factor of chill must be remembered. Bicycling may be a great boon or an equal curse. Golf is of value, and can be regulated, but is selfish. Cricket is difficult to estimate, but, unless the player excels, it seldom leads to over-exertion and has very great advantages. Heavy field-days with a rifle corps are among the most strenuous of all occupations.

DISURRANCES OF RHYTHM.

Disturbances of rhythm are at present the burning question of heart disease. This provisional classification may be helpful as a guide:

1. Physiological: Arrhythmias—viz.

(1) Respiratory.
(2) Orthostatic.
2. The premature contraction or extra-systole

(1) of auricular origin.
(2) of ventricular origin.
(3) of auriculo-ventricular origin.
3. Paroxysmal tachycardia and other forms of tachycardia.
4. Bradycardia

(1) False, associated with the extra-systole.
(2) True—(a) nervous; (b) due to lesions of the bundle of His.
5. The pulsus alternans.
6. Auricular fibrillation.

Physiological irregularities are well marked in childhood in association with respiration and change in posture. If there should be respiratory embarrassment, the irregularity may be very striking, and false bradycardia result from beats not reaching the wrist. In other cases the condition may closely resemble that of the perpetual arrhythmia associated with myocardial degeneration in later life.

Remarkable though these conditions are, clinical observation shows that the heart is not seriously affected, and soon recovers the normal rhythm when the respiratory difficulties are over.

A study of the pulse in young children during sleep will show most curious disturbances in rhythm. Grouped beats and irregular pauses are frequent in the healthy child, and illustrate that the easy circulation at this age rises above the need for precision in the heart's action.

In many children, particularly those of a nervous temperament, there may be a

considerable increase in the rate of the heart-beat when a change is made from the recumbent to the upright posture. This, again, is compatible with a sound heart. Again, at puberty it has long been thoroughly recognized that respiratory irregularities are often well marked, and this may be very puzzling to the medical attendant if not understood. Should the heart have been disturbed by some infection, such as influenza or diphtheria, this irregularity may be exaggerated during convalescence. This point needs more particular consideration, and will be referred to later in the section. These respiratory alterations are looked upon as of vagal origin.

Premature contractions occur in functional cardiac affections under various circumstances. Digestive disturbances are the most frequent exciting causes at this age, but sometimes a direct toxic origin may be suspected, such as, for example, smoking. Nervous disturbances at puberty, both in the male and female, have been attributed as reflex excitants of the extra-systole, which may also be met with at the termination of the rare attacks of paroxysmal tachycardia which have been from time to time described in childhood. In highly nervous children this type of irregularity has been noticed also as a result of mental strain. In some cases a regularly-recurring premature contraction, usually of ventricular origin, gives rise to the well-known coupled rhythm of the heart. In childhood this is most frequently met with in organic heart disease, being produced by the action of digitalis upon a heart in a condition of auricular fibrillation. So far as one can see, the outlook is good in most of these cases, and the problem of treatment is not concerned with the extra-systole, but with the general condition of the patient.

Tachycardia.—There may be remarkable acceleration of the heart-beat in many diseases of childhood, and also nervous influences apart from organic disease may greatly affect the pulse-rate. The general outcome is that the characters of the pulse in the young are a far less valuable guide to the condition of the heart than in adult life.

As an example of tachycardia, there is probably no better one than that met with in some cases of tuberculous meningitis when the intracranial pressure is high. This is the more interesting because it may follow an earlier phase in which the pulse has been notably slow and hesitating. In rheumatic heart disease, scarlet fever, and shorea, there may be sometimes persistent tachycardia.

It is not, however, these forms of tachycardia that are of especial interest, but that which has been termed "paroxysmal," differing in that the rapidity of the heart depends, not upon an increase in the rate of the normal beat, but upon the appearance of numerous pathological beats, termed by Lewis "ectopic."

This paroxysmal tachycardia may commence in childhood, as Herringham pointed out in his series of cases, and persist through adult life. In some instances it has been dated from an infection such as measles, and it occurs more frequently in females than in males. The attacks resemble those in adult life, and may last from minutes to days, the pulse-rate rising to 180 or 200, or even higher. The onset of the attack is usually sudden, and the exciting cause trivial or not apparent. The child looks ill, and the face is drawn and pale. There is some dyspnoea, and the child avoids active movement. In some instances abdominal pain has been the complaint. Physical examination at first shows no dilatation, although sometimes a systolic murmur becomes audible at the impulse. At the end of a severe attack dilatation may develop and the liver enlarge. The writer is not aware of a fatal case in childhood, but the condition may persist as a disability

throughout life. The average duration of attacks is two days, and several may occur in a year, or they may be even more frequent. Fortunately, the condition is a rare one, and very seldom arises before the eighth year.

Tachycardia and Epilepsy.—There are some cases in which paroxysms of extreme rapidity of the heart's action occur in children who have previously suffered from epilepsy. Two such cases have come under the writer's notice. The first, a boy of nine years, had been subject to epileptic fits up to the age of four years, and from infancy had attacks of what the mother termed "palpitation" of the heart. Although the epileptic fits had ceased, about every three weeks violent attacks of "palpitation" occurred without apparent cause, and always at night. Between the attacks the rate was about 80 per minute, and the action slightly irregular. The second, a female aged a year and five months, four days after a fit had some palpitation of the heart, lasting several days, and since then had been subject to attacks about once a week. These always commenced in the morning and lasted for some hours. The heart was apparently sound. Both these cases slowly improved under treatment with bromides.

Every effort must be made to discover the cause of paroxysmal tachycardia, for, if there is any hope of controlling the condition, it is natural to believe that the best chance is offered before what may be called the "pernicious heart habit" is thoroughly established.

It is to the general management of the patient that we look for most help—quiet and rest during the attacks, attention to the digestive system and bowels, and the exhibition of bromides, valerian, and digitalis. Between the attacks much attention must be paid to grappling with undue nervousness. Exercise must be regulated and an open-air life advised.

Various devices have been used to stop the paroxysm in the adult. Among the most important, so far as children are concerned, are the recumbent posture, slow swallowing of some inert drug in bolus form, the encouragement of vomiting, and pressure upon the *vagus* in the neck. It must be admitted that all methods of treatment are uncertain, and the condition is so rare in childhood that there is little *in vivo* evidence upon the subject to guide us.

Bradycardia.—In some cases a slow pulse, of a rate of 55 to 60, is apparently physiological and compatible with perfect health, and Moursous records several members in the same family as showing this peculiarity.

A slow, irregular pulse is not rare in convalescence from acute disease, particularly when the temperature falls below the normal line after the prolonged pyrexia of typhoid. This irregularity and slowness may persist for days or weeks, but is usually unassociated with any symptoms of importance, except it appear in the course of a diphtheritic paralysis.

Morquio recounts a remarkable event in the occurrence of bradycardia with syncopal and epileptiform attacks in eight children of the same family. The pulse-rate varied between 30 and 40 to the minute, and three of the children died suddenly. In one of these cases fatty degeneration of the cardiac muscle was found. Van der Heuvel describes a bradycardia associated with a patent interventricular septum, and Fleming, Kennedy, and others have described bradycardia as the result of true heart-block in fatal cases of diphtheria.

It is surprising to the writer that some degree of heart-block is not more common in rheumatic children, with their extensive and frequent cardiac lesions; but there

is little doubt that the future will teach us more about this condition in childhood, and already the literature is beginning to record examples illustrating heart-block in rheumatic heart disease studied by the most recent methods. Among examples of heart-block there is the important case fully investigated by Armstrong, Hay and Mockenberg of a boy of five years suffering from bradycardia, with Stokes-Adams symptoms, in which after death a lympho-endothelioma of the auriculo-ventricular node was discovered. There is Fleming and Kennedy's case, already alluded to—a girl of ten years dying of cardiac failure in diphtheria with bradycardia. The vagus nerves were reported to be free of change, the auriculo-ventricular bundle, and auricular and ventricular myocardium, to be much damaged. A less complete but interesting case also occurring in diphtheria is Magnus-Alsleben's. Fulton, Judson and Norris reported an example of congenital heart-block, but, in view of a subsequent criticism by T. Lewis, were later disposed to modify this interpretation. Echerique's case was remarkable for the severity of the syncopal attacks. The patient was an excitable girl of eleven years, one of a family of eight. There was no history pointing to congenital syphilis. The pulse-rate was 33 to the minute, slow and regular. Attacks



FIG. 48.—ELECTROCARDIOGRAM ILLUSTRATING SEVERE HEART-BLOCK IN A CHILD WITH MYXAL SYNDROME. (LEWIS.)

If we follow this tracing from left to right, interpreting the lettering as we go, it reads as follows: Auricular contraction (P), ventricular contraction (Q, R, S, T), short diastolic space, auricular contraction (P), ventricular contraction (Q), which when T is reached is smothered upon by another auricular contraction (P). This is blocked and not followed by a ventricular contraction.

of unconsciousness lasted from one to twenty minutes, and were associated with slight convulsions and incontinence of urine. Recovery was accompanied by crying and groaning and deep respiration.

The *Pulsus alternans* is usually associated with organic heart disease, and may be sometimes well marked in acute pericarditis. The general consensus of opinion shows that it is of ill omen. Admitting this to be true, it is difficult to know what exact weight to attach to the statement. For example, in a case of severe aortic regurgitation under the writer's observation for seven years, a very distinct pulsus alternans occurred during a relapse of acute carditis, yet the boy recovered from the attack. Three months later he dropped dead. So severe, however, was the cardiac lesion that seven years before this it did not seem he could have lived six months. Here neither the general condition of the heart nor the pulsus alternans were really helpful in making an accurate forecast.

It is also met with in such functional conditions as paroxysmal tachycardia, and as a result of cardiac overstrain.

FUNCTIONAL BRUITS.

Functional bruits in childhood are a puzzling study, and made more difficult by the possibility of the occurrence of congenital heart disease. There can, however, be no doubt as to their existence at all ages in childhood. Some writers have, indeed, doubted their occurrence in infancy, but a complete proof, coinciding with the experience of Marfan, came under the writer's observation. A child aged eighteen months, suffering from splenic anemia of infants, was brought to hospital with a loud systolic basal murmur that all regarded as evidence of congenital heart disease. The necropsy showed that this was entirely functional.

In rachitic infants, or in febrile conditions with anemia, a loud systolic bruit will frequently be audible at the base or apex or over the entire precordial area. In older children functional bruits are met with in anæmic conditions, and may be much intensified if the chest should be deformed.

A murmur of considerable interest is the cardio-pulmonary, which is best considered here. This is produced in the lungs by the pulsations of the heart, which so alter the vesicular murmur as to make it resemble a true cardiac murmur. It is met with most frequently in later childhood and adolescence, and its occurrence is favoured by rapid respirations and an accelerated and nervous heart's action. Sometimes this sound is audible on the left side, and cegethred breathing upon the right.

The most frequent sites are the left border of the heart in the second and third spaces, or just internal to the apex or at the right border of the heart. More rarely it is audible in the left axilla or at the lower angle of the left scapula. It is systolic in time, and the intensity rises with inspiration and falls with expiration, and disappears when the breath is held. Sometimes the recumbent position intensifies the loudness. When this murmur is complicated, as it may be, by the presence of an organic mitral murmur, the interpretation is most difficult, and the effect of posture upon the intensity of functional bruits has, in the writer's experience, been almost valueless as a means of distinguishing them from organic murmurs.

The following short scheme for the differentiation of the various systolic murmurs in childhood may be helpful to the reader.

Systolic Apical Bruits.—1. Due to organic mitral disease: A systolic bellows murmur following or accompanying the first sound conducted towards the axilla, sometimes associated with a thrill, and sometimes audible at the back. This murmur becomes louder with increasing cardiac power.

2. Due to functional conditions: A systolic murmur with its maximum intensity internal to the impulse, and fading away towards the left axilla. Influenced sometimes greatly by posture and respiration, and tending to disappear as the health improves.

3. Due to pericarditis: A systolic friction sound may occur in the region of the apex. It is superficial and diminished by pressure with the stethoscope, localized, and often alters into a to-and-fro friction rub.

4. Due to patent interventricular septum: Not strictly apical, but with its maximum intensity at the level of the third costal cartilage; superficial in character, and often accompanied by a very definite thrill. There is no definite line of conduction.

Basal Systolic Bruits.—1. Due to aortic stenosis (rare): Maximum over the aortic cartilage, and conducted into the neck. There is also a systolic thrill, a hypertrophied left ventricle, and a pulse of small amplitude.

2. Due to functional conditions: Usually louder over the pulmonary than the aortic area. No thrill. Often varying with the respirations and with position, and often associated with venous murmurs in the neck.

3. Due to mediastinal glands compressing the large vessels. There are usually evidences of mediastinal tumour.

4. Congenital aortic stenosis: The murmur may be at its maximum at a higher level than the aortic valve, being the result of coarctation. There may be no signs of a cardiac lesion other than this murmur.

Clinical Types of Functional Affections.

In studying functional affections of the heart, we have usually to consider, not one particular event, such as an irregularity of the heart or a bruit, but the entire history of the case and a complete physical examination. The position is often identical with that in the adult, in that we find many evidences of general functional disturbance; but of these, those affecting the heart attract particular attention.

In some instances the fear of a cardiac affection is not justified, for it is based upon the oversight of an anatomical peculiarity. In such cases the heart has developed rapidly at puberty, but the thorax has lagged behind, retaining its infantile characteristics. As a result the heart seems unduly large and the impulse unduly forcible. If with this there is anaemia, and possibly dyspepsia, functional bruits and irregularity may also be present. Such cases show no definite symptoms of cardiac disability, however, and in the course of a year or two the chest develops and the suspicion of cardiac disease vanishes.

There seems to be no convincing evidence that masturbation produces any particular form of cardiac disease or functional weakness, apart from that which is met with as a result of a general nervous instability. It frequently appears as an incident in the group of cases to be now considered, but takes no decided place in the causation.

This group is one of considerable uniformity, and is often associated with a history of overstrain after some infectious disease. The condition is one which is more apt to occur in the nervous, excitable child, but may also supervene in those of outstanding physical activity. Illnesses which precede the breakdown may be slight, as, for example, a sore throat of unknown origin; or more definite, such as a mild attack of influenza or diphtheria. As a rule, after this illness the health has not been quite satisfactory, although no particular symptom has been obvious. The physical overstrain may be sudden or gradual, and is not uncommonly the result of winter games which previously had been easily undertaken. Both boys and girls are liable. There is loss of nerve, undue tiredness after exertion, and unusual breathlessness. The digestion is disturbed, and constipation and flatulence are frequent. Some authorities lay much stress on the occurrence of dental caries as an added factor.

The general demeanour changes, and depression and pallor or sudden flushing are remarked, the face is drawn, and orthostatic albuminuria may be present. Giddiness, fainting attacks, and palpitation, are always alarming, and may reach a high degree of severity. Anaemia is frequent.

Physical examination of the pulse and heart show at once a change from the

normal. The pulse is rapid, sometimes reaching 100 or more to the minute; the wave is ill-sustained, and there may be much irregularity.

The heart is slightly, sometimes markedly dilated, and the impulse thumping but ineffectual. Upon auscultation, the first sound is short, and followed sometimes by a systolic murmur. At the pulmonary base there is generally a definite systolic murmur, which may vary with posture and respiration. Cardio-pulmonary murmurs may also be found to be present. Vaso-motor symptoms are frequent. Cold extremities and flushings in particular are noticeable. Exertion greatly increases the rapidity of the heart's action.

In this group there are different clinical types. If the child has been normally healthy before, the symptoms of nervous instability will be less, and the evidences of heart strain more evident in the dyspnoea and enlargement. On the other hand, if the child has always been nervous and excitable, the vaso-motor symptoms will be prominent.

In some cases the condition of the heart is suggestive of Graves's disease, although no other symptoms of this affection may be present; and it is quite possible that there may be some degree of hyperthyroidism.

Fainting attacks in early school and in morning chapel may cause much alarm about puberty, but in such cases the condition is almost invariably evidence, not of any serious cardiac disorder, but of reflex disturbance, due in some cases to the atmosphere, in others to a lack of food before the early school. Some have ascribed as a cause vicious habits in the early morning, but the writer has no evidence upon this point. Again, cyclical albuminuria is not uncommon in such cases.

There is another group of cases ascribed by Rekitansky and Virchow to a congenital hypoplasia of the aorta and its branches. In these the prominent feature is a persistent anemia rebellious to treatment. The patients are usually delicate in build and easily tired by exertion. Anomalies and retardation in the development of the sexual organs have been noticed in a considerable number. They suffer from palpitation, and their general circulation is poor. The left ventricle is hypertrophied. Such patients, if their strength is seriously overtaxed by physical overstrain, may develop acute heart failure. This condition should perhaps be more properly placed among the congenital affections, but the diagnosis of arterial hypoplasia is exceedingly difficult.

DIAGNOSIS.—The diagnosis is so intimately bound up with the symptomatology that no further comment is needed except to direct attention once more to the two conditions that usually come into the problem—viz., (1) congenital and (2) acquired heart disease—and to emphasize also the possibility of the conjunction of slight organic disease with well-marked functional disturbance.

PROGNOSIS.—When the diagnosis is assured, prognosis is good so far as the heart is concerned, although much time and care may be needed when there has been overstrain after an infectious disease.

TREATMENT.—From the first the child should be kept, if possible, in ignorance that the heart is at fault. The degree of folly that parents and relations reach in attempting to frighten a nervous boy about his heart is almost incredible.

In those cases in which there is no disorder of function the parents must be reassured, and probably no more will be needed than a general tonic or some digestive mixture. When dealing with the more definite cases, the general plan is one of a steady forward policy.

If these are symptoms of breathlessness and dilatation, and the child itself alarmed, absolute rest is the first step. Much attention must be paid to diet and digestion, and dental defects should be remedied. As soon as the signs of dilatation subside, steady forward progress should commence, for prolonged inertia is harmful to the great majority of these cases. Massage will be useful if there is restlessness and muscular flabbiness and loss of vasomotor tone. Judgment will be needed in separating the functional symptoms of a general nervous instability from those actually due to the heart. Irregularity in the rate and rhythm of the pulse must not, for example, carry undue weight, and is often enough no indication to stop progress. As a general statement, the sooner the majority of these patients are out of bed, the better; but there is no doubt that if there is too much haste, and a breakdown results, much ground will be lost.

One of the great practical difficulties in the highly excitable cases is to prevent advantage being taken of the slackening of restrictions; another is the complex nature of the disorder. Thus, the digestion may become upset, or the children may overtax themselves or suffer some nervous upset. Frequently a great advantage is gained if the patient can be enticed away from a bad mother.

Already an indication has been given of the various grades of exercises, and there are two great difficulties to be faced. In the mild cases the child or parents invariably want to continue the violent games undisturbed, and in the severe cases there is often a tendency to prevent any attempt at games. This question of violent exercise will largely depend upon the character of the breakdown. A strong boy who has been overstrained after an attack of diphtheria or influenza, for example, must be thoroughly tested by mild exercise first, and the wise plan is to err on the side of caution. The more neurotic cases in which no history of strain can be obtained often benefit by encouragement. For such a bracing climate, cold baths, and the spinal douche, are often most helpful when recovery has set in.

Medicines take a secondary place to general management. When dilatation is obvious, digitalis may be needed; when there is much nervousness, bromide and tincture of cortex; when dyspepsia, alkalies, aperients, and bitter tonics; when anaemia, iron. An occasional dose or repeated small doses of calomel is indicated when there are constipation and dyspepsia.

If the medical attendant has convinced himself that a child's heart is functionally damaged, he is wise to turn a deaf ear to the efforts of parents, patient, and masters, who would urge any risk to achieve some fleeting athletic success.

A few lines may be helpful upon *hyperthyroidism* produced in the partially convalescent. In these cases, if the treatment with thyroid extract is overpushed—and it must be remembered that only very small doses may be tolerated—there will arise tachycardia, with nervousness, sweating, pigmentation, and even slight proptosis. The remedy lies in stopping the treatment for awhile and substituting doses of the bromides. Later, resumption of the thyroid treatment should be undertaken, with minute doses. If the warnings are overlooked and the drugs persisted with, severe and even fatal diarrhoea may result.

MALIGNANT GROWTHS OF THE HEART AND PERICARDIUM.

This is a rare condition in childhood, and when it occurs is usually the result of mediastinal lympho-sarcoma. The most important lesion produced is pericarditis with effusion. The effusion is a result of metastatic deposits in the visceral or parietal pericardium.

The cases may commence in the most insidious manner, and attention be only directed to the child's health on account of shortness of breath. Examination may then reveal a large pericardial effusion, or a pleural effusion with or without the presence of pericardial friction. The temperature is usually raised, but wasting is not a feature in the earlier stages. Almost invariably either one pleura or the pericardium will require paracentesis, and a large quantity of fluid which may be blood-stained is withdrawn. This in itself raises suspicion, but is not conclusive, for in tuberculous pericarditis the same kind of fluid may be obtained. The tendency is for the fluid to re-accumulate, and there may be much distress from enlargement of the mediastinal glands, causing pressure upon the large veins.

The course of the disease is steadily progressive, and death may be sudden from pulmonary embolism or thrombosis, or from progressive heart failure.

The **DIAGNOSIS** is one of great importance on account of the hopeless nature of the malady. Pleural or pericardial effusions of mysterious and insidious onset are always suspicious events, and should at once arouse the fear that there is some unusual cause behind them. This suspicion is deepened by the presence of a blood-stained fluid, which should be carefully examined by culture and cytologically for cells pointing to malignant disease. Enlarged glands should be searched for above the clavicles and in the axilla, and any unusual dulness over the manubrium sterni or on either side of the vertebral column posteriorly, be noted. A radiogram may demonstrate the presence of enlarged glands within the chest.

TREATMENT.—It is in cases of this kind that the physician may be confronted with the puzzling combination of a large left pleural and pericardial effusion. If these are suspected, the method of procedure should be to deal with the lesser evil first, and aspirate the left pleura. In most cases relief will follow, and allow time to determine whether the condition is one of extreme pleural effusion coming forward in front of the pericardium or of the combined lesion. Further, when the pleural cavity is emptied, physical examination may disclose the presence of enlarged mediastinal glands and aid in the diagnosis. Should severe symptoms continue, and the signs point to a pericardial effusion also, this can be then dealt with more easily. There is a growing tendency to advise a deliberate opening of the pericardium in cases of effusion, but for these cases the writer is convinced that such a step is not always needed. Guided by the character of the pleural effusion, and choosing the spot advised by S. West—viz., the outer limit of the dulness on the left side beyond the impulse—the pericardial cavity can be sufficiently emptied by a simple paracentesis.

An X-ray examination will be of much assistance if it can be obtained, but if the dyspnoea is severe it may be advisable to relieve the pleural effusion first. No drugs, except for palliation of distress, have the least effect on the disease, and we must be prepared to find the glands enlarge with great rapidity in the virulent cases.

In the rare developmental diseases, tubercles, sarcomas, tumours are met with in the heart wall, which have been shown by Fowler and Carnegie Dickson to be rhabdomyomata.

The remarkable case of Armstrong, in which a lymphango-endothelioma of the auriculo-ventricular node was found after death, and in which the first clinical symptom of importance was an epileptiform attack, stands out as a unique example of a local malignant growth in the heart of a child.

HEART DISEASE THE RESULT OF CHRONIC PULMONARY DISEASE.

Although chronic pulmonary affections—such, for example, as asthma and repeated bronchitis—associated with more or less obstruction in the upper air-passages, are comparatively frequent in childhood, any striking evidence of cardiac failure as a result is rare. Irregularity of the action of the heart and dyspnoea upon exertion are common, but it is difficult to decide how much these are dependent upon the respiratory difficulties rather than upon a decided failure of the heart. Severe hooping-cough may produce a certain degree of heart strain and dilatation, and this may prove a distinct danger when the strength is undermined by repeated vomiting and inability to take food. Again, where there is grave deformity of the chest, and consequent diminution in the volume of the lungs, respiratory disease throws a great strain on the heart, and an acute exacerbation may terminate abruptly by a sudden syncope. In some exceptional instances the writer has seen repeated bronchitis and consequent emphysema, accompanied by such signs of cardiac weakness as dilatation of the right side, with enlargement of the liver, dropsy, and oedema of the bases of the lungs. The interpretation of these symptoms has been that they were the direct result of the respiratory disease, and post-mortem examination has shown that there was no valvular lesion. In such cases the heart has been found dilated, especially upon the right side, and the cardiac walls have shown degenerative changes fibroid in character. In some instances ante-mortem thrombosis in the right auricle or ventricle may occur.

There is another group of cases in which extreme cyanosis and grave dilatation of the heart may occur with unexpected severity as a result of an acute respiratory disease. The explanation of these has been the existence of some congenital defect in the heart which has been unsuspected. A patent foramen ovale or a congenital weakness and smallness of the entire heart may be the actual cause of this sudden heart failure.

The association of sudden heart failure with adenoids, a large thymus, and general lymphatism, has been already alluded to under Sudden Asystole, but these cases cannot be claimed as examples of heart failure resulting from respiratory embarrassment.

Under the section of Heart Disease in Diphtheria emphasis was laid upon the great frequency with which the ultimate cause of death has been paralysis of the diaphragm and massive pulmonary collapse; but in the vast majority of these cases the heart has been already weakened by diphtheritic paralysis.

Acute heart disease may be associated with such respiratory affections as erysipelas and pneumonia, but here the explanation usually lies in the occurrence of an invasion of the heart by the infective agent that has caused the respiratory lesion.

DIAGNOSIS.—Bearing in mind that in childhood the heart can almost always cope with the strain of respiratory affections, this must be made with caution, and some primary fault in the heart should be sought for, whether congenital or acquired.

When, however, there have been repeated attacks of bronchitis with consequent emphysema, and possibly, also, obstruction in the upper air-passages, and if in addition there is deformity of the chest wall and an absence of any clear cause for a primary cardiac lesion, we are led to the diagnosis by the occurrence of dilatation of the heart, particularly on the right side, enlargement of the liver, oedema of the feet, cyanosis and dyspnoea, synchronously with an exacerbation in the chronic respiratory affection. The diagnosis is clinched by relapses in the dilatation, occurring with renewed attacks of bronchitis.

PROGNOSIS.—This is grave, for it is evident that heart failure occurring at such an early age from such a cause must mean either that the respiratory affection is very advanced or the heart unusually feeble. Good recovery may ensue, but, unless the original cause can be largely prevented by some method of treatment, the liability to relapse and gradual systole is very great.

TREATMENT.—The dilatation of the heart must be treated on general lines. If there is severe asthma with the bronchitis, the paroxysms must be promptly dealt with and the bronchitis carefully treated. Digitalis and strychnine are both valuable heart tonics in such cases, and it is well to give small doses of digitalis for many weeks after the bronchitis has ceased and the heart recovered. If the heart failure is urgent, it must be relieved by leeches applied to the præcordia, and by suitable doses of calomel and salines. When the child is convalescent, steps must be taken to deal with the respiratory disorders by climatic, surgical, or medical measures.

THE HEART IN RENAL DISEASE.

Among the most important differences between the child and adult that are met with by the student of heart disease is the rarity in the former of severe cardiac affections in renal disease. In the child those chronic renal conditions which particularly damage the heart and arteries are rare. There is, however, no doubt that the heart suffers in some degree in many cases of nephritis, and two classes of lesion are to be distinguished: either the heart may be damaged by the same agent that injures the kidneys, or it may suffer as a consequence of a primary renal affection. Examples of the first group are afforded by rheumatic heart disease and rheumatic nephritis, or pneumococcal pericarditis and nephritis, and with this class we are not concerned here; for the lesions in the heart are independent of the renal affection.

In acute nephritis some dilatation of the heart occurs frequently, as is shown by the increased cardiac area, feeble first sound and impulse. Should the renal condition have been overlooked and the child have been untreated, a dangerous degree of dilatation may follow. When the renal disease becomes chronic, the blood-pressure rises, and the radial artery develops a certain tenderness of the vessel wall and incompressibility of the waves that are characteristic. With this there occur a prolongation of the first sound of the heart or a spacing of the sounds, and a clear ringing aortic second sound. The fluttering rhythm so often met with in the adult is much less frequent in the child.

In exceptional cases of small white or granular kidney there may be extreme alterations in the arteries and heart. In these the pulse is small, rapid, and incompressible. The retinal arteries may look wiry, and severe retinitis may be present. The præcordia is lifted by the heaving impulse of the powerful left ventricle, the cardiac sounds are prolonged and dull in tone, and the aortic second sound is ringing and much accentuated. The rhythm may be cantoring. Later in the illness dilatation may occur, with anasarca or cerebral hæmorrhage; pericarditis appears to be unusual. As a rule in these cases the cardiac symptoms are masked by uræmic manifestations, such as headaches, attacks of vomiting and diarrhœa, wasting, foul uræmic breath, epileptiform attacks, renal asthma, and fatal œdem. Death may be unexpectedly sudden.

DIAGNOSIS is dependent upon the detection of a primary renal disease.

PROGNOSIS is bound up with the renal disease. If the acute nephritis passes on to recovery, all cardiac signs will disappear; if it remains subacute, they will become obvious to physical examination, but as a rule give rise to no troublesome symptoms, and are no doubt in some degree a response to the necessary increase in the blood pressure. In the chronic cases where there is great hypertrophy the prognosis is the worst possible, for renal disease of this degree in early life is a hopeless condition.

TREATMENT.—In all cases in which there is evidence of acute dilatation complete rest is imperative, and every effort must be made to lessen the strain upon the heart. Later, when the nephritis is more under control, if the heart remains feeble, cautious doses of digitalis and sodium iodide may be used. In the chronic cases all attention is usually directed to relieving the uræmic symptoms. The nitrites and occasional doses of calomel are of value in assisting to relieve the high blood-pressure.

VASCULAR DISEASES.

Diseases of Arteries.

The rarity of extensive disease of the arteries in childhood is remarkable, when we recall how dominant a part infective processes play in the cardio-vascular diseases of this time of life. It is difficult to see why the arteries should not be severely damaged, and one is forced to the conclusion that wear and tear and high blood-pressure—factors conspicuously absent in the young—must be additional elements of supreme importance in the history of arterial disease. There is, however, a steadily increasing literature upon the occurrence of arterial disease in childhood.

Acute Arteritis.—This may be primary or secondary. Among the more important primary causes are congenital syphilis, rheumatism, typhoid fever, influenza, and pneumonia. Among the secondary causes, apart from injury, is embolism the result of a malignant endocarditis or of an ante-mortem thrombosis in the cardiac chambers, as in diphtheria.

The results of primary arteritis are various. The vessel may be inflamed, but the lumen not occluded; or there may be an obliterative process with consequent thrombosis; or, again, as in periaortitis nodosa, the formation of numerous aneurysms.

When the arteritis is secondary, there usually results occlusion of the vessel, and if the embolus is infective an aneurysm may develop, which sometimes reaches an enormous size. In other cases the occlusion is partial; in others, after a period of occlusion, the vessel may become permeable again, as occurs in some of the rheumatic examples.

SYMPTOMATOLOGY.—With some pyrexia there may be local pain and redness over the damaged vessel, spreading along its course; local thickening may be discovered, and the limb feels heavy and stiff, with subjective symptoms of tingling and numbness. The diagnosis, which is often uncertain, becomes clear when with these symptoms there is diminution in the pulsation of the vessel. When there is complete obliteration, there may be intense pain, with coldness and numbness of the extremity, and a dry or moist gangrene follow.

DIAGNOSIS.—The chief problem is to decide whether the occlusion is the result of a primary arteritis or an embolism. Much will depend upon the abruptness of the onset and the exact condition of the heart, taken into consideration with the cause of the illness. Acute arteritis of the smaller vessels is sometimes caused by an acute infection, such as, for example, broncho-pneumonia, associated with diphtheria, hoarse cough, measles, and influenza. Should this occur in cerebral vessels, a sudden monoplegia or hemiplegia may result.

Periarteritis Nodosa, a very rare disease under all circumstances, is occasionally met with in childhood. The cause of the disease is unknown. The lesions consist in the formation of small nodules, the results of inflammatory foci around the vasa vasorum upon the medium-sized and minute arterioles.

The symptoms are indefinite, and the diagnosis can be made only when subcutaneous nodules in association with the arterial system are discovered, and there coexist such symptoms as vague pains, muscular tenderness, feebleness, attacks of albuminuria or hematuria, syncope, tachycardia, dyspnea, and shivering. Blood-examination shows a leucocytosis. For further references to this disease, Kussmaul and Meier, Carnegie Dickson, and Longcope, may be consulted.

Chronic Arteritis, Arterio-Sclerosis, and Atheroma.—Various infections may produce the conditions, and in French literature much attention has been directed to rheumatism as a cause. It appears to the writer that in this country any degree of chronic arteritis or arterio-sclerosis is exceptional.

Congenital syphilis is undoubtedly an important cause of severe arterial disease of a chronic nature, and in this disease more than in any other the arterial lesions in childhood have been studied. In the new-born an acute arteritis may result with changes in the adventitia and media. In the adventitia the vasa vasorum are thickened, surrounded with perivascular exudation, and even obliterated. In the middle coat the muscular fibres are degenerated and infiltrated with inflammatory cells and permeated by newly-formed bloodvessels. Then, again, chronic arteritis may result which may be gummatous or sclerosing in type. Atherosclerotic patches may be formed in the deeper part of the inner tunic or even in the middle coat, and aneurysm may follow.

The writer has described two clinical types of this diffuse arterial disease which are of considerable practical importance. In the first there appear symptoms of an idiopathic epilepsy, followed some years later by jaundice, dropsy, and meningism. Post-mortem examinations show endarteritis obliterans of the renal, radial, coronary, and cerebral arterioles, with a chronic meningitis. In the second

group double hemiplegia is the feature, the second hemiplegia following the first at a variable interval extending from months to years. These vascular lesions are, however, of such varying degree and distribution that the symptoms are protean.

Arterio-sclerosis of high degree may be associated with rare cases of chronic interstitial nephritis in childhood, and the condition of the arteries is identical with that found in adults under similar circumstances. Thyroid insufficiency and excessive suprarenal activity are thought by some to be a cause of chronic arteritis.

Aneurysm.—Aneurysms in childhood may be connected with the aorta or with the peripheral vessels, and may be congenital or acquired. In both instances the event is a rare one. The causation is usually some infective process in the cardio-vascular system, or more rarely an injury.

Lebeuf in twenty-four cases found that nine were situated upon the intra-thoracic aorta. Hallé and Schreifer have described a very complete case of this kind in which the Wassermann reaction was negative, and the aneurysm confirmed by X-ray examination of the chest.

The peripheral arteries are particularly likely to be affected in malignant endocarditis of rheumatic origin. In this condition the embolus produces a secondary arteritis, which is not sufficiently acute to produce an immediate rupture of the vessel wall, but causes a local inflammation and weakening, with the result that a sacular aneurysm follows. Such aneurysms may reach a great size. Injuries such as a fall on the head sometimes damage the internal carotid as it enters the base of the skull, and an aneurysm follows. In two cases under the writer's observation death resulted from recurrent hæmorrhage from the mouth.

Aneurysm of the thoracic aorta gives rise to symptoms that are similar to those met with in adult life, but special emphasis must be laid upon attacks of dyspnoea resembling asthma and argual pains. The diagnosis from mediastinal tumours is difficult, and will need the assistance of radiography.

Diseases of the Veins.

Phlebitis and Thrombosis.—There are various causes in childhood which fall into two main groups—those the result of direct pressure, as from a mediastinal tumour or an extensive mediastinitis; and those which result from infective processes, which are often associated with general weakness, or even marasmus.

In the group that results from pressure, particular attention must be directed to the importance of tuberculosis of the bronchial glands and lymph-sarcoma. As a result of mediastinitis, thrombosis may occur in either the superior or inferior vena cava.

Venous thrombosis may occur in association with rheumatic heart disease, and whether or not the condition is one of primary phlebitis or primary thrombosis is difficult to determine. The French writers attach considerable importance to rheumatism as a cause of venous thrombosis in the lower extremities, but the writer's experience of six cases has been that the large veins of the neck and upper extremities are affected. In four of the six cases the condition was verified by necropsy. In two the superior vena cava was more or less occluded; in two the internal jugular, together with many other tributaries. In some cases of Raynaud's disease hyperplasia of the inner coat of the veins has been found in the situation of the lesions.

SYMPTOMATOLOGY.—During severe rheumatic heart disease the sudden appearance of oedema of the face or chest or of one arm should at once suggest the possibility of thrombosis. When the superior vena cava is blocked there is considerable mental obtusation, and the facial aspect resembles that in trunks.

The temperature usually rises, and enlargement of collateral veins, together with tenderness along the course of the affected ones and the discovery of firm cords upon palpation, are the important physical signs.

In severe cases there is danger of the process spreading into the right auricle, and of the occurrence of pulmonary embolism. There may, however, be recovery, with establishment of a collateral circulation.

There are other important causes of phlebitis and thrombosis, some of which may be suppurative. In infancy cerebral venous thrombosis and sinus thrombosis must be mentioned as complications of acute gastro-intestinal affections, middle-ear disease, typhoid fever, broncho-pneumonia, scarlet fever, and other infections.

In advanced tuberculosis with profound anaemia, phlegmasia alba dolens may occur. Lastly, Martin and Tull and Frey have recently directed attention to a chronic phlebitis ending in phlebosclerosis as a result of infective processes.

RAYNAUD'S DISEASE.

Raynaud's disease is not frequent in childhood, and fatal cases are rare. There is only one case in the post-mortem records of the Hospital for Sick Children, Great Ormond Street. The disease is, however, frequent enough to be well recognised, and occurs more often in females than in males. The children affected are not necessarily feeble, but may be apparently strong and well nourished. Friedel has described its occurrence in an infant of six weeks, and Beck records a case which was fatal in the seventh month. Colman's patient at Great Ormond Street was eight months old, but at this early age the disease is most exceptional. Raynaud in his original treatise mentions having seen five cases in childhood, and describes two of these. Cassirer, in an analysis of 168 cases, found 22 examples under five years, and 8 between five and ten years.

The CAUSATION is obscure, but there is no doubt that exposure to cold and sudden changes in temperature are frequent excitants of an attack in those who are subject to this affection. In some instances, particularly of the milder forms of the disease, there is found to be a family tendency. Congenital syphilis has also been claimed as a factor, and although Moussy's figures would point to it as exceptional, Cassirer quotes the following suggestive example of a child of two years with undoubted signs of congenital syphilis, who after exposure to cold had attacks of cyanosis of the hands, feet, and ears, with patches of local gangrene which abated under mercurial treatment. In some instances there has been a history of a previous infectious disease, while others remain without any apparent antecedent cause at all.

PATHOLOGY.—Morbid anatomy has shown in some cases no definite changes; in others neuritis has been found, but its meaning is uncertain. In Beck's recent case of an infant of seven months all the main arteries of the extremities showed patches of hyperplasia in the inner coat; in these there was unusual development of the elastic element, and the same condition was found in the veins of the upper

extremities. In Colman's case elevations suggesting an endarteritis extending from the tunica media into the intima were found in the iliac arteries, and a streptococcus was isolated from the heart's blood after death.

The explanation of the instability of the vasomotor centres is obscure, although it is clear that a remarkable local spasm of the bloodvessels results. The gangrene that may follow is doubtless the result of the block to the circulation, whether this be from the first complete, or, as is more usual, prolonged in duration, but not absolute in the earlier stages.

SYMPTOMATOLOGY.—The symptoms in childhood resemble those in later life, and all degrees of severity occur. Local syncope, local asphyxia, and local gangrene, may each be the predominant lesion, or two or more may be associated in the same case.

In the mild cases local syncope, characterized by the dead-white colour of the affected part, usually the fingers or hand, may be the only phenomenon. The part



FIG. 50.—RAYNAUD'S DISEASE IN CHILD OF EIGHT MONTHS, SHOWING GANGRENE OF THE LEFT FOOT. (COLMAN'S CASE.)

(From Photograph kindly lent by R. Wainwright.)

feels cold, and its temperature falls; with this there is a sensation of numbness or heaviness, or of pins and needles. In these mild cases the ischæmia seldom lasts long, and frequently is only a matter of a few minutes; but it may recur several times in the day. With the relaxation of the spasm the part becomes red, and may throb painfully.

In the more severe cases local asphyxia occurs, and this may or may not be preceded by local syncope. Should syncope precede, it may last for some hours; when asphyxia develops, the affected part becomes intensely painful, and is dark blue and deadly cold. In this condition it may remain for three or four hours. When the spasm relaxes, there supervenes a stage of active hyperæmia, with intense throbbing and burning, and sometimes almost unbearable pain. These attacks may recur day after day in a cycle, and then disappear, sometimes not to return; in other cases to reappear, particularly if there is undue exposure to cold or to abrupt changes of temperature.

The most severe cases are marked by the occurrence of local gangrene. As a rule this is preceded by a prolonged stage of asphyxia, but this is not invariable. The gangrene is usually only superficial; the affected part remains cold; blebs form on the surface, and are followed by excoriation or desquamation. In cases which are fortunately of great rarity a more severe degree of gangrene may occur. No more striking instances of these can be given than those of Coleman, Varlet and Tannabill. Coleman's case, a baby of eight months, showed first a partial cyanosis of the legs and feet and to a lesser degree of the hands and arms. Suddenly one day with evidences of acute pain there developed a purple patch on the inner side of the left calf. Later gangrene occurred, affecting the left foot and toes, and the right great toe, left thigh, nose and upper outer aspect of the left arm. Death took place four days after the first appearance of gangrene. Varlet's patient, who was a boy and a deaf-mute, was aged three and a half years. These developed, without any crises of syncope or asphyxia, gangrene of the tip of the nose, portions of the ears, both hands and the lower parts of the forearms, both ankles and feet. In Tannabill's case, a child of seven years, in addition to local asphyxia of the extremities and ears, with gangrene of the left foot, there developed areas of local asphyxia on the extensor aspect of the left forearm and the inner side of the right leg. In these severe cases a line of demarcation forms at the junction of the living and dead tissues.

As a rule, in Raynaud's disease the lesions are symmetrical, but this is not invariable.

Complications.—When attacks of local asphyxia recur frequently, the skin of the hands may become indurated and suggest scleroderma; the nails also are ribbed and discoloured. Coincident with the crises of asphyxia, the child may suffer from attacks of abdominal pain, which in some cases of Barlow's were referred to the epigastrium. Urinary changes may follow, and, as in adult life, hemoglobinuria and paroxysmal albuminuria, with a deposit of calcium oxalate, have been described. Eastace Smith states that there is no apparent relation between the attacks of pain and changes in the urine.

Coleen has observed the association of mitral disease, and Barlow has proved that where this association occurs, the local asphyxia is independent of embolic phenomena. Such cases are, however, unusual.

Seuther records the occurrence of maniacal attacks during the period of active hyperæmia, but it would appear that cerebral manifestations, epileptic attacks, and arthritic complications, are more likely to occur in the adult cases. The writer is not aware that spasm of the retinal arteries, described by Raynaud and Panas, have been observed in childhood.

Differential diagnosis.—The diagnosis is as a rule easy, on account of the symmetry and paroxysmal nature of the attacks.

Chilblains, although recurrent, cannot be termed "paroxysmal" in their course, and are, as is well known, generally situated some short distance from the tip of the extremity. There is also as a rule some distinct evidence of inflammatory oxidation.

Congenital heart disease often gives rise to a general cyanosis, which, though it may be intermittent, is not paroxysmal or confined to the extremities. Frequently there is also clubbing of the digits.

The writer has seen extreme and remarkable cyanosis of the extremities in cretinous infants, but, again, this is not paroxysmal, and these are the additional signs of cretinism. Lastly, this symptom actively disappears under thyroid treatment.

Erbell's gangrene, and even gangrene from failure of the circulation in the extremities, sometimes occurs in lingering cases of rictal disease, but the distinction is usually easy and of no practical importance. Gangrene may occur after acute infections or in extreme cachexia, but the history of the infection and the distribution of the lesions very seldom allows of a mistake in diagnosis. Of gangrene in malarial and chronic ergotism in childhood the writer has no knowledge, but the diagnosis in this country is not of practical moment.

When the diagnosis has been made, it is advisable, where there is any suspicion of a syphilitic taint, to submit the blood to Wassermann's test.

PROGNOSIS.—The outlook in the majority of cases appears to be good. As a rule the symptoms are of a mild or moderate degree of severity, and after a while disappear with the assistance of treatment. Fatal cases are recorded in which gangrene has occurred, but they are exceptional. It is nevertheless difficult to forecast how long the symptoms in any particular case may last, on account of the tendency to recurrence.

TREATMENT.—The general health should be carefully treated. If there is clear evidence of congenital syphilis, mercurial treatment is indicated. If cold and damp are obviously the important excitants, every care should be taken, by the use of pure woollen garments next to the skin, and thorough protection of the extremities, to avoid changes of temperature; wintering in a warm climate is indicated where possible. Washing in cold water is to be forbidden. In the mild cases massage, hydrotherapy, and electricity, are indicated. The local application of radiant heat, followed by skilful massage, may be employed. High frequency and diathermy for the older children, if given by skilful hands, are also indicated. Galvanism given as advocated by Barlow, in the form of an electric bath, has given good results in the milder forms of local atrophy. In the worst cases morphia may be needed to relieve the pain. Harvey Oshing has suggested for very obstinate and recurring cases the use of Esmarch's bandage. It is to be feared that children will hardly be able to bear the immediate pain that results. Local treatment of the gangrenous part is conducted on the usual antiseptic lines. Cod-liver-oil, iron, and arsenic, have all been advocated, but have no special action upon the chief symptoms.

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CHAPTER IX

DISEASES OF THE HEMOPOIETIC AND LYMPHATIC SYSTEMS

II. THURSFIELD

CONSTITUENTS OF THE BLOOD IN INFANCY AND CHILDHOOD.	ENLARGEMENT OF THE SPLEEN.
SECONDARY ANEMIA.	PERIPNEURIA.
PRIMARY ANEMIA.	HEMOPNEURIA.
LEUKEMIA:	LYMPHEDEMA.
ACUTE (PEYER)-LEUKEMIA IN- FANTUM (VON JAKSCH).	LYMPHADENOMA.
POLYCYTHEMIA.	AFFECTIONS OF THE LYMPHATIC VESSELS AND GLANDS.

Introductory.—In order to understand the clinical phenomena met with in the blood-diseases of childhood, it is necessary to recapitulate briefly what is known of the histology of the blood in normal conditions.

CHARACTERS OF THE BLOOD IN INFANCY.—The new-born child possesses a number of red blood-corpuscles considerably in excess of that found normally in the adult. In the first day or two of life the average number is nearer 6,000,000 than 5,000,000, and in exceptional cases may be as high as 7,000,000. Various theories have been propounded to account for this excess, but none is entirely satisfactory. Perhaps the chief factor is an increased activity of the blood-forming organs to meet the changed conditions of extra-uterine life—a supposition which finds support in the considerable number of nucleated erythrocytes which are present.

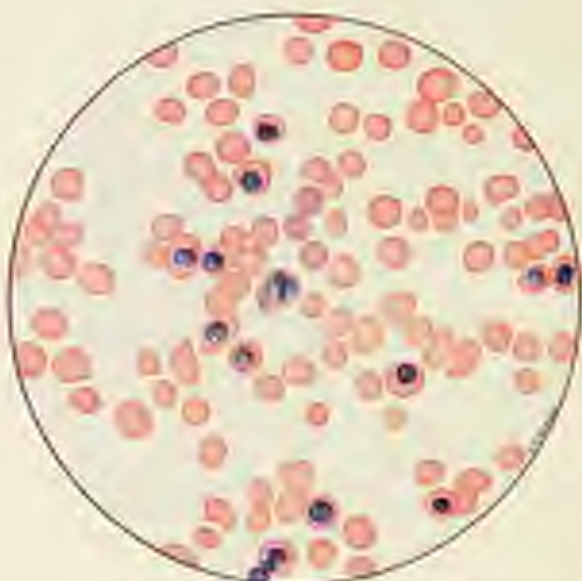
With this excess of red blood-corpuscles there is a corresponding *excess* of hæmoglobin, the percentage rising to 110 to 120.

The total numbers of the white corpuscles are also considerably in excess of the normal adult figure; the average of many observers is between 20,000 and 30,000, and of these at least 70 per cent. are polymorphonuclear cells.

These marked differences do not, however, persist. The number of the red corpuscles sinks rapidly in the first week of life, and by the second week attains the normal figure of 5,000,000 per cubic millimetre, which remains remarkably constant during the whole of life. At the same time the nucleated red corpuscles become much less numerous, and after the first week in the blood of a healthy infant are rarely found.

The hæmoglobin in the same period sinks steadily, but to a lower level, finally reaching the percentage of 70 to 75 some time before six months of age. At this figure it remains during the whole of infancy, and does not as a rule increase again until the end of the second year.

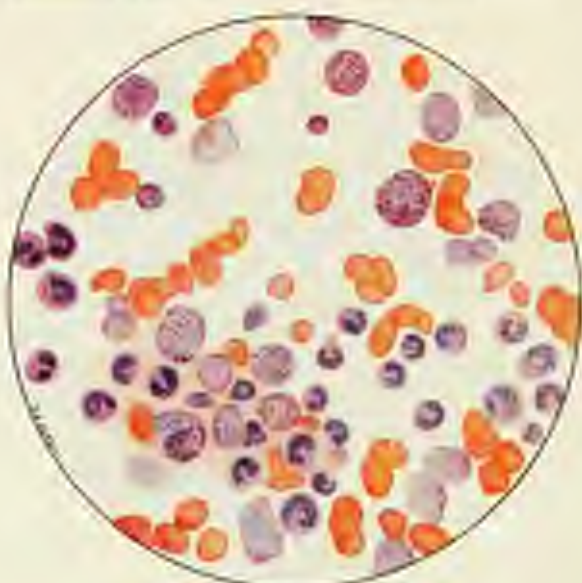
The white corpuscles also undergo a diminution, but very slowly. There is a marked fall in the first few days; then for the first year the number remains about



NUMEROUS RED CORPUSCLES AND LEUKOCYTES
FROM A CHILD (AGE 5 DAYS)

LEUKEMIC STAIN

MAGNIFICATION 2000 TIMES



MIXED LEUKOCYTES

FROM A BOY (AGE 11 YEARS)

LEUKEMIC STAIN

MAGNIFICATION 2000 TIMES

15,000 per cubic millimetre, and in the second, third, and fourth years lies between 15,000 and 10,000 per cubic millimetre. The average number found in the adult—8,000 per cubic millimetre—is not reached until the time of the second dentition. This constant excess above the average adult standard is due entirely to the large number of lymphocytes present in the blood of the child. While the total number of polymorphonuclear cells, which is from 18,000 to 25,000 per cubic millimetre at birth, quickly reaches the normal adult level of 5,000 to 6,000, the total number of lymphocytes, which at birth is from 3,000 to 5,000 per cubic millimetre, rises during the first few weeks of life from 7,000 to 9,000, between which figures it remains during the first and second years, gradually sinking to the normal adult level of 1,600 to 2,400 by the date of the second dentition.

The causes of these physiological alterations in the blood of children are obscure, but they represent activities which we know from pathological manifestations to be exceedingly liable to disturbance.

Turning from the consideration of total numbers to the characters of individual cells, it must be noted that the erythrocytes do not exhibit the uniformity of size which is usual in the adult; both small and large corpuscles are frequent, and even in health there are corpuscles seen of abnormal shape (poikilocytosis); further, that the reaction of the corpuscles to staining reagents is not uniform, but that a certain number will be seen stained with tints differing from the standard (polychromatophilia). The nucleated red corpuscles are for the most part normoblasts—*i.e.* a red corpuscle of the average size, with a round, deeply staining nucleus. Megablasts are found chiefly in pathological conditions.

Of the leucocytes it is usual to recognize five varieties, which differ from one another in the shape of their nucleus and the presence or absence of granules in the cell protoplasm.

1. The polymorphonuclear cell has a nucleus of twisted or divided shape, and possesses neutrophil granules in abundance.

2. The lymphocyte is a cell of somewhat varying size in which is a round nucleus, centrally placed, and an absence of neutrophil granules in the cell protoplasm.

3. The large mononuclear cell, or hyaline cell, is somewhat larger than the lymphocyte; it possesses an oval or kidney-shaped nucleus, which is usually marginal in position, with the hilum towards the bulk of the protoplasm; there are no neutrophil granules.

4. The eosinophil cell has a nucleus which is usually divided and irregular, with deeply staining coarse eosinophil granules dotting the protoplasm of the cell.

5. The basophil cell has a divided or plume-shaped nucleus with a few granules in the protoplasm, which stain a purple colour with the eosin-blue stains.

Besides these forms, which are readily recognizable, there are cells which have been described as "transitional," which resemble the large, mononuclear variety, except that their nucleus is very irregular instead of round or oval; and lastly the myelocyte, which is occasionally seen in the blood of healthy infants, but is never found in any large number except in pathological conditions. It is a round cell, usually, but not necessarily, larger than the polymorphonuclear or large mononuclear cell, with a round or oval or kidney-shaped nucleus, usually marginal, and an abundance of fine neutrophil granules.

These various forms are identical in the bloods of the adult and the child; the only variation is that the lymphocyte of the child is often somewhat larger than that of the adult.

The characters of the blood of the child with regard to coagulability and to vulnerability do not differ, so far as is certainly known, from those found in the adult; but there is much clinical evidence to show that the red corpuscles are more easily destroyed in childhood, or, at any rate, that the destruction takes place more rapidly.

PATHOLOGY.—The pathological changes which take place in the blood as the result of disease may be summarized as follows:

1. Decrease of hæmoglobin.
2. Increase of hæmoglobin is polycythæmia.
3. Decrease of the numbers of red corpuscles—all anæmias.
4. Increase of the numbers of red corpuscles—polycythæmia.
5. Decrease of the numbers of white corpuscles—pernicious anæmia and certain infections.
6. Increase of the numbers of white corpuscles—leucocytosis; leucæmia.
7. Decrease of coagulability.
8. Abnormal fragility of the red corpuscles.

Most of these conditions will be noted in their places, and it will be sufficient here to remark that an increase in the number of red corpuscles occurs in certain diseases which are not essentially blood-diseases—e.g., in congenital pulmonary stenosis, in which counts of 9,000,000 and 10,000,000 per cubic millimetre have been recorded; and in diseases which have been accompanied by a rapid loss of fluid from the body; in acute diarrhoea the writer has made counts many times of more than 6,000,000 per cubic millimetre. With regard to decrease of the numbers of leucocytes, it is a valuable indication in typhoid fever, but otherwise is a rare phenomenon.

LEUCOCYTOSIS as commonly employed means a polymorphonuclear increase, and if used to denote increase of the other varieties has a qualifying epithet prefixed. Polymorphonuclear leucocytosis is most marked in the suppurations and in the majority of the infective fevers. Lymphocytic leucocytosis is marked in the earlier stages of hooping-cough, and is often declared to exist where a knowledge of the normal figures in infancy would have prevented the mistake. Eosinophilic leucocytosis is most marked in cases of infection with the parasitic intestinal worms, and in rare cases of chronic bullous infections of the skin. Leucocytosis of the large mononuclear cells or of the basophil cells does sometimes occur, but the clinical significance is unknown.

The importance of determining with accuracy the degree of coagulability of the blood is obvious; but it cannot be said that as yet the efforts of pathologists to invent a method of investigation which will give consistent and trustworthy results have been fully successful. Certain facts are known in this respect, but there is considerable difficulty in assigning their proper importance to the observations, and still more in comparing the observations on patients with widely differing diseases.

Classification of Blood-Diseases.—The classification of the various forms of blood-disease met with in childhood is even more difficult than that of similar diseases in the adult; and the difficulty is increased by the absence of agreement among writers as to the position of certain well-marked types. The classification which is used here divides blood-diseases into five groups: The first includes all those diseases in which anæmia appears to be obviously secondary to disease elsewhere

in the body; the second, those cases in which the blood-condition appears to be the essence of the disease; the third, those diseases in which the alteration of the blood, while a prominent feature in the disease, is yet not the dominating factor. The fourth group is that where the total numbers of the corpuscles are increased. The fifth group includes those diseases in which the alteration of the blood makes itself manifest by hæmorrhages, but in which our present methods show little or no alteration; it is the group of hæmorrhagic diseases.

This classification is, like all others, necessarily imperfect, in the absence of knowledge of causes, but it represents the present conceptions of the relation of these diseases to each other and to general disease.

CLASSIFICATION.—1. *Secondary anæmia* :

- (a) Following hæmorrhage.
- (b) Due to the absorption of poisons, whether mineral, chemical, or bacteriological.
- (c) Following defective feeding.
- (d) Anæmias of the school age.

2. *Anæmias in which the alteration of the blood appears to be the essential factor in the disease* :

- (a) Congenital anæmia.
- (b) Chlorosis.
- (c) Pernicious anæmia.

3. *Diseases in which the alteration of the blood appears to be an essential feature, but not the dominating factor* :

- (a) Myelocytic leukaemia.
- (b) Lymphocytic leukaemia.
- (c) Atypical leukaemia.
- (d) Chloroma.
- (e) Anæmia pseudo-leukaemia infantum.

4. *Polycythæmia*.

5. *Diseases in which the alteration of the blood, manifested by hæmorrhages, is not recognizable by our present methods* :

- (a) Purpura.
- (b) Hæmophilia.
- (c) Scurvy (see p. 124).

I. SECONDARY ANÆMIAS.

The alterations recognizable in the blood in this class include a diminution in the hæmoglobin content, a diminution of variable extent in the number of the red corpuscles, and sometimes a slight increase in the polymorphonuclear cells. In the most severe cases nucleated erythrocytes, both normoblasts and megaloblasts, are seen.

(a) The most obvious cause of a *secondary anæmia* is the occurrence of *hæmorrhage*. This may take place from the umbilicus, from a wound, or in the course of one of the hæmorrhagic diseases. The anæmia so induced varies in degree with the severity of the loss; but even in the most serious cases, in which the patient is

pale and pulseless, recovery is as a rule rapid and complete. In the case of severe hæmorrhage the actual loss of red corpuscles is the most marked feature, and the rapidity with which these are restored is remarkable. In the less severe cases the actual numbers of red corpuscles are but very slightly diminished, while the loss of hæmoglobin is proportionately more severe and much more slowly repaired. In the majority of instances one or two weeks will restore the patient to a normal condition as regards the corpuscles, but the hæmoglobin does not reach its former level for at least a month. In a few of the most severe cases the power of repair seems to be exhausted, and the anemia is prolonged for several months, or may lead to death from exhaustion. Treatment of such anæmias is simple. After the hæmorrhage has been controlled, subcutaneous injections of normal saline solution must be given, with the patient kept absolutely at rest. Drug treatment is not essential, since the loss of blood in itself acts as a stimulation to the blood-forming organs; but in the case of milk-fed infants, since milk is poor in iron, it is wise to give small doses—e.g., *ferr. sedacti*, 1 grain.

(b) Anæmia due to the absorption of poison, whether mineral, chemical, or bacteriological. Mineral poisoning by lead, arsenic, or other inorganic metals, is rare in children; occasionally arsenical poisoning may occur as a result of the medical administration of the drug, but in such cases the anæmia is seldom a marked feature of the affection.

The more important group in this class is that which arises from the chemical or bacteriological toxins which circulate in the body as a result of infection. The type is best studied in such a disease as diphtheria, but includes all the acute infectious fevers, such chronic diseases as syphilis and malaria, and infections with the parasitic worms. Of course, in many of these the anæmia is not purely toxic; in some defective nutrition plays an important part; in others loss of blood directly. In the case of rickets it is doubtful whether there is a toxæmia, or whether the anæmia is due merely to malnutrition. But in the majority of acute infectious fevers anæmia appears so suddenly, or is so prominent a symptom, that it is impossible to doubt that a toxin with direct effects upon the blood is present. In a moderately severe case of diphtheria, for instance, so early as the third or fourth day of the disease an examination of the blood shows a marked diminution in the amount of hæmoglobin and in the number of the red corpuscles. The same is true, sometimes even to a more marked degree, in cases of acute rheumatism and in the various types of pneumococcus infection. Acute nephritis is also often accompanied by a marked anæmia, but in this case the general oedema of the body tissues, including the blood, produces an effect which is probably more apparent than real; in cases where the oedema is trifling the anæmia is seldom marked.

In the more chronic intoxications it is difficult to separate the various factors in the production of anæmia. In congenital syphilis there is often a considerable degree of anæmia, principally shown in the diminution of the red corpuscles, but in many cases there is little or no alteration from the average. In chronic tuberculosis, again, the anæmia seems to depend far more upon the complications or accidental accompanying conditions than upon the infection; indeed, it is doubtful whether uncomplicated tuberculosis produces any alteration in the blood. Again, in the chronic intestinal diseases the anæmia seems to depend more upon the malnutrition than upon any toxin manufactured in the course of the disease.

Constipation is not *per se* a cause of anæmia, though it is permissible to believe that chronic constipation may indirectly contribute to the anæmic condition by

the general ill-health which often accompanies it. There is no available record of the occurrence of methæmoglobinæmia or sulphæmoglobinæmia in a child.

Intestinal parasites produce anemia in at least two different ways: they may do so, as in the case of the *Anchylostomum* by the hæmorrhage from the mucous membranes which they excite, or by the general condition of ill-health which accompanies their presence; whether they manufacture a toxin which is absorbed and directly affects the patient is doubtful.

(c) *Anemias due to defective nutrition* are a large and varied group, though it is impossible in the majority of instances to separate this factor in any individual group. The anemia of rickets is to some extent certainly due to defective nutrition, and yet in some cases of obvious rickets the anemia is of very slight extent; in another group there are children who have been nourished too long on feeds—milk and starchy foods—which are deficient in iron; and in another group, again, the children who, from one or another cause, never procure sufficient food to supply the necessary output of energy in their growth and development. Lastly there is a group in which chronic affections of the intestinal tract produce a constant drain upon the nutrition of the child, leading to anemias more or less prolonged and severe. It must be noted here, also, that the acute diarrheal infections of young infants frequently, in addition to the blood-destruction which accompanies them, leave behind them a legacy of anemia, which may be prolonged for some weeks or months.

(d) *The anemias of children no longer infants*, who have reached school age, belong generally to one or other of the foregoing groups, but other factors also come into play, especially in the case of delicate children—what may, for want of a better name, be called the "nervous" factor. The instability of the nervous system is a condition which is in many instances congenital, or at least acquired very early in life, and its influence on the general health is exaggerated when school life is begun. Even in infancy babies differ widely in their outlook upon external phenomena, and what may not disturb one child will produce excitement, trembling, and general disturbance, in another. At the school age such children are apt to feel the necessary confinement and discipline far more acutely than the average child, and slight defects in their surroundings, such as insufficient ventilation or overheating of the classroom, which leave others unaffected, tend in their case to produce general ill-health. Sleeplessness also may be added to their other ills, with night terrors, and in some cases somnambulism. Incapacity to digest ordinary food or refusal of food is another link in the chain, which in nearly all cases leads sooner or later to a considerable degree of anemia. Of the more purely physical causes of anemia at school age, there is probably none so important as dental caries, with the chronic infection of the lymphadenoid tissue which always accompanies it. This factor, however, is now much better recognized, and with children of the wealthier classes seldom comes into play. Overpressure of school work is not a common cause, except in the case of nervous children, who may require relief from school work for some prolonged period.

CHARACTERS OF THE BLOOD-CHANGES.—In all the anemias which we have been considering the alterations in the blood are practically identical. The hæmoglobin nearly always suffers considerably, and, instead of the 70 to 75 per cent., which is the normal figure in infancy and childhood, may sink to 50 or 40 per cent.* The red corpuscles vary much in their numbers, according to the efficient cause of the anemia. Where this is due to extensive or repeated hæmorrhage, the total number

often sinks to as little as 2,000,000, or even less, but in the majority of instances the destruction of red blood-corpuscles is not marked, and the colour index sinks to 0.6 or 0.5. Thus, the anemia, so far as the relation of the erythrocytes and the hemoglobin is concerned, is of the type met with in chlorosis—a disease which in reality does not exist in children of the age below that of puberty. Changes of shape and alterations in the staining reactions are uncommon in the secondary anemias, but in the severest cases occur to a mild extent, and the same is true of nucleated red corpuscles. An increase in the total number of leucocytes is met with in the stage of reaction following hemorrhage—an increase due even in infancy to the greater number of polymorphonuclear cells. This leucocytosis quickly passes away, and in the majority of secondary anemias the number of white corpuscles does not exceed the normal. In the case of infections with the intestinal worms there is often, but not invariably, a marked increase in the numbers of eosinophil cells, which may reach as high as 2,000 per cubic millimetre. This eosinophilia is most marked in *necylasteriasis* and *trichiniasis*, but has been met with in cases where the only infection was the common roundworm or threadworm. In these, however, it is uncommon, and so, also, in the instances of tapeworm infection which the writer has seen.

In some cases of rickets there is an increase in the numbers of lymphocytes, but this is by no means a constant feature, and in the majority of instances there is no increase in the numbers of the white corpuscles in this disease, unless there is some complication.

SUMMARY.—Secondary anemia is characterized by a moderate count of red corpuscles, a low hemoglobin percentage, a low colour index, the absence of poikilocytosis and polychromatophilia, and the absence, except immediately after hemorrhage, of a leucocytosis.

TREATMENT.—The treatment of all secondary anemias is to a large extent the treatment appropriate to the cause, and where that is obvious the treatment is plain. In many cases, however, the cause is obscure, and it is necessary to proceed with treatment on the lines already indicated in the preceding paragraphs. In the case of anemia following acute infections, the treatment appropriate to convalescence—rest, graduated exercise, fresh air, simple and abundant food—is generally sufficient to restore the blood to a normal condition. Whether iron administered as a drug really helps the work of repair is doubtful, but there can be no possible objection to prescribing it. The *syrup. ferri phosphat. co.* and the *syrup. ferri iodidi* are both as a rule easily tolerated; and other useful preparations are *tartarate of iron and reduced iron*. *Fellows' syrup* contains hypophosphite of iron. The various preparations which contain iron in an organic form have not, on the whole, anything to recommend them above these forms.

Arsenic also is a drug which occasionally appears to help in the restoration of health in the secondary anemias. It is best given in the form of *liq. arsenicalis*, $\text{℥} \frac{1}{2}$, or as a pill in the form of *sodii arsenat.* gr. $\frac{1}{2}$ – $\frac{1}{4}$.

Of bone marrow and hove marrow preparations, all that can be said is that their use is disappointing; and this is equally true of the various forms in which hemoglobin is offered to a credulous public. At the same time these preparations are for the most part entirely innocuous, a merit not always to be found in patent medicines. On the whole, the medical practitioner will do best to direct all his energies to the hygienic, physical, and nutritional treatment of his patients, supplementing these by the simplest and most easily tolerated forms of iron and arsenic.

II. ANÆMIAS IN WHICH THE ALTERATION OF THE BLOOD APPEARS TO BE THE ESSENTIAL FACTOR IN THE DISEASE.

(a) **Congenital Abnormalities** of the blood might reasonably be expected, just as congenital malformations occur in other tissues of the body; but the writer has been unable to find any cases recorded in which this error of development has been noted. In cases of congenital heart disease with cyanosis, the individual elements of the blood are of normal shape, and the leucocytes present in their usual proportions.

From time to time cases are met with in which the anæmia dates from birth, but in those cases where an accurate blood-count has been made the picture is always that of a secondary anæmia, and the children usually recover quickly with the administration of iron.

The same is true of the blood-counts which have been made in cases of congenital acholuric jaundice; but in this case investigations seem to show that the blood-corpuscles themselves are abnormally fragile, and give up their hæmoglobin much more easily than the blood-corpuscles of normal individuals.

(b) **Chlorosis** is an anæmia which is almost confined to the female sex, and to the period after puberty. Its cause is unknown.

The alterations of the blood are, a diminution in the quantity of hæmoglobin, out of all proportion to the diminution in the numbers of red corpuscles; consequently a low colour index, with a normal number of leucocytes. Such a blood-picture is common enough in children—for instance, in the secondary anæmias of school age; but the characteristic greenish complexion, the cardio-vascular changes, and the extraordinary therapeutic effect of iron, are features of chlorosis which are not present in children. Hence, though it is permissible to speak of anæmias of the chlorotic type as occurring in children, chlorosis in the true sense is unknown.

(c) **Pernicious Anæmia** is a name which has had many connotations. Here it is used to signify a chronic disease of unknown origin, characterized by a progressive destruction of the red cells and a degree of anæmia such as is rarely met with in any other affection. Such a disease is, whatever the reason, exceptionally rare among children in the first decade of life. Hutchison in 1904 was able to collect only eleven cases in the literature, and of these six were doubtful. The truth is that typical pernicious anæmia as we know it in the adult does not occur in children, and that the cases described as pernicious anæmia are almost without exception atypical in their course, in the changes in the blood, and in the post-mortem appearances.

Of the ætiology of the disease as it appears in children nothing is known.

SYMPTOMATOLOGY.—The chief symptom is the exceeding pallor of the skin and mucous membranes, which often have a slightly yellow tint. The pallor is so extreme that the lips often show scarcely more than a tinge of pink. With this profound anæmia objective symptoms of lassitude, weakness, loss of appetite, headache, and irritability, are common. (Edema of the ankles, and also of other parts of the body, is usually present, but seldom in a marked degree. The general condition of nutrition is not often much impaired. There is a considerable amount

of subcutaneous fat, and there is comparatively little muscular wasting, though the muscles are always flabby and soft. Hemorrhage of any extent is uncommon, but subcutaneous petechiæ, bleeding into the retina, and epistaxis, occur.

Fever is usually present, of an irregular character, and seldom high. The heart is dilated and the rhythm irregular. With this there is often a systolic murmur to be heard at the base of the heart, and also in the vessels of the neck. The pulse is feeble, not usually much above the average in frequency. The blood-pressure is low. The liver is not usually obviously enlarged, but the spleen in children almost always undergoes a considerable degree of hypertrophy, and can be felt an inch or more below the costal margin.

Diarrhœa is present at some time in the disease, and at times there is blood in small amount in the motions. The urine, which is often described as dark with urobilin, is more usually pale, and may contain a small amount of albumin.

The blood is sometimes watery, with diminished specific gravity, but in some instances, owing to the high ratio of hæmoglobin, appears to be well coloured. The hæmoglobin may be diminished to as little as 10 per cent. of the normal, and the red corpuscles to 1,000,000 or under, while the leucocytes are always markedly diminished. The colour index is at or near 1, not, as in other anæmias, markedly below this figure. In the stained films the red corpuscles are found to have irregular shapes, and to take up the stains in irregular fashion, the majority, however, staining well. Nucleated red corpuscles, both normoblasts and megaloblasts, are present, and the latter are one of the chief features of the disease.

No case should be classed as one of pernicious anæmia unless it has three features:

1. A progressive and severe loss of red corpuscles, with the presence of nucleated erythrocytes, especially megaloblasts.
2. A high colour index.
3. A low leucocyte count.

With regard to the first of these, it must be remembered that in adults remissions are not uncommon, during which the red corpuscle count may become nearly normal.

PATHOLOGY.—The chief features found at the autopsy are the extraordinary bloodlessness of all the organs; the high degree of fatty degeneration of the liver and heart; hæmorrhages in the viscera and beneath the serous membranes; and the deposit of free iron in the liver, spleen, kidneys, intestinal mucous membrane, bone marrow, and heart muscle. This deposit of iron, demonstrated by the Prussian blue reaction, is not peculiar to pernicious anæmia, but is exceptionally found in other forms of anæmia. It has been seen, for instance, in a case of anæmia pseudo-leukæmica infantum. The transformation of the yellow bone marrow into red, which is so characteristic in the adult, fails in the infant, whose marrow is normally red.

The **DIAGNOSIS** of pernicious anæmia should, in the light of its great rarity, be made with the utmost reluctance in the case of a child. The majority of cases described as examples of the disease are undoubtedly cases of severe secondary anæmia, and careful examination of the blood will usually enable this fact to be recognized. The early diseases in which the alterations in the blood in the least resemble those of pernicious anæmia are those produced by infection with some of the parasitic tapeworms. Several cases of infection with *Bothriocephalus latus* have been described in children by foreign observers. With these exceptions, the

blood-condition of pernicious anaemia is in children unique, although in adults there occur from time to time cases—e.g., of gastric carcinoma—in which the diagnosis is difficult or impossible.

PROGNOSIS.—In all the recorded cases of good authenticity death has occurred within a year from the first observation of the illness.

TREATMENT has always proved unavailing. The drug most in favour is arsenic, which can be given in increasing doses by the mouth, or may be given in the form of one of the preparations for intramuscular injections. Oxygen inhalations, bone marrow, and transfusion of blood, seem to be quite useless.

III. DISEASES IN WHICH THE ALTERATION OF THE BLOOD APPEARS TO BE AN ESSENTIAL FEATURE, BUT NOT THE DOMINATING FACTOR.

Leukæmia.—**DEFINITION.**—A disease characterized by enormous increase of one or more of the leucocytic elements of the blood.

INTRODUCTION.—The disease was discovered almost simultaneously in 1841 by Virchow and Bennett, and the two main types sharply differentiated by Ehrlich in 1883. In the last decade an enormous amount of literature has accumulated regarding the pathology of the affection and of allied conditions, and the general tendency has been towards unification. Neumann in 1878 had already shown that in all cases of leukæmia the bone marrow was affected, whatever the type of the disease, and held that the affection of the marrow was the essential feature. Ehrlich and his pupils, basing their view on the brilliant results of the staining reactions devised by them, believed that the two main types were differentiated by their origin in the bone marrow and in the lymphoid tissue of the body respectively. This conception of the nature of the disease is undoubtedly supported by the examination of the blood in typical cases, and it cannot be denied that the course, duration, and blood-picture of the two types, myeloid and lymphoid, are sharply differentiated. But further examination, particularly of the bone marrow in cases of lymphocytic leukæmia, tends to show that without exception this tissue is extensively affected, and that there is no reason to suppose that this is a change secondary to the hyperplasia of the glands; further, that the atypical cases of leukæmia—i.e., those in which the alterations of the blood depart in some way from the expected type—differ in no respect in their broader anatomical lesions from the typical leukæmias. Thus the modern theory is slowly shaping itself again to Neumann's conception of the disease: that it is essentially a disease of the whole hæmopoietic system, marrow, spleen, and lymph-glands, and that in every case of leukæmia all these are involved, though with differing degrees of intensity. If this theory be accepted, the so-called atypical and "mixed" leukæmias fall naturally into place as mere varieties of disease of the hæmopoietic system; while in Ehrlich's conception they constitute a very distinct difficulty.

THE RELATIONSHIP OF LEUKÆMIA TO ALLIED CONDITIONS.—The difficulties in this part of the subject relate—(1) To cases in which there are blood-changes characteristic of leukæmia, with glandular swellings which behave like new growths, giving rise to metastases. The most striking of these is the disease called

"chloroma" (p. 526). (2) To cases in which, with the symptoms, course, and post-mortem findings, of a typical leukemia, the blood never shows the characteristic increase in leucocytes. (3) To cases in which the blood-picture combines some of the features of a leukemia and a pernicious anemia—so-called "leukæmia."

1. Sarcoma-like growths of the hæmopoietic system assume two main types: (a) Chloroma; (b) growths apparently originating in the thymus gland, or in the lymphoid tissue in other parts of the body, which behave like sarcomata, in bursting through the capsule and infiltrating neighbouring tissues. In some of these the changes found, at the autopsy, in the bone marrow were identical with those of an acute lymphocytic leukemia, and both in these cases and in the reported cases of chloroma the changes in the blood were exactly similar.

2. Cases have been reported in small number in which the symptoms, course, and duration resemble those of a chronic leukemia, and in which changes in the bone marrow and other tissues of the hæmopoietic system have been found resembling those of leukemia, and yet the blood does not exhibit any characteristic change. Such cases are undoubtedly rare, and it may be doubted whether they constitute a separate type. Without accurate post-mortem examination, especially of the bone marrow, the diagnosis must remain doubtful.

3. The cases of leukæmia are also a doubtful type. Some are probably cases of pernicious anemia; more of them are certainly cases of leukemia, either in remission or of an aplastic type when seen. The general consensus of English and American writers appears to assign the majority of the reported cases to the group of atypical leukemias.

The question still remains, however, of the first group. Are we to regard acute lymphocytic leukemias, on the strength of these rare cases, as examples of new growths of a character only slightly malignant as a general rule, but occasionally assuming great virulence and giving rise to widespread metastases? Or are we to regard leukæmia, not as a neoplasm at all, but as in all probability an infection, and assume that the leukæmic characters of the new growths—chloroma and its like—in reality have no direct connection with leukemia? On the whole, the facts seem to agree best with this last theory. Chloroma is to be regarded as a primary new growth of bone marrow, behaving in most respects very like new growths elsewhere; while leukemia, especially in view of its remissions, must be considered to be more nearly allied to the infectious diseases.

Ætiology.—In children the types of leukemia are somewhat less sharply differentiated than in adults, but the two main forms, myelocytic and lymphocytic, are prominent. In them, however, the myelocytic type, which in adults is so rare, is distinctly less frequent than the lymphocytic, though, with the increasing recognition that acute cases of leukemia are often of the myelocytic variety, the difference tends to become less accentuated. The so-called "atypical" leukemias appear to belong more especially to the acute myelocytic variety, and these are proportionately more frequent in children than in adults.

(a) **Myelocytic Form.**—**PREDISPOSING CAUSES** are unknown. There is no tendency to inheritance of the disease. Children are born of affected mothers without any trace of the disease.

PREVALENCE.—Hutchison in 1904 was able to find only five cases which he considered genuine; since that time Benjamin and Shaka have reported five

others which are beyond criticism; and the writer has himself seen three undoubted examples: one in a boy of eight years of age, one in a girl aged four, and the third in a boy aged three and a half years—a total of thirteen cases.

SEX AND AGE.—The numbers actually reported are too small to allow of any dogmatic statement as to the sex of the patients; but taking all cases below twenty years of age, the male sex appears to be slightly more liable. As to age, it is remarkable that, so far as the writer has been able to discover, there are but one or two undoubted cases occurring in the first two years of life, a peculiarity which has been noted also in the case of pernicious anaemia.

DURATION.—In adults the chronic character of the disease is well marked. A duration of two or more years is by no means uncommon. In children the reported cases have all been of brief duration, at most six to eight months, and in a few instances not more than a month to six weeks.

SYMPTOMATOLOGY.—HUNK OR ONSET.—The patient is brought to the doctor either on account of increasing lassitude or for the enlargement of the abdomen, which is in fact due to the enlargement of the spleen. In a few instances the anaemia has also been marked, but as a general rule, as in adults, the anaemia is not in the early stages a prominent symptom.

Besides these symptoms, there is often little which is the subject of complaint. The appetite remains good, and there is seldom any diarrhoea or constipation till the later stages. A distinction must be made, however, between the chronic and the acute varieties. In these latter the anaemia may be marked early in the disease, and there may be epistaxis, rarely of large amount; petechiae on the skin of the trunk or the thighs, and some oedema of the extremities; whereas these symptoms mark the closing stages of the more chronic varieties. *Pain* is an uncommon symptom, and when it occurs, seems to be due generally to the dragging on the adhesions formed by the spleen, or to sudden hæmorrhage within the spleen, which causes stretching of the capsule. *Dyspepsia* appears to be due principally to the anaemia, and occurs in the later stages. The *heart* is usually somewhat dilated, and a hæmic murmur, often rough in quality, is heard at the apex and over the præcordia. *Fever* is usually slight and very irregular, with occasional rises to 102° or 103° F. for a few hours. These are not readily explained.

The marked feature of the physical examination is the size of the *spleen*. This organ occupies the greater part of the left side of the abdomen, often reaching the iliac crest, and to the middle line of the abdomen at the umbilicus. It forms a hard, smooth tumour with rounded edges, is not tender to palpation, and is easily recognized by the presence in its anterior margin of the characteristic notches. It fills the loin, and can be traced upwards by percussion as high as the ninth or eighth rib in the mid-axillary line. The size of the spleen varies sometimes in the course of the disease; sudden enlargement, accompanied by pain, may be due to hæmorrhage into its substance; and in nearly all cases the approach of death is associated with a marked diminution in size.

The *liver* is increased in size—sometimes moderately; more frequently it reaches the level of the umbilicus below and of the fifth rib above.

The *lymphatic glands* are usually palpable in the neck and axillæ and groins, and in a few of the more acute cases form considerable masses, but in the typical myelocytic cases are rarely prominent. They are never in any case of the size familiar to the physician in cases of lymphadenoma.

The Blood.—The principal characteristics of the blood are the progressive loss of hæmoglobin and of red corpuscles, accompanied by an enormous increase in the number of white corpuscles, a large part of the increase being due to the appearance of myelocytes. The following is the record of a boy of ten years of age:

Red corpuscles	3,362,000 per cubic millimetre.
White corpuscles	400,000 " "
Hæmoglobin	70 per cent.
Colour index	0.99

In the next few months these figures varied considerably, but the white corpuscles were always above 100,000 in number, and generally above 400,000. The hæmoglobin alone remained steady. The varieties of white corpuscles present were as follows:

	Percentage.	Absolute Numbers per C.Mm.
Polymorphonuclear leucocytes	24.0	96,000
Lymphocytes	5.0	24,000
Large mononuclear leucocytes	16.0	64,000
Eosinophil leucocytes	2.5	10,000
Basophil leucocytes	15.5	62,000
Myelocytes	37.5	152,000

It will be seen that the absolute numbers of all forms are considerably increased, but the striking feature is the enormous number of myelocytes, more than a third of the whole number. This preponderance is apt to distract the observer's attention from the increase of the other forms, and stress must be laid on the fact that the polymorphonuclear and lymphocytic cells are both present in enormous excess. In this particular instance the basophil or mast cells were present in an unusual proportion, and, though their numbers are always greater than in normal blood, they are seldom seen in such profusion. The percentage of eosinophil leucocytes is within normal limits: the absolute numbers of course vastly increased. The cells of all these types vary in their morphology considerably, both in size and in staining reactions. The polymorphonuclear cells are often undersized, and so also are the myelocytes; while the staining reactions, especially with the congo-blue stains, present all shades and tints. The red cells also vary in size, and exhibit a tendency to take on darker tints than normally is the case, in some instances showing a distinct purple. Nucleated red cells, both megakaryoblasts, normoblasts, and microblasts, are common, but the normoblast predominates. Blood-platelets are said to be increased.

Taking the mean of the counts in the thirteen cases mentioned above, the red corpuscles were 2,500,000 per cubic millimetre—i.e., about 50 per cent. of the normal; while the white corpuscles were 438,000 per cubic millimetre, and of these an average of nearly one-half were myelocytes.

The condition of the blood does not correspond in any way to the condition of the patient. The patient who had the highest of all these counts lived fully six months from the time of the recognition of the disease.

In this series remissions were not observed, but in the adult it is not infrequent to find the total number of white cells sinking to a comparatively low figure, and even reaching a normal or subnormal level, but retaining as a rule the characteristic abnormal cells.

Acute Myelocytic Leukæmia.—The description given applies to the cases in children corresponding to the chronic myelocytic leukæmia of adults. More rarely cases are met with corresponding to the acute myelocytic leukæmia. Benjamin and Sluka record two such, but it is difficult to understand on what grounds they separate them from the chronic cases. The writer has seen one such case which may be called "acute," on the ground that, besides the well-defined myelocytes present in the film, there were numbers of mononuclear cells of the myelocytic type, but without granules—the so-called "promyelocytic" type. It is possible that with the recognition of these cells the diagnosis of acute myelocytic leukæmia will become more frequent.

(b) **Lymphocytic Leukæmia**—**PREDOMINANT CAUSES.**—As in the case of myelocytic leukæmia, nothing of importance is known with regard to the ætiology of the disease.

FREQUENCY.—In children it is distinctly more common than the myelocytic type. Hutchison in 1904 collected twelve cases. Benjamin and Sluka in 1907 found some thirty more; Forbes and Laignes reported twelve cases from the records of the Hospital for Sick Children, London; and in the last three years there have been at least double this number recorded in the German, English, and American literature.

SEX AND AGE.—The sexes are probably, on the whole, equally affected, though in Benjamin and Sluka's series boys predominate as 2 to 1. On the whole, also, the age at which the affection makes its appearance is earlier than in the myelocytic type. In Forbes's series ten out of twelve cases were under four years, and five of these were under two years of age.

DURATION.—Short as is the duration of the myelocytic type, that of lymphocytic leukæmia is much briefer. Nine of Forbes's twelve cases were dead in six weeks from the onset, and of the total above-mentioned, fifty-four in number, only eight survived more than two months.

SYMPTOMATOLOGY—**MOOD OR ONSET.**—Very frequently the first indication of disease is the occurrence of petechiæ on the skin and bleeding from the gums; much more rarely the child is brought for increasing pallor or enlargement of the abdomen; and in a few cases the glandular enlargement is the cause of alarm. This is in marked contrast with the same disease in adults, in whom the enlargement of the glands is in more than half of the cases the warning symptom.

A child suffering from lymphocytic leukæmia is nearly always markedly anæmic, the skin being waxy or a greenish-yellow in appearance, and the lips almost colourless. The skin is mottled over with petechiæ, which are generally small and sparsely scattered over the trunk and limbs. If the gums are affected they are swollen, purple in colour, and oozing blood; and there may be hæmorrhages in addition on the palate or in the fauces. Retinal hæmorrhages are not uncommon, conjunctival rare. Epistaxis is frequent, seldom severe. Hæmorrhage from the bowels and from the urinary tract is common, though seldom marked in amount—rather a continuous oozing than a sudden outburst.

The glands at the angle of the jaw are often considerably enlarged; the superficial glands in the body elsewhere are seldom more than palpable. The liver is nearly always enlarged, generally to a considerable extent, even to the level of the umbilicus.

The spleen also is always enlarged, generally only to a moderate degree, but

in exceptional instances reaching to the level of the umbilicus. Enlargement to the degree found in myelocytic leukemia is unknown.

Fever is always present, seldom reaching normal, and sometimes remaining at a high level, though more frequently intermittent in type. The patient is restless, without appetite, liable to attacks of diarrhoea, and rapidly passing to a state of exhaustion and unconsciousness.

The Blood.—The principal characteristics of the blood are the steady fall in the total number of red corpuscles, and the considerable increase in the number of white corpuscles, for which increase the non-granular cells are almost wholly responsible. The average number of red corpuscles is from 1,500,000 to 2,500,000, and in the most severe cases this number may sink below 1,000,000. Nucleated erythrocytes are nearly always present in considerable numbers, chiefly as normoblasts. The hæmoglobin also is much diminished. Percentages of above 30 are uncommon, and those of 20 to 25 are most usual. The colour index is usually well below 1.0.

The number of white corpuscles seldom reaches the totals recorded in myelocytic leukemia. Counts below 50,000 per cubic millimetre are frequent, and those above 200,000 are rare. The largest total, however, 1,527,000, which the present writer has met with in leukemia, was in a case of the lymphocytic type.

It is in the differential count, however, that the distinction becomes obvious. In place of the predominance of the granular cell in myelocytic leukemia, we find in this type a predominance of the non-granular cells, which constitute from 70 to 99 per cent. of the total number. It is a frequent experience to examine a film of the blood, and find less than 5 per cent. of granular cells present. In twenty-one counts recorded by Forbes and Langmead, the small and large lymphocytes counted together averaged 90 per cent. of the total. The lowest figure was 61 per cent., the highest 99 per cent.

It is customary to speak of the non-granular cells of lymphocytic leukemia as "lymphocytes," and to classify them as small or large according to the standard of the average lymphocyte of normal blood. But in these acute leukemias many of the cells called "lymphocytes" are distinct from the lymphocytes, and also from the large mononuclear cells of normal blood. Many attempts have been made by different writers to define the exact variety present in a given instance, but on the whole without success; and at present the physician must be content to accept Cabot's summing-up: "(1) That these cells belong in all probability to a group very near the primordial cells, whence both leucocytes and erythrocytes are derived; (2) that they are probably marrow cells rather than lymph-gland cells."

(c) *Atypical Leukemia.*—While the majority of leukemias fall easily into one of the two types discussed, there remain a few cases which are difficult, from one reason or another, to classify directly.

(1) "Mixed Leukemias."—Cases are often reported under this heading, chiefly owing to the fact that in all cases of myelocytic leukemia the lymphocytes are enormously increased as regards their absolute numbers. This is especially the case with children, and towards the termination of the disease, when a marked increase in the number of the non-granular cells is apt to occur. On the other hand, a considerable proportion of the cases of lymphocytic leukemia, especially those which are prolonged beyond a few weeks, tend to show a moderate number

of myelocytes in the films. To give to these variations such a title as "mixed leukemia" is merely to add to the confusion, especially now that it is believed that many of the so-called "large lymphocytes" are really derived from the marrow. Where, as Cabot says, there is a high leucocyte count, with a large proportion of granular cells, polymorphonuclear, eosinophil, basophil, or myelocytes, such a case should be classed as a "myelocytic leukemia," even though the percentage of non-granular forms is fairly high.

(2) **LEUKEMIA WITHOUT A LEUCOCYTOSIS.**—In a few cases the disease may run its course without any increase in the total number of leucocytes circulating in the blood. Such cases are very rare, and it is more usual to find that the fall in the total numbers is an initial and temporary phenomenon, and that subsequent counts give increasingly larger numbers. But even where the total number is small the proportion of the various forms of leucocytes will invariably give the key to the problem. Such a case was reported by Hand. A boy of two and a half years was admitted to hospital for anemia and hemorrhages beneath the skin and from the mucous membranes. The blood-examination showed only 6,800 leucocytes per cubic millimetre, but the differential count showed that of these 85 per cent. were lymphocytes. The axillary and inguinal glands were considerably swollen, and the spleen and liver enlarged. Death occurred within six weeks from the onset of the illness, a few days after the child was first seen.

(3) The cases already referred to, in which, with the blood-changes characteristic of leukemia, there are present glandular swellings which behave like neoplasms (sarcoma, chloroma).

PATHOLOGY.—Besides the alterations in the hemopoietic system, the alterations found post mortem are slight.

The spleen is always enlarged, varying in size from the immense firm, fibrous organ of chronic myelocytic leukemia to the soft, dark purple, moderately-sized viscus of acute leukemia. In chronic cases there is often some peri-splenitis, with adhesions to the neighbouring peritoneum. On section the firm spleen is greyish-red in colour, often mottled with darker patches, and showing areas of hemorrhage or infarction. It is rare to obtain a reaction showing the presence of free iron. Microscopically the structure of the spleen is altered by three processes: myelocytic or lymphocytic infiltration, fibrosis of the interstitial tissue, and hemolysis. The lymphatic glands, both of the superficial and deep groups, are variably enlarged, rarely to a great size, and often not larger than is common in some of the infectious fevers. Microscopically they are purplish-red from the amount of infiltrating cells, or greyish from the increase of interstitial tissue. Microscopically they exhibit the same features as the spleen.

The bone marrow of the long bones in children commonly contains far less fat than that of adults, so that the red transformation, which is a marked feature in the latter, is not obvious macroscopically. Microscopically the marrow contains large numbers of mononuclear non-granular cells in all forms of leukemia, and in the myelocytic type a considerable number of fully-differentiated granular cells. In addition anucleated erythrocytes are abundant.

The liver is always enlarged, usually pale or mottled with pale areas. Histologically the periportal and capillary spaces are thronged with leucocytes—myelocytes in the one form, lymphocytes in the other. In the more acute cases these deposits are more scattered, and large areas of the liver substance will show no other change than a moderate degree of fatty degeneration.

The kidneys are large and pale, and in some cases mottled with hemorrhages. Microscopically, in lymphocytic leukemias, the infiltration of the interstitial tissue with lymphocytes may be so marked as to obscure the structure of the organ, and to simulate that of a round-celled sarcoma. The cellular infiltration does not, however, in leukemia invade or destroy the structure, but lies in the capillary spaces.

The lungs also often show infiltration, especially in the capillary and lymphatic spaces around the bronchi and beneath the pleural membrane.

The heart muscle is pale, and shows "tabby cat" striation; microscopically the tissue may show patches of lymphocytic infiltration.

Hæmorrhages.—Petechiæ are often found beneath the pericardial and pleural membranes; and occasionally small extravasations have been noted in the meninges or in the mucosa membrane of the stomach and intestines. Extensive hæmorrhage in any part of the body is unusual.

NATURE OF THE DISEASE.—Three views may be noted: (1) The generally accepted theory that the disease is an affection of the whole hæmopoietic system, affecting the marrow, glands, spleen, and the lymphadenoid deposits in the viscera, with special incidence on one or other of these structures in different cases; (2) that it is primarily a malignant growth of the bone marrow; (3) that it is an infection by bacteria.

The first of these views leaves open the question of the exact cause of the disease, and maintains only the essential unity of all forms of leukemia; that it is a disease, rarely, which has the hæmopoietic system as its site, and that the various manifestations are all to be explained as variations of the efficient cause, acting with more or less vigour upon the different portions of the system. The more the disease is studied, the greater appears the probability of this view. "Differences," as Cabot says, "tend to disappear, identities to be accentuated."

The second view, that acute leukemia is primarily a malignant growth of marrow, is in the writer's opinion untenable. It finds its support in the close relationship of the certain forms of the disease with certain forms of sarcomatous growth; but it leaves out of sight altogether the relationship of the acute with the chronic leukemias, and it fails entirely, as Forbes and Langmead point out, to offer any explanation of the fulminating cases in which death occurs in a few days.

The parasites described some years ago by Löwen have not found acceptance with pathologists; they are difficult to demonstrate, not found at all in many cases, and are probably not living organisms at all.

If one confines one's attention to acute leukemia, it is extremely difficult to resist the conception, at least for some cases, of a bacterial infection. The sudden onset, the fever, the resemblance to septicæmia, all heighten the likeness to acute bacterial disease. Further, in a certain number of cases streptococci have been recovered from the heart's blood, bone marrow, and viscera. The writer was enabled by Forbes to examine two of these organisms, and found that they corresponded to the ordinary streptococcus of the mouth, which is not infrequently found as a terminal infection in other diseases. The evidence is on the whole against a bacterial origin, for, however strongly the course of the acute disease suggests it, there is still the group of chronic leukemias to be accounted for, and no explanation which would separate the two groups can be satisfactory.

A possible conception is that the hæmopoietic system is liable to attack by

a variety of toxins, which have affinity for one or other of the different tissues, and are manufactured inside the body in morbid metabolic conditions, whether originating in bacterial infection or not.

It must, then, to summarise, be concluded that leukaemia is a disease of the hæmopoietic system differing in type according to the particular portion of the system primarily attacked, and that its efficient cause is entirely unknown; that the disease is not a bacterial infection, and not a neoplasm, but possibly caused by some toxin or group of toxins manufactured in the body.

DIAGNOSIS.—The diagnosis of leukaemia from other anæmias rests chiefly upon the examination of the blood. In the chronic myelocytic type the splenic tumour can hardly be mistaken for anything else; both kidney tumours and tumours of the intestines are utterly unlike it, both in general shape and outline and in position; and a very cursory examination of the blood will at once show the characteristic changes. The only condition which in the least degree resembles a chronic myelocytic leukaemia is the peculiar form of infantile anæmia described by von Jaksch, under the cumbersome title of "*anæmia pseudo-leucæmica infantum*." In this disease, however, the total leucocyte count rarely rises above 30,000 to 40,000, and of these cells the proportion of myelocytes is usually comparatively small. The age of the patient is also of importance, since von Jaksch's anæmia is almost confined to the first two years of life, whereas, as has been seen, myelocytic leukaemia commonly attacks older children. The distinction is important, for 60 to 70 per cent. of cases of von Jaksch's anæmia recover completely.

In lymphocytic leukaemia the diagnosis with the aid of a blood-examination is also as a rule easy. The chief difficulty arises in cases where the absence of glandular or splenic enlargement has led to the neglect of a blood-count. Such cases are apt to be described as purpura, or septicæmia, or even typhoid fever.

There are, however, difficult cases:

1. In purpura with a high degree of pallor, and possibly some conjunctival hæmorrhage or epistaxis, a blood-count may show a marked lymphocytosis. Cabot quotes a case in which the leucocytes numbered 94,000 per cubic millimeter, 75 per cent. of which were lymphocytes.

2. In a few cases of *aplasia* of the lymphatic glands, instead of the polymorphonuclear leucocytosis expected, a blood-count reveals a lymphocytosis. The writer has never met such a case.

3. In a few cases of *recoarctation* of the intestine, the circulating blood may contain a large number of lymphocytes, but as a general rule the total count is well below 20,000 per cubic millimeter.

4. Cases of purpura may superficially resemble acute leukaemia, but the leucocytosis in this disease is rarely high, and the increase is invariably in the polymorphonuclear cells.

5. The real difficulties arise in connection with the *atypical leukaemias*, and in some of these it may be impossible to make the diagnosis during life.

The diagnosis between the two forms of leukaemia is not a matter of great clinical importance in children, since the duration of either is extremely limited. But it may be briefly stated that in myelocytic leukaemia the leucocyte count is on the average high, above 200,000, while in the lymphocytic form it is more likely to be below 100,000; that hæmorrhage is more frequent in acute leukaemias; that a considerable number of acute leukaemias show cells which, though non-granular, are in general appearance more nearly allied to marrow than to lymph-

gland cells; and that where such cells are present the disease is, on the whole, more rapidly fatal than in the other forms.

PROGNOSIS.—Leukæmia in children is probably invariably fatal. From time to time cases of recovery are reported, but the writer knows of none which will bear critical examination. The average duration of life after the disease has declared itself is in the acute cases a few weeks, in the chronic cases a few months, at best. Of remissions, as seen in adults, during which the health is regained and the blood becomes normal, the writer has found no record in children.

TREATMENT.—The treatment of either form of leukæmia offers a gloomy outlook. In acute leukæmia, as a general rule, the end comes swiftly; but in a small proportion of cases temporary recovery from the severe symptoms takes place. The writer has seen an adult patient of Drysdale's who recovered from what seemed a desperate condition, and lived to enjoy two years of good health, but knows of no similar instance in a child. Drugs innumerable have been given without apparent effect. In the case above-mentioned, naphthalene tetrachloride was employed in doses of 30 grains a day, and the result certainly seems to warrant further trial of this or similar medicines. Arsenic, iron, iodides, extracts of various organs, and antimitotic serums, seem quite useless to avert the fatal termination, but occasionally benefit the patient temporarily. Arsenic, whether given by the mouth or subcutaneously, is probably the best of these. If used, it should be employed in increasing doses. For chronic myelocytic cases there can be no doubt that X-ray treatment offers some hope of improvement in a few cases. The spleen becomes smaller, the blood tends to assume its normal condition, and the patient is less anæmic and begins to enjoy life again. The improvement is in most instances not long maintained, and no authentic case of cure is yet recorded, but the results as regards the comfort of the patient are considerable. In the acute cases the treatment appears to offer less benefit; indeed, in some cases it has seemed to hasten the end.

(d) **Chiocoma.**—**DEFINITION.**—A sarcomatous growth of marrow-like tissue with green tint, invading the bones, especially of the skull, accompanied by changes in the circulating blood resembling those found in lymphocytic leukæmia.

FREQUENCY.—In 1907 Benjamin and Sluka collected forty-two cases, in nineteen of which the blood had been studied carefully. Of these, sixteen were in children below twelve years of age. Since then Treadgold has collected eight others, two in children.

SYMPTOMATOLOGY.—The disease manifests itself insidiously: the child becomes pale, listless, and irritable, and complains of headache. This is due to the pressure of the tumours in the skull, which, as it increases, produces deformity of the temporal and frontal bones, exophthalmos, optic neuritis, swelling of the veins of the head and neck, and deafness. Hemorrhages take place in the majority of cases, from the nose or mouth, and occasionally from the stomach or bowel. The lymphatic glands in the neck and axilla are enlarged; and the spleen, though never enormous, is generally definitely palpable. Emaciation is rapid; fever is inconstant, occasionally high. The duration of the disease is short—when the symptoms have manifested themselves, from one to two months only.

PATHOLOGY.—On section, the tumours which infiltrate the bones show a vivid green colour, which varies in tint in different cases, and is apt to fade quickly.

The tumours are found especially in connection with the bones of the orbit, the temporal and frontal bones, the vertebrae, ribs, and sternum. Metastases have also been found in all the viscera, the glands, the long bones of the limbs, and even in the skin. The growths are extremely soft and pliable, but do not appear to be liable to hemorrhages. In some cases, not all, the tumours exhibit the green colour.

DIAGNOSIS.—Where the typical infiltration of the skull bones has taken place, the disease can be recognized at a glance, the only possible confusion arising from the sarcomata originating in the suprarenal bodies described by Hutchison. From these it is easily differentiated by an examination of the blood, which shows the characters described under acute lymphocytic leukemia. In a few cases the blood has been described as of the myelocytic type.

PROGNOSIS AND TREATMENT are alike hopeless.

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(c) **Anæmia Pseudo-Leukæmia Infantum** (von Jaksch)—**DEFINITION.**—A disease of infants characterized by great enlargement of the spleen, a high degree of anæmia, and the constant presence of myelocytes in the circulating blood.

INTRODUCTION.—In the year 1889 von Jaksch published an account of a form of anæmia in a young infant which, in his opinion, constituted a distinct type. It resembled closely true leukemia in its clinical aspect, but there were certain characteristic symptoms which differentiated it sharply from that disease. In his original description the chief characteristics of the type were a marked enlargement of the spleen, a moderate enlargement of the liver, occasional enlargement of the superficial lymphatic glands, and alterations in the circulating blood. These were principally leucocytosis of a considerable degree, and a great diminution in the total numbers of the erythrocytes. The name which he proposed for the condition was the cumbersome title of "*anæmia pseudo-leukæmia infantum*"—a title which, in spite of its obvious drawbacks, is in the writer's judgment better than that more usually employed in this country—"splenic anæmia of infants."

For a considerable time there was not much attention paid to von Jaksch's paper, especially in England; but in Germany, and of recent years in this country, there has been published a considerable literature, devoted for the most part to the consideration of the point whether the disease in question is merely a severe anæmia owing its peculiarities to the age of infancy, or a special and distinct type of anæmia.

Von Jaksch's original communication laid no stress upon the character of the leucocytes present in the circulating blood, a point which, in the writer's opinion, is of considerable importance; but his clinical description was exact and complete, and but little has been added to it. The following account is based upon a series of cases investigated by Drysdale and the writer in 1904.

ÆTIOLOGY.—AGE.—The disease is practically confined to the first three years of life, and the larger proportion of all cases accurately reported in Germany, America, and this country, falls within the first two years.

DATE OF ONSET.—The average age at which patients come under observation is eighteen months; but since in all cases the disease is fully developed when the patients are first seen, it is probable that they are attacked about the beginning of the second year of life.

SEX.—The sexes are about equally affected. In the series studied, nine were females, fourteen males.

RACE.—Some writers believe that the Hebrew race is more liable to the affection, but the present writer does not share that opinion.

FAMILY INFLUENCES.—There is no evidence that heredity plays any part in causation, but there is a distinct tendency for children of the same family to suffer from the disease, as they successively reach the age of usual incidence—a fact which is probably to be explained by similarity of circumstances producing similar effects. The same explanation would also hold good if it is the case, as alleged, that twins form an undue proportion of the cases.

PREDISPOSING CAUSES.—Of these little is known. There is no good evidence pointing to the influence of any mode of feeding, or any previous infection preceding the onset; nor that rickets, congenital syphilis, or a marasmic condition, are factors in the peculiar characters of the anemia. These diseases are, it is true, found in conjunction with it, but not more frequently than would be expected, for the patients who are the subjects of anemia pseudo-leukemica are almost invariably drawn from the lower ranks of the social scale. The writer has not seen, nor been able to hear of, a case among the children of the wealthier classes.

SYMPTOMATOLOGY.—MODE OF ONSET.—The onset of the disease is always insidious, and by the time that the increasing pallor or the enlargement of the abdomen attracts the attention of the parents the condition is fully developed. In some instances the beginning of the illness is dated from an epistaxis, in others the petechial eruption excited alarm; but in the majority the pallor, wasting, and the abdominal enlargement, are the cause of the visit to the doctor. As a general rule it is easy to distinguish these cases at a glance. The pallor, which is invariably marked, is of a waxy character, often with a yellow or greenish tinge. The patients are sometimes wasted, but more often retain a considerable amount of subcutaneous fat, and seldom show any sign of wasting in their facial aspect. The lassitude and listlessness are betrayed by their slow and uncertain movements, and also frequently by their lack of irritability. They appear too tired to resent examination, even of the fauces. The trunk and limbs are in the majority of cases spotted with small petechiae, and often show more signs of wasting than the face. The muscles are soft and flabby, and in severe cases the ankles and dorsum of the feet pit slightly on pressure. The abdomen is in all cases protuberant, the enlargement being due principally to the increase in size of the spleen. The enlargement of this organ is truly enormous. In the twenty-three cases there was but one in which it did not reach the iliac crest, and in most cases the middle line of the abdomen. It is hard, smooth, and free from tenderness.

The liver, on the contrary, is seldom of any great size, and probably never much above the normal, although its edge may be felt easily below the costal

margin. In these patients, as also inrickety infants, owing to the wide costal angle and the distended abdomen, a liver of normal size is often unduly palpable.

The Glands.—Glandular enlargement is unusual in the superficial groups, but occasionally one or more glands may be felt somewhat more easily than usual. After death it is not unusual to find the mesenteric glands considerably enlarged, and in some cases the bronchial and tracheal groups also.

The degree of wasting is usually not severe as regards the subcutaneous fat—at least, in uncomplicated cases; but in the course of the disease, extending as it does over many weeks, the body-weight remains stationary or slowly declines, until convalescence begins. Indeed, an increase in weight is one of the earliest favourable signs. The muscular wasting, on the other hand, is often considerable.

Edema, slight in degree, of the hands and feet is not uncommon, and a slight puffiness of the face, especially below the eyes, is often present. Such edema is intermittent, and the cause of it is never obvious. It is unconnected with changes in the urine, which is normal in quantity and appearance. It occasionally contains a slight trace of albumin, but there is never any evidence of nephritis.

Hæmorrhages.—Epistaxis is common, and often repeated, but seldom severe enough to excite alarm. Petechiæ on the skin of the trunk and limbs have been already mentioned. Besides these two forms of hæmorrhage all others are rare. Blood has been seen in the stools, in the urine, and oozing from the gums has been once observed. In no case was it serious or prolonged.

Fever of an irregular type is generally present—at least, at some part of the illness—but in a few cases is absent altogether while the patient is under observation. The temperature rarely reaches more than 102° F., and then only for a few hours at a time. The fever which accompanies an intercurrent broncho-pneumonia is often, of course, more severe and prolonged.

Diarrhoea occurs in about half the cases in the form of intermittent attacks. In the intervals the motions are as a general rule normal. Vomiting, except early in the illness, is not common. Bronchitis and broncho-pneumonia are frequent and often fatal complications. Of the fatal cases, nearly all die of respiratory disease or of an acute and uncontrollable diarrhoea.

The Blood.—The prick of the needle in a severe case is followed by a drop of watery blood of a paler tint than normal. The hæmoglobin is as a rule diminished considerably, but seldom sinks to the level which the degree of pallor would lead one to expect. The lowest record was 19 per cent., the average 38 per cent. The red corpuscles showed an average of 2,500,000 per cubic millimetre, and low counts are very uncommon. The colour index averaged 0·6. Alterations in shape and size of the red corpuscles are not very prominent features, but here and there individual corpuscles may show marked changes. Nucleated erythrocytes, usually normoblasts, are always present in the severer stages of the disease, sometimes in very considerable numbers. Megaloblasts are less common, but in one case at least outnumbered the normoblasts. The leucocytes numbered on an average 24,100 per cubic millimetre, but in this circumstance the variations are extensive. Counts as low as 5,000 per cubic millimetre, and as high as 60,000 per cubic millimetre, are not uncommon. An increase is certainly far more often observed than a decrease. The highest count observed by the writer—82,000 per cubic millimetre—was in a case not included in the present series. The count was made at a time when there was no recognizable complication, and the patient eventually recovered. The degree of leucocytosis does not indicate the degree of severity of

the disease. In two cases known to the writer, death ensued without any increase in the number of the leucocytes, while in several instances where the leucocytes numbered 30,000 and upwards recovery has taken place.

The varieties of leucocytes are all increased, but the proportionate increase is greatest in the lymphocytes. Hence, where percentages alone are stated, the polymorphonuclear cells are apparently decreased, though their actual numbers are larger than the normal.

The feature, however, of the blood-film is the constant presence of myelocytes. In no other disease except leukemia are these cells present in anything like the same proportion, though a few may be found in some of the acute infectious diseases—e.g., diphtheria or scarlet fever. The great majority of the myelocytes possess neutrophil, but a few may show eosinophil or basophil granules. The average percentage present in the series of cases which is under review was 5.7 per cent. The number steadily diminishes with the improvement of the patient, and their estimation is probably one of the best means of testing the progress of the case.

Summary of the Blood Changes.—A diminution of the red corpuscles, with a slightly more marked loss of hemoglobin, accompanied by an increase, usually, of the total number of leucocytes, of which myelocytes invariably constitute a marked percentage.

Pathology.—The gross anatomical appearances of this disease are practically confined to the spleen, whose size is always very great, though usually less than has been estimated during life. This is probably due to a rapid shrinkage in volume, which has been observed clinically during the last few days of life. The organ is smooth, hard, and on section shows nothing abnormal, beyond a slight increase of the fibrous septa. Microscopically there is no change, unless it be an increase in the amount of the connective tissue. The mesenteric glands are often enlarged, but show no microscopical alteration except a general hyperplasia. The Peyer's patches and solitary glands of the intestine are often prominent, but there is no ulceration. The liver shows fatty degeneration. The lungs, except where broncho-pneumonia has terminated the illness, are normal. The bone marrow shows no gross alteration, and microscopically little more than an excess of the more recently formed erythrocytes and leucocytes. Bacteriological researches have failed to demonstrate any infecting organisms in the viscera or bone marrow, and blood-cultures made during life are sterile. No decisive observations have been made with regard to parasitic protozoal forms. Some of these have been described as occurring in the spleen, but the bulk of trustworthy observation is entirely negative. The cause of the disease is therefore entirely unknown, and here, as in true leukemia, medicine is at present reduced to hypothesis. The most reasonable hypothesis is that the condition is the result of the circulation in the body of a toxin or combination of toxins manufactured in the processes of morbid metabolism, whether these are initiated by bacterial infection or no.

Relationship to Other Diseases.—1. *Other Anemias.*—It has been contended that this affection is not a separate disorder, but an anemia, which owes its special characteristics to the age of the patients; that severe rickets, congenital syphilis, or chronic intestinal disease, will produce the condition. In the writer's judgment, there are two facts which compel a contrary opinion—that in none of these conditions, however severe, has an anemia with the peculiar characteristics ever been observed to develop; and, secondly, that there are a number of cases of

this disease in which all the alleged predisposing conditions can be definitely excluded.

2. *Relationship to Leukæmia.*—This relationship is much harder to define. The disease has some of the characteristics of myelocytic leukæmia, but, on the other hand, its course and the comparatively low degree of mortality are very unlike those of leukæmia; moreover, the condition of the blood, though recalling that of myelocytic leukæmia, never presents the striking pictures that are familiar in that disease.

In one or two cases observed by the writer, which have ended fatally, the terminal scenes have been signalized by enormous influx of lymphocytes into the circulating blood, producing the picture seen in acute lymphocytic leukæmia, and it is, he thinks, difficult to resist the conclusion that these cases have terminated by an attack of that disease. But it is certain that these are exceptional instances, and on the whole the disease is distinct in its onset, course, and prognosis, from any form of leukæmia known in adults or older children.

It follows, from what has been said, that *anæmia pseudo-leukæmica infantum* must be considered as a special form of anæmia, a disease *sui generis*, confined to the first three years of life, running a protracted course, characterized by a great hyperplasia of the spleen and a condition of the blood peculiar to itself.

DIAGNOSIS.—The diagnosis is seldom difficult, but must rest upon the combination of three conditions—anæmia, the splenic enlargement, and the peculiar myelocytic condition of the circulating blood. From any ordinary anæmia occurring in infants the blood-condition alone will easily distinguish it; from myelocytic leukæmia, a rare disease in infants, the moderate leucocytosis, the low percentage of myelocytes, and especially the course of the illness, separate it at once. In rickets and congenital syphilis the spleen rarely attains the size that is common in this disease, and the destruction of the red corpuscles is seldom so severe. Infantile scurvy and idiopathic purpura bear no sort of resemblance to it either in clinical features or in the condition of the blood. The only difficulties which have, in the writer's experience, occurred have been to decide whether a given case was an example of myelocytic leukæmia or of von Jaksch's disease, a difficulty correctly solved by careful blood-examination; and, secondly, in one of the rare instances above referred to, whether the patient was suffering from this disease or from acute lymphocytic leukæmia.

PROGNOSIS.—From the consideration of all the cases carefully reported, it appears that from 65 to 70 per cent. of the patients affected eventually recover, and that recovery is complete and permanent. Patients who suffered from this disease in infancy have been examined at later ages, and showed no traces of the affection, except that their spleens could be perhaps rather more easily felt than in normal children of the same ages. The duration is always prolonged. Weeks, or even months, pass without there being the least alteration in the patient's condition; then slow improvement begins, heralded, as mentioned above, by the disappearance of the myelocytes from the circulating blood. The spleen diminishes slowly in size, and even a year or more after the anæmia has completely vanished, and the general health has become excellent, may still be unduly palpable. This is a phenomenon which a consideration of the histology of the enlarged spleen, with its overgrowth of connective tissue, would naturally lead one to expect.

When a fatal result occurs, it is almost invariably directly produced by acute

diarrhoea or by an attack of broncho-pneumonia. In exceptional cases, as mentioned previously, the onset of an acute lymphocytic leukaemia causes death. The writer has never seen a fatal result from hæmorrhage.

TREATMENT.—During the chronic period of the illness all the usual methods of attacking the anaemia appear to be without the least effect. Iron, arsenic, bone marrow, cod-liver-oil, mercury, and raw-meat juice, have all failed to produce the slightest improvement. The patients appear to do best when treated as far as is possible out of doors, with abundance of light and air. Whenever there is obvious rickets or the signs of congenital syphilis, it is, of course, wise to pursue treatment directed specially to these affections.

In the absence of any knowledge of the cause, it is advisable to act upon the hypothesis suggested above, and to use careful dieting combined with intestinal disinfectants; but in the use of these it is necessary to proceed with caution, for diarrhoea is one of the most fatal of complications. Hence the intestinal disinfectants used should be of the coal tar group—salol, iodo, and the like—and they should be employed in small doses.

So far as the writer knows, excision of the spleen, which has been attended by such brilliant results in certain cases of enlargement in adults, has not been practised in this class of case; nor does it seem likely to be attended with good results; nor, indeed, do the majority of cases, since recovery takes place, call for such a measure.

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IV. POLYCYTHÆMIA.

DEFINITION.—Polycythæmia is the name given to conditions in which the numbers of red blood-corpuscles per cubic millimetre exceeds the normal.

RELATIVE POLYCYTHÆMIA.—This excess may be due simply to concentration of the blood, either locally or generally. Local concentration, which has been shown, both experimentally and clinically, to exist in local cyanotic conditions, has no clinical importance.

Generalized concentration and the consequent polycythæmia is chiefly associated with conditions in which there has been a severe and rapid loss of the body fluids. Thus, it is common to obtain high erythrocyte counts in cases of diarrhoea or repeated vomiting in infants. The writer has made several counts of 6,000,000 and 7,000,000 in such conditions.

ABSOLUTE POLYCYTHÆMIA.—Here the total number of red blood-corpuscles is increased, not merely locally or temporarily, but over the whole body and for a considerable period. It is also probable that the total volume of the blood is increased. The most widely known example of an absolute polycythæmia is met with in congenital pulmonary stenosis, in which counts of 10,000,000 corpuscles and upwards have often been recorded. Chronic cardiac and pulmonary disease associated with cyanosis also give rise to a similar but less pronounced excess. The polycythæmia which occurs at high altitudes must also be mentioned.

To these conditions recent research has added another, which is at present believed to be due to an excessive activity of the bone marrow, excited by some stimulant the nature of which is unknown. The disease is characterized by cyanosis, polycythæmia, and in most cases considerable enlargement of the spleen. The cases so far reported have been observed in middle life, but since in several instances the cyanosis and splenomegaly are known to have existed since childhood (four cases up to 1909, Weber), it is only a question of time for cases to be recognized and recorded before puberty. The polycythæmia is as a rule persistent, reaching an average of 8,000,000 per cubic millimetre in the reported cases, and frequently above this figure. The hæmoglobin is from 120 to 180 per cent. as compared with the normal 100 per cent.; and the leucocytes, usually, but not always, increased, show a preponderance of polymorphonuclear cells. Nucleated erythrocytes are also often present, and occasionally myelocytes. The symptoms are chiefly those of peripheral congestion, epistaxis, vertigo, headache, dyspnoea, and the marked cyanosis of the skin and mucous membranes. The diagnosis is easily made by the exclusion of the ordinary causes of cyanosis and polycythæmia. The duration is protracted, often many years, and the termination comes from intercurrent disease or intracranial hæmorrhage.

TREATMENT has so far been unsuccessful, but diminution of the spleen and decrease of the polycythæmia has followed the use of X-ray applications.

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ENLARGEMENT OF THE SPLEEN.

Besides the diseases already discussed in this section, there are other conditions in which the enlargement of the spleen is a well-marked clinical feature, and a brief enumeration of these will be of service to the clinician.

1. Acute Infective Diseases.—There are probably few infective disorders in children in which the spleen never becomes enlarged. Even in such mild affections as rubella or varicella the organ is sometimes palpable below the costal margin, but in these cases the enlargement is of slight extent and short duration. But there are three conditions in which an acute infection is marked by a considerable and more persistent enlargement: (1) Typhoid fever, (2) septicæmia, (3) acute tuberculosis.

(1) **TYPHOID FEVER.**—The spleen in this disease usually becomes palpable about the end of the first week of the disease, and may attain a very considerable size, so as to form a definite tumour in the left hypochondrium. It is, however, more usual for the enlargement to be moderate, the lower edge of the organ reaching an inch or an inch and a half below the costal margin. It is very rarely tender to palpation, and is easily identified by its position and rounded edge, even if the notches cannot be felt.

(2) In **SEPTICÆMIE CONDITIONS** the spleen is often the site of infarcts, which may progress to abscess formation; but, again, as a rule the enlargement is moderate.

(3) **ACUTE TUBERCULOSIS.** See below.

2. Chronic Enlargements of the Spleen.—These are a much more numerous class. The enlargement of the spleen in leukemia, in lymphadenoma, and in von Jaksch's anemia pseudo-leukemica infantum, has been already mentioned. Other conditions in which there is a chronic enlargement of the organ are enumerated below:

(1) **CHRONIC VENOUS CONGESTION.**—In some cases of heart disease with backward pressure the spleen may undergo considerable enlargement, becoming hard and firm. In some of these cases the spleen is found post mortem to contain numerous infarcts.

(2) **HEPATIC CIRRHOSIS.**—Banti has described a condition in which an enlargement of the spleen with a marked anemia are the initial symptoms. To these are added, in the course of years, ascites and cirrhosis of the liver. The anemia is characterized by oligocythæmia, deficiency of hæmoglobin, and a low leucocyte count in which lymphocytes predominate. After a duration of many years the patients succumb with dropsy or repeated hæmatemesis.

The relationship of this disease to "splenic anemia" is obscure. This name has in the past been applied indiscriminately to a great variety of diseases which had in common the symptoms of splenomegaly and anemia. In especial, the disease which has in this chapter been called "anemia pseudo-leukemica," was often styled "the splenic anemia of infants." Recently, however, the name has been confined to a group of cases in which, with splenomegaly, there is a leucopenia with predominance of lymphocytes. In this group it is certain that there are several different conditions included: the type of disease to which Banti's name has been attached is one such condition; the splenomegaly with anemia of late congenital syphilis is a second; and there are no doubt others which will be separated from the group.

Cases which fall into this category, of whatever type, occur more commonly in adolescence than in childhood, and are unknown before the period of the second dentition.

(3) **TUBERCULOSIS.**—The spleen is enlarged in tuberculosis, both acute and chronic, and the enlargement is always due to the formation of tubercles in the organ. These tubercles are in the acute cases generally of the miliary type, and occur sometimes more obviously on the capsule than in the pulp. In the cases of generalized tuberculosis, both miliary and caseating tubercles are present, and the whole spleen may be dotted with grey or yellowish nodules both in the pulp and in the capsule. The most marked enlargements occur, however, in the more chronic types of tuberculosis, and are associated with the development of multiple caseous tubercles in the organ. In many instances such a spleen post mortem resembles both in size and appearance the "hardbake" spleen of lymphadenoma, from which it may be distinguished as a general rule by the yellower colour and less regular outline of the nodules, and the more frequent involvement of the capsule of the organ, with consequent peritoneal adhesions. Clinically an enlarged spleen is more often found in tuberculosis than in any other disease to which children are liable, and, in the writer's experience, is the only disease in which the splenic tumour is often distinctly tender to palpation.

(4) **SYPHILIS.**—The spleen in congenital syphilis is enlarged to different degrees at different ages and periods of the disease. In the initial period of the rash and the condyloemata in young infants, it reaches usually half an inch or more below the

costal margin, but very quickly diminishes, and can, in the writer's experience, seldom be felt after the age of three months. In the later periods of the disease after the second dentition, the spleen may be enlarged either as a result of lardaceous disease (see below) or in association with gummatous deposits, which, however, are rare.

(5) **RICKETS.**—Uncomplicated rickets is not per se a cause of enlargement of the spleen, but the common deformities of the chest in this disease lead frequently to such displacements of the abdominal viscera that the organ is frequently much more readily palpable than is natural. In some cases practically the whole spleen can be palpated owing to the depression of the diaphragm. In those instances where there is a definite enlargement of the spleen in a rickety child, it is nearly always possible to point to some contributory factor.

(6) **LARDACEOUS DISEASE.**—The chief causes of lardaceous disease are long-continued tuberculosis, syphilis, or chronic suppuration. The forms of the disease are two: (a) The "sago" spleen, in which the Malpighian bodies are the chief seat of the lardaceous deposit, the intervening pulp being unaffected; (b) the diffuse lardaceous type, or "hæm" spleen, much less common than the first, in which there is a diffuse lardaceous change in the walls of the arterioles and blood-sinuses throughout the organ. This form of the disease leads to greater enlargement than the first. In adolescence, and young adults the subjects of congenital syphilis, the combination of lardaceous disease and fibrosis produces occasionally enormous enlargement, comparable in size to the spleen of chronic leukaemia.

(7) **CONGENITAL ACHOLURIC JAUNDICE.**—A group of cases is designated under this name which probably includes several slightly different diseases. In some instances the jaundice is truly congenital, in others it appears only after some years; in some only one member of a family is affected, in others several. In nearly all, however, there is enlargement of the spleen, which often reaches a considerable size (see Chapte. V., p. 212).

Tumours of the Spleen.—Tumours of the spleen are rare pathological curiosities rather than clinical phenomena. Those found are—

1. **CYSTS.**—(a) **PARASITIC**—hydatid.
- (b) **NON-PARASITIC**—dermoid or simple fluid cysts.

2. **NEW GROWTHS.**—These, whether innocent or malignant, are of extreme rarity. Fibromata and sarcomata have been described—in the latter case almost invariably due to metastasis. The tumours are seldom observed during life.

V. DISEASES IN WHICH THE ALTERATION OF THE BLOOD, MANIFESTED BY HÆMORRHAGES, IS NOT RECOGNIZABLE BY OUR PRESENT METHODS.

(a) **PURPURA.**—**DEFINITION.**—Purpura is the name applied to spontaneous extravasations of blood in and beneath the skin and mucous membranes. More particularly it is applied to a disease in which this symptom is the chief feature, the cause being unknown.

INTRODUCTION.—In the earlier history of medicine, purpura was a name given almost indiscriminately to any disease in which hæmorrhages beneath the skin

were a prominent feature, and it is certain, for example, that many of the fulminating exanthemata were so described. However, in the latter half of the eighteenth century these were definitely separated off from the confused category, and a clinical description of the disease which we now recognize as purpura was published. Since that time it can hardly be maintained that any great advance has been made, although it has been fully established that purpuric manifestations accompany all sorts of morbid conditions. But when we have separated from the mass all those conditions in which we can recognize an antecedent cause, there yet remains a group of diseases to which the name is perforce applied because we cannot recognize any other symptom of prominence.

I. Secondary or Symptomatic Purpura.—In considering the classification of the group to which the name has been given, it will be most convenient to take in the first place those cases which may be legitimately classed as secondary—i.e., those in which the symptom purpura is not the chief manifestation, but only an accompaniment of another recognizable disease.

1. Infective Purpura.—Here in the first place we must put the purpuric or hæmorrhagic varieties of the different infective fevers—smallpox, measles, scarlet fever, and diphtheria. It is still uncertain why in rare instances these diseases, which usually run a well-defined course without hæmorrhage, should develop abundant subcutaneous and submucous hæmorrhages; but in the case of smallpox, at least, there is some evidence to show that the hæmorrhagic tendency is the result of streptococic infections which produce an active septicæmia. For another group of infective diseases such evidence is not forthcoming—the group including epidemic cerebro-spinal meningitis and typhus fever, both of which are accompanied by a profuse purpuric eruption. In yet a third group of infections, that of which infective endocarditis is the type, purpuric spots are a frequent manifestation, and the explanation of their occurrence is that small emboli of the infecting organism have blocked the vessels of the skin and subcutaneous tissues, leading to extravasation of blood in the neighbourhood. It must, however, be admitted that this is a supposition based upon known analogies rather than an ascertained fact, for the demonstration of such emboli has not yet been made.

A variety of purpura concerning which there is much dispute may here be discussed—the so-called "rheumatic purpura." That purpura does occur in close association with rheumatism is a well-ascertained fact; but the difficulty which faces the observer is whether this purpura is to be placed among the symptomatic purpuras, or whether it is to be elevated into a class by itself. Schönlein many years ago seems to have taught that a purpuric disease, which he entitled "*pebrosis rheumatica*," was closely associated with rheumatic fever. Later writers have found it difficult to appreciate what was the type of disease which he was describing, and have separated themselves into two camps: the one school maintaining that a purpura is an occasional complication of rheumatic fever, the other that a purpura with arthritic manifestations occurs quite distinct and apart from rheumatic infection. Poynton, in a recent lecture on the complications of rheumatic fever, has no doubt of the occasional rheumatic origin of purpura, and quotes a case of rheumatic fever with endocarditis in which the final scene was ushered in with a severe purpuric rash. In spite of this and other instances, several of them in the series on which this chapter is based, it is the belief of the writer that there is no essential connection between rheumatic fever and purpura. Some of the cases, such as the one Poynton quotes, should in his opinion be referred to the class of

symptomatic purpura—that is, it should be recognised that, just as there is a purpuric smallpox, scarlet fever, measles, and diphtheria, so there is a purpuric rheumatic fever; and that, just as there is some reason to suppose that these varieties are due, not to the primary, but to a secondary, often streptococcal, infection, so it may be with acute rheumatism. Other cases which have been styled “rheumatic purpura” or “purpura arthritica” show no evidence of rheumatic infection beyond the swelling and pain in the joints; never develop endocarditis, or, indeed, show any cardiac lesion; and have no trace of rheumatism in their past or family histories. The joint lesions in such cases are not any more rheumatic, in the judgment of the writer, than are the joint pains and swellings which occur frequently in patients who exhibit the signs of simple, of Henoch’s, or of hæmorrhagic purpura.

Thus the purpura of infective origin may be subdivided into three groups: (1) The group of infective fevers of the hæmorrhagic type, in which should be placed those cases where purpura is associated with acute rheumatic fever or with endocarditis of probable rheumatic origin; (2) the group of infections in which the purpura is a constant phenomenon—*e.g.*, typhus fever; and (3) the group of pyogenic infections—*e.g.*, infective endocarditis and septicæmia.

2. *Cachectic Purpura*.—The next main division of the secondary purpuras is that which may be called the “cachectic purpura.” In children these are probably less common than in adults, simply because the diseases which especially tend to excite them are more frequent in adults. Such is, for example, chronic Bright’s disease. In adults it is a common occurrence to meet with purpuric extravasations beneath the skin of the lower extremities in sufferers from chronic Bright’s disease; and the same is true of patients suffering from cardiacosia, from chronic tuberculosis, and from the severer grades of jaundice. In children the purpura of cachexia is chiefly seen in marasmic infants and those suffering from chronic diarrhoea. In such patients the appearance, not infrequent, of a purpuric rash over the chest and abdomen is almost invariably a death-warrant. The writer has twice seen infants recover after showing this sign in a marked degree, and in the present series of cases there is one other recovery; but the phenomenon generally heralds the approach of death.

In this class, also, may be placed the purpura which accompanies the so-called “blood diseases”: the leukæmias, lymphadenoma, scurvy, and anemia pseudo-leukæmia infantum. In these diseases, however, the purpuric spots are rarely more than scattered petechiæ upon the trunk, though more marked manifestations are not unknown.

3. *Toxic Purpura*.—Certain drugs, among them the iodides, antipyrin, and diphtheria antitoxin, may produce purpuric rashes; but these are rare, and the simple knowledge of the possibility of their agency is sufficient to secure their recognition.

4. *Nervous Purpura*.—Again, purpuric spots and subcutaneous hæmorrhages occur occasionally in the course of certain diseases of the nervous system, and the local asphyxias and symmetrical gangrenæ of Raynaud’s disease may be considered as purpuric phenomena.

5. *Mechanical Purpura*.—Lastly there are the purpuric hæmorrhages of mechanical origin, not with, *e.g.*, in pertussis, and occurring in dropical limbs. Therefore, to summarize the secondary purpuras, we may classify them—

- (1) Purpura of inductive origin.
- (2) Purpura of coeliacic origin.
- (3) Purpura of toxic origin.
- (4) Purpura of nervous origin—a doubtful division.
- (5) Purpura of mechanical origin.

II. Primary or Idiopathic Purpura.—When the secondary purpura have been put on one side, there remains a large and important class, which may be called "primary" or "idiopathic" purpura, a name given them to distinguish them from those purpura of which at least the predisposing causes can be traced. In this class only the signs, symptoms, and prognosis are known. Diagnosis must be negative—*i.e.*, rest solely upon the exclusion of all the previously noticed factors, and treatment is purely empirical.

FREQUENCY OF OCCURRENCE.—In a series of cases of children collected for the purposes of this chapter from the records of St. Bartholomew's Hospital and the Hospital for Sick Children, of a total of 96 cases, 28 could be definitely assigned to the one or other of the groups already mentioned. Of the remaining 68 cases, 27 were cases of simple purpura—*i.e.*, where the purpuric manifestations were confined to subcutaneous hemorrhages, generally under the skin of the lower extremities, but sometimes affecting the arms and trunk as well; 19 cases, in addition to subcutaneous hemorrhages, had more or less free bleeding from the mucous membranes of the nose, mouth, urinary and intestinal mucous membranes, and are classed as "purpura hemorrhagica"; 12 cases exhibited the peculiar association of purpura with paroxysmal abdominal pain, diarrhea, and occasionally nephritis, to which the name of "Henoch's purpura" has been given; and the remaining 10 cases could be variously described as "rheumatic" or "arthritic" purpura. If we give the most liberal interpretation to the term "rheumatism," 7 out of these 10 may be accounted "purpura rheumatica"; in the other 3 there was no evidence of rheumatism beyond the swelling and pain of the joints.

Studying the figures a little further, a striking fact emerges. Excluding symptomatic purpura, there are 46 girls and 23 boys affected, as nearly as possible 3 girls to 1 boy, while the cases classed as "hemorrhagic purpura" show a preponderance of 11 boys to 8 girls. What may be the explanation the writer is at a loss to suggest, more especially as he finds that these figures do not correspond at all with those of other series which have been collected, from which it would appear that, taking all ages, purpura is twice as common in males.

It is usual to divide the cases of idiopathic purpura into the four varieties named: Purpura simplex, hemorrhagica, arthritica or rheumatica, and Henoch's. The division is purely arbitrary and artificial, and depends chiefly on the predominance of one or other symptom. Thus, where the joint pains and swellings form a prominent feature of the clinical course of the disease, the patient is said to have arthritic purpura; where there is hemorrhage from the mucous membranes, it is hemorrhagic purpura; and where paroxysmal abdominal pain, diarrhea, hematuria, or nephritis, marks the clinical course, the disease is called "Henoch's purpura." Where none of these symptoms occurs in a marked degree, the case is said to be simple purpura. It is obvious that the dividing lines must be ill-marked, and depend upon the personal bias of the observer, and there is no doubt that in the present series another writer would divide the cases differently.

SYMPTOMATOLOGY.—In describing the symptoms, the attempt has been made to construct a composite picture emphasizing those features which are considered sufficiently distinctive to place the cases in which they occur in one or other division.

The Hemorrhages.—The common symptom which all cases of purpura present, to which the name is due, is the subcutaneous hemorrhage. In the majority of cases these hemorrhages occur early in the illness, but occasionally are preceded by general malaise, fleeting pains in the limbs and trunk, and edema of the legs, feet, or hands. In Henoch's purpura severe and paroxysmal abdominal pain often precedes by some hours or days the appearance of the rash. In rather more than half of the present series the distribution of the hemorrhages was confined to the limbs, especially the lower limbs; while in the smaller residuum the trunk, face, neck, and even the scalp, were covered with the eruption. The more severe the illness, the greater the extent and closeness of the spots; but this rule has exceptions, and some of the most obstinate and prolonged attacks have comparatively trivial eruptions confined to the legs. Nevertheless, it is the rule that in the hemorrhagic cases the subcutaneous hemorrhages are more extensive and more widely distributed than in the simpler attacks. The hemorrhages are usually small rounded spots, not elevated above the surface, which change their colour from the bright red of their first appearance to purple and brown as they fade. Not infrequently successive eruptions present all shades of colour, from a brilliant red to a dingy brown, in the same patient. The colour does not fade on pressure. The absence of elevation of the spots is the rule, but cases are met with wherein the hemorrhages occur in localized patches of edema, and then present the appearance of hemorrhagic papules or wheals. The small round hemorrhagic spots are called "petechiæ"; the lines and streaks of hemorrhage "vibices"; and the larger irregular patches "ecchymoses." In rare instances these larger macular hemorrhages become gangrenous and slough. Such gangrene occurs in cases which have received the name of "purpura fulminans," which is closely allied on the one hand to the hemorrhagic varieties of the infective fevers, and on the other to the rare group of cases of symmetrical gangrene of the extremities (see p. 306).

The hemorrhages occur with great suddenness, the spots frequently recur in crops, and many patients are subject to recurrent attacks. Thus, in one patient, a boy of eleven, there were eight successive eruptions in a period of about three months. In another, a girl of ten, there were four separate distinct illnesses, each with an eruption of purpuric spots within the year. In a third, a girl of five years, there were nine eruptions in the course of three months, in two of which the lesions were articular wheals and papules elevated above the surface of the skin, deeply purple in colour.

In rare instances petechial spots appear under the mucous membranes. In one case in the present series they were present on the gums, on the hard palate, and on the tongue.

Other Symptoms.—This subcutaneous eruption is the only symptom which is common to all forms of the disease; the remaining symptoms appear in varying proportions. Hemorrhages from the mucous membranes of the nose, mouth, urinary and intestinal canals, occur in the hemorrhagic variety of purpura, and also less frequently in the form known as "Henoch's purpura." In the cases of hemorrhagic purpura, bleeding from the bowel and from the nose were the most frequent, one or other occurring in half of the cases. The bleeding is rarely serious,

though its persistence is always a cause of anxiety. In no case in this series was the bleeding sufficient to cause any material loss of strength or colour.

The Blood.—In this connection the condition of the blood may be noted. In those cases where the percentage of hæmoglobin was recorded, the lowest figure was 54 per cent., and the average was 74 per cent., and these included the more severe cases. Once only did the numbers of red corpuscles fall below 4,000,000 in a severe case of hæmorrhagic purpura, while the average count of the leucocytes was 11,700. The highest count was 24,000, of which 19,000 were polymorphonuclear leucocytes, and this was an estimation made after very considerable hæmorrhage had occurred from the nose and urinary tract, and the high count was due to leucocytosis after hæmorrhage. In the differential counts of the leucocytes, there is nothing to note unless it be a tendency to an increase of the eosinophil corpuscles in the more severe cases of hæmorrhagic purpura. It is stated that in severe cases the number of blood-platelets is considerably diminished, but there are no records of this in the present series.

Purpure Simplex.—In purpura simplex, besides the eruption there is often little else to notice. The fever is extremely slight, never above 100° F., and never of more than a few days' duration; the disturbance of nutrition practically negligible; there may be fleeting pains at the onset in the muscles and joints, and there is often some oedema of the affected portions of the skin, sometimes preceding, sometimes accompanying, the eruption. In one case in this series there was at the onset some diarrhoea, and in two other cases, at the beginning of the illness, there was severe abdominal pain. The duration of the affection is from one to three weeks, and in rare instances recurrences of the eruption may delay complete recovery for a longer period.

Purpure Hæmorrhagica.—In the cases classed as "purpura hæmorrhagica" all the above symptoms are repeated, with the addition of bleeding. The fever is apt to be more prolonged, and to run at a higher level, though rarely above 101° F., and usually below this. The duration of the disease is longer, the average being just under three months. Recurrences also are apt to be more frequent. In hæmorrhagic purpura the joint pains are often severe, and effusion into the joints not uncommon. This effusion has never, so far as the writer has been able to ascertain, been found to be bloody. Vomiting is common and often blood-streaked; diarrhoea is rare. Nephritis, usually of a hæmorrhagic type, is a serious, but fortunately a rare, complication. In the thirty-one cases of purpura hæmorrhagica and Henoch's purpura combined, there were only three instances in which there could be said to be a nephritis apart from the hæmaturia, and in one only of these was albuminuria still persisting on discharge. Nephritis is said to be most frequently met with in Henoch's purpura.

Henoch's Purpura.—Henoch's purpura is distinguished from the other varieties only by the severe crises of abdominal pain, often accompanied by vomiting and diarrhoea with bloody stools. In some instances there is a definite arthritis also. Otherwise there is no distinguishing characteristic. The purpuric eruption and the bleeding from the mucous membranes are similar in every respect to those met with in other purpuras. It will be obvious that the division is artificial, and that there must be border-line cases; yet cases of purpura with well-marked abdominal crises constitute a distinct type of the disease. These crises occur in paroxysms. The pain is often extremely severe, causing vomiting and prostration. In cases of these crises, where the bowels are not moved, a diagnosis of intestinal

obstruction has been made; in others, owing to the passage of blood by the bowel, intussusception has been diagnosed; and in both conditions operation has been undertaken. One such recently, in a boy of seventeen, came under the writer's observation. At the operation a length of bowel was found swollen and of a deep red colour. At the extremities this faded off gradually, and showed discrete bright scarlet patches in the wall of the gut. The abdomen was closed, and the patient made a good recovery, but had one other comparatively slight attack of pain, accompanied by a purpuric eruption on the hands and legs. In this series of cases there was no death, but fatal results have occurred from cerebral hæmorrhage and nephritis.

In Henoch's purpura there seems to be an unusual tendency to recurrence. Children may have a large number of attacks in the course of a few years; and though this is also true of the other varieties of purpura, it is much more common with Henoch's purpura. Pratt quotes a case in which in the course of five years there were at least sixty distinct attacks.

The cause of the abdominal pain is unknown. Various conjectures have been made, and the association of urticaria and œdema have suggested that the abdominal colic is due to a localized œdema and extravasation in the wall of the gut, a conjecture to which the condition found at the operation mentioned above lends support.

RELATIONSHIP WITH OTHER DISEASES.—The relationship of purpura and rheumatic fever has been already discussed. The connection between purpura and urticaria, angio-neurotic œdema, and erythema multiforme exudativum, is much more certain and definite. Reference has already been made to the fact that in some cases of purpura the skin lesions resemble urticarial wheals into which hæmorrhage has taken place. But, further, in one case of this series attacks of definite urticaria without purpura alternated over several weeks with definite purpuric eruptions. Osler has emphasized this aspect of the question, and would place the purpura in the same class with the exudative erythemata, especially Henoch's variety. Erythema nodosum, however, which is a typical variety of erythema exudativum, is not usually purpuric, though the difference of appearance is a question of degree rather than of character. Cases are quoted in which purpuric spots and ecchymoses have coincided with a typical attack of erythema nodosum, but they must be uncommon. It is always difficult to gather exactly what an author is thinking of when he speaks of erythema exudativum or erythema nodosum, and there can be no doubt that much of the confusion which exists in this connection is due to the fact that an attempt is made to bring into one common group a number of diseases which will eventually be found to be due to a variety of different causes (*cf.* Chapter III., p. 136).

PATHOLOGY.—What these causes may be there is at present no evidence to indicate. Arguing from the analogy of certain cases of symptomatic purpura, some have believed that purpura is due to a bacterial infection. No evidence is yet in our possession, in spite of many attempts to isolate an infective agent. Blood-cultures were made in several cases of the present series, but in all proved sterile. Others tend to the belief that the hæmorrhages and other manifestations are due to a poison, bacterial or chemical, manufactured in the intestines, but of its existence there is no good evidence. Experimentally purpura has been produced by the injection of the serum of a hæmophilic patient into the spinal canal of a rabbit

whose liver had been injured by a temporary ligation of its bloodvessels. The injection of diphtheria toxin produced similar results. The name "idiopathic purpura" must remain, but only in order to distinguish the purpura of unknown origin from those whose predisposing causes we can at least partially recognize.

PATHOLOGICAL ANATOMY.—In patients who have died of hemorrhage, the extreme anemia of the viscera is the only striking feature. Petechiae are common on all the viscera and beneath the serous membranes. Other hemorrhages are uncommon. Cerebral hemorrhage has been noted, and also hemorrhage into the lumbi marrow. No cases of hemorrhage into the serous cavities are on record. In the intestine and stomach small extravasations are not uncommon, and in one case hemorrhage had occurred in a number of the Peyer's patches. Extensive bleeding into the walls of the gut is very uncommon. The kidneys are a frequent source of blood, but very few examinations of these organs in death from purpura have been recorded. In one case there was a well-marked glomerular nephritis.

DIAGNOSIS.—The diagnosis of the condition is almost always easy. The obvious pitfall is to mistake a symptomatic purpura for an idiopathic case, with a consequent grave mistake in prognosis. It is, however, a mistake which in practice is rarely probable. In one instance the severity of the fever will protect from error; in another the examination of the blood, as in the leukemias; and in a third the prevalence of an epidemic of measles or some other of the infective fevers. Scurvy, again, has features which make for confusion, but here the tenderness of the limbs and the clinical history should render a mistake unlikely. The chief difficulties would appear to arise in connection with the abdominal form, or Henoch's purpura. Here in the early stages of the disease the eruption may be slight, and the abdominal symptoms so severe as to lead to the diagnosis of intestinal obstruction or intussusception. Several cases have been operated upon before the true nature of the attack has been recognized. The difficulty is increased by the fact that at least one case is on record where, during an attack of purpura, an intussusception did actually occur.

TREATMENT.—The treatment of idiopathic purpura can be summed up in a very few words. As yet there is no specific treatment, and, of the various drugs which have been employed, none seems to have any controlling influence. Prolonged rest under the best available conditions, in the great majority of cases, restores the patient to health. The mortality from the disease is very small. In this series of sixty-eight cases there was one death, and that was due, not to the purpura, but to the long-standing heart disease acquired during the rheumatic fever several years previously. The same fact appears in all other series which have been published. There is one exception, and that is in the rare type known as "purpura fulminans." In that disease, which is in reality very slightly connected with the purpura we have been considering, death is invariable, and comes with great rapidity. What these cases really are is unknown, but there can be very little doubt that they represent an acute and virulent infection, and are more closely allied to the hemorrhagic types of the infective fevers than to idiopathic purpura. In patients who suffer from such an attack the purpuric lesions quickly become gangrenous and slough, and in some instances gangrene attacks the extremities symmetrically. With regard to treatment of this affection, none has

proved efficacious. There is, however, a condition to which new-born babies are liable—the hæmorrhagic disease of the new-born—in which Welch has lately shown that the injection of human serum exercises a most strikingly beneficial effect. How it acts is unknown, but the analogy between such cases and the more severe types of purpura leads one to hope that the injection of human serum may prove advantageous in these desperate cases.

(b) **Hæmophilia**.—**DEFINITION**.—By “hæmophilia” is meant a constitutional tendency to uncontrollable hæmorrhage, either spontaneous or arising from wounds or other lesions which in the normal person scarcely bleed at all. It is an hereditary disease of males, and in the majority of cases the tendency is known in the family, and can be traced backwards for several generations.

INTRODUCTION.—The disease is apparently of comparatively recent recognition. The earliest detailed accounts were published in the first quarter of the nineteenth century, and its remarkable family incidence was emphasized by the publication in 1813 of the Appleton family tree. John Hay, the author of this paper, was able to find eighteen “bleeders” among the descendants of Oliver Appleton in six generations. Numerous other families have since been studied, but in the majority of cases the disease seems to exhaust itself or the family to become extinct.

ÆTIOLOGY.—Isolated cases of hæmophilia, though rare, are not unknown; but in many of these instances the family histories are defective. It is more usually the case that the bleeding tendency is known in the family, and that one or more of the child's ancestors has suffered from the complaint, or, where a “bleeder” has been born of healthy parents, that one or more of the succeeding generations is affected.

The peculiarity of the disease lies in the mode of inheritance: the males of the family are alone affected, while the tendency is transmitted exclusively through the females. To this rule there are, according to some authorities, exceptions, but the careful research of Bulloch and Fildes establishes beyond doubt that there is no authentic case of an affected female in a known hæmophilic family; and that none of the reported instances of transmission through the male is entirely free from suspicion of error or omission in the pedigree. The conclusion is that the law, as usually stated, has up to the present no authentic exception.

This mode of inheritance is not unique. There is evidence that cases of partial albinism, in which the eyes are chiefly affected, follow the same law (Nottship); and it has also been observed in cases of colour-blindness and of night-blindness. It has been suggested that these and some similar instances are examples of inheritance which can best be explained on the Mendelian theory, and certain hereditary deformities lend themselves easily to such an interpretation. In the case of hæmophilia, however, though the facts are suggestive, there is not sufficient evidence yet available to determine the scheme of inheritance. “It is possible,” says Punnett, “that we are here dealing with a character which is dominant in one sex, recessive in the other. But the evidence so far collected points to a difference somewhere, for in hæmophilic families the affected males, instead of being equal in number to the unaffected, show a considerable preponderance. . . . Our knowledge of the offspring of ‘bleeding’ males is as yet far too scanty, and until it is improved . . . the precise scheme of inheritance for hæmophilia must remain undecided” (*cf.* *Proboscidea*, II., p. 16).

AGE.—As a general rule the disease manifests itself early in life, usually in infancy. A late onset is rare.

RACE.—It is a disease of Northern climates, unknown in the tropics, and the majority of the reported cases are of Teutonic, English, or American origin. The Latin races are apparently much less frequently affected.

SYMPTOMATOLOGY.—*Hæmorrhage.*—In the vast majority of persons who suffer from hæmophilia, the tendency to bleeding manifests itself early in life. In 60 to 70 per cent. of the recorded cases the disease was recognized before the completion of the second year. The hæmorrhage which establishes the diagnosis may occur spontaneously, but more usually follows slight wounds or injuries. The newborn infants of hæmophilic families very rarely suffer. Most of the recorded instances rest on insufficient evidence. Circumcision, however, has often led to the recognition of the disease, and in some instances the bleeding has proved fatal. In a few instances where bleeding has occurred early in life in a member of a hæmophilic family, the onset of puberty has put a term to the tendency, and no further manifestation has occurred; but as a general rule, although the severity of the hæmorrhage may vary at different periods of life, the tendency remains unaltered throughout life.

The special characteristic of the bleeding of hæmophilia is its persistence rather than its severity. It is rarely the case that hæmorrhage is profuse enough to cause immediate danger to life; the danger lies in the fact that all efforts to stop the slow continued loss may prove abortive.

Spontaneous hæmorrhage in hæmophilia is not nearly so common as traumatic; indeed, it is doubtful, considering the slight nature of the lesions which are sufficient to cause it, whether all hæmorrhages should not be classed as "traumatic." The evidence in favour of the existence of spontaneous hæmorrhage lies chiefly in the facts that in certain cases prodromal symptoms, giddiness, palpitation, sweating, or dyspnoea, precede the attack; and, secondly, that visceral hæmorrhages are occasionally met with in hæmophilic individuals.

Site of hæmorrhage.—The majority of hæmorrhages occur either from the skin or the mucous membrane of the mouth or nose. The hæmorrhages beneath or into the unbroken skin are chiefly in the form of ecchymoses. Small purpuric spots are decidedly uncommon. Oozing of blood from the unbroken skin of the finger-tips has been reported. In most cases, however, the bleeding proceeds from some slight injury—a superficial cut or a scratch.

Epistaxis is the most frequent phenomenon among the hæmorrhages of the mucous surfaces; and next in order comes oozing from the gums or from a tooth socket. Some patients cannot use a toothbrush, because it excites bleeding, which they find it difficult to stop.

Hæmorrhage from the lungs, the stomach, the intestines, the urethra, and the kidneys, occur much less frequently.

Another form of hæmorrhage must be mentioned because of the danger of a mistake in diagnosis. This is the subcutaneous hæmatoma which may present all the appearance of an acute abscess, and has been incised as such, with fatal results. These hæmatomata occur most often on the lower extremities.

But the most remarkable form of hæmorrhage is that which occurs into the cavities of the joints. The joints most often involved are the knees and the elbows, but any of the joints in the body may be affected, the pre-eminence of the two

mentioned being due to their special liability to injury. The swelling and effusion take place often with great rapidity, and with a corresponding amount of pain. The effusion may be absorbed fairly quickly, and the joint restored to complete mobility; or, owing to the slow absorption or to repeated extravasations, contraction and ankylosis may cripple the patient permanently. In the stage of effusion the affected joint may be painful, tender, hot, reddened, and the surrounding tissues swollen, with fever of 101° or 102° F. In some instances, however, the swelling is a "white" swelling with but slight pain or tenderness. At this early stage the fluid is almost pure blood. As absorption proceeds, the fluid becomes a dirty brown colour, which stains the synovial membranes and cartilages a yellowish or yellow-red tint. The cartilages become softened and eroded, and in long-standing cases rarefaction and atrophy of the bony surfaces occur. In a few cases osteophyte formation has been described, but this is certainly rare. Fibrous ankylosis of the joint, owing to the contraction of the inflamed structures and their adhesion, is much more common. Contrary to what might be expected, it is rare to find any discoloration of the skin over the joint, and the absence of this has led some observers to express the belief that the effusion is serous rather than bloody—a belief contradicted by the actual fact.

Muscular hæmorrhages and extravasations into the serous cavities are among the least common manifestations of hæmophilia.

GENERAL SYMPTOMS.—The majority of the symptoms of general disturbance are not the effects of the constitutional malady so much as the result of the anemia induced by the hæmorrhage. Thus, cardiac murmurs are not uncommon, but usually disappear with the restoration to full health. The goldiness, headache, palpitation, and excitability, which have been already mentioned as prodromal symptoms of spontaneous hæmorrhage, may quite possibly be better interpreted as the effect of small local hæmorrhages which precede the more severe loss. Periodicity of the attacks has been noted in some cases, the intervals becoming longer as the patient grows older; and Wright records his belief that the bleedings are much more apt to occur at night than by day. The same author notes, also, that the sufferers from the disease not infrequently have depraved appetites, and are ready to eat dirt and grit of all kinds—a feature observed also by Wickham Legg.

PATHOLOGY.—In children who die of the hæmorrhage, the chief alterations found are due to the draining of blood from all the organs. With this exception there is no constant anatomical change, which can be said to bear any relation to the disease, though in a few cases abnormal thinness of the arteries, causing them to resemble veins, has been noted.

The Blood-Changes.—There is generally a slight diminution in the number of leucocytes, especially of the polymorphonuclear leucocytes, but beyond this the changes are transitory. After a severe hæmorrhage the red corpuscles are quickly restored to their normal number, and the hæmoglobin much more slowly; so that an appearance of anemia may be present for many weeks, or even months.

As to the coagulation time of the blood of hæmophilia the statements are conflicting. Wright believes that the coagulation time is generally much prolonged beyond that of normal individuals. Other authorities have been able to demonstrate so marked difference, either during an attack or during an interval. Salké believed

that the coagulability was diminished in the intervals, normal or increased during the height of an attack. In the writer's opinion, after trial of various methods of estimation, these differences are due to the fact that we do not yet possess a trustworthy method; all those hitherto employed are subject to fallacies which entirely vitiate the results.

Various hypotheses have been propounded to explain the abnormal tendency to hæmorrhage. Abnormal fragility of the vessel walls, observed by Virchow in a single case, for some time held the field, supported by the weight of his authority against the accumulating weight of facts. Immersman postulated a high blood-pressure with a small volume of the bloodvessels, whereas the blood-pressure in such cases as have been accurately observed is normal, or even low. W. Koch maintained that hæmophilia, together with scurvy and purpura, is an infective disease—an hypothesis quite improbable and wholly unsupported by facts. Sahli believes that the malady is the result of a chemical alteration in the walls of the bloodvessels, which results in the failure of a substance (thrombokinase) which is essential to the formation of a clot—an hypothesis which offers a plausible explanation of the observed fact that clotting may take place in the extravasated blood, while bleeding still goes steadily on beneath it. There must be, however, as Sahli recognizes, a further factor, since this will hardly account for the extreme rapidity, for instance, of the effusions into joints; and this factor must be assumed to be an abnormal permeability of the vessel walls, which cannot be recognized by our present methods of examination.

DIAGNOSIS.—The diagnosis of hæmophilia must rest to a large extent upon a knowledge of the family history. Where there is no known "bleeder" in the pedigree, the diagnosis of hæmophilia is always open to criticism. It must be remembered that there are other causes of repeated and obstinate hæmorrhages than hæmophilia—that, for instance, in recurrent idiopathic purpura—such hæmorrhages are met with, and that effusions into the joint cavities occur in both diseases. In hæmophilia, however, the effusion is bloody; in purpura it is invariably serous. It should be added that the needle-prick requisite to make the diagnosis of the nature of a joint effusion is not in hæmophilia a source of bleeding; even a vein may be punctured with impunity. The real difficulty occurs in the fact that in known hæmophilic families a single isolated hæmorrhage—*e.g.*, epistaxis—may be the only evidence that the child is a "bleeder"; or without any of the liability to obstinate hæmorrhage from slight cuts there may be a recurrent hæmatoma. These cases have been called examples of "local hæmophilia," but they certainly cannot be included as manifestations of the typical constitutional malady until we can find some trustworthy criterion. Such a criterion Pratt believes may exist for some cases in the estimation of the number of blood-platelets per cubic millimetre. In hæmophilia these are normal—*i.e.*, about 450,000 per cubic millimetre in number—whereas in purpura they are greatly reduced; to 50,000 or lower. The writer has no personal experience of this feature.

Another point to be remembered in the presence of an obscure joint effusion or of a hæmorrhagic extravasation is that the knowledge of the prevalence of the "bleeding" disease in a family is often not considered worth mentioning, or may even be concealed intentionally. The hæmorrhagic effusions of infantile scurvy bear no resemblance to those of hæmophilia, being chiefly round the shaft of the bone, and leaving the joint unaffected.

The spongy and bleeding gums of acute leukaemia are sometimes associated

with an exaggerated tendency to ecchymosis and with epistaxis, but in these cases the briefest examination of a drop of blood reveals the true condition.

PROGNOSIS.—As regards life, the prognosis is on the whole bad in childhood. From 50 to 60 per cent. of hæmophilic patients die before reaching their eighth birthday, and less than 12 per cent. survive to puberty (Littar, V. Ellinger). These figures probably refer to the definite, well-recognized cases in which repeated hæmorrhages occur, and in any individual the prognosis as to life must depend almost entirely upon the severity of the hæmorrhages. The first manifestation rarely kills. In those patients who survive, the joint troubles are likely to cause much inconvenience, unless the prevention of contractures is secured by appropriate treatment.

TREATMENT.—1. *Prophylaxis.*—In a hæmophilic family in whom the females are known to transmit the disease, marriage should be discouraged. Such advice, however, is rarely followed, as the records of the various "bleeder" families show.

Operation, even of the slightest character, should be so far as possible avoided, and, if necessary, should be performed only after the most careful preparation, with the means to stop the hæmorrhage all to hand. Similarly, education and the forms of physical exercise must be of a special kind, devised to eliminate so far as possible the risk of traumatism.

Residence in warm climates is beneficial, and sea-bathing and general tonics, such as iron, quinine, and arsenic, will do something towards the improvement of the general condition of the patient.

Medicinal prophylaxis is at present purely empirical. The older remedies, preparations of lemon, and the dilute inorganic acids, are probably of no benefit. Calcium salts, the chloride and the lactate, have in the hands of some physicians appeared to act beneficially, while others have found them useless. Wright advises the intermittent use of calcium lactate in doses of 15 grains, holding that, after a certain amount of the calcium salts have been absorbed, the tendency to delayed coagulation will be increased, but in many cases the best effects have been obtained by uninterrupted administration, sometimes in larger doses. Ovarian extract has been given with good results in one case, and Wright advises the use of thymus extract and of yeast to increase the nucleo-albumins of the blood and the number of leucocytes.

2. *Treatment of Hæmorrhage.*—Where the site of the hæmorrhage is accessible, pressure long continued remains the best means of controlling the oozing. The gauze compress may be soaked in a solution of calcium chloride. Gelatine applied locally is harmless but inefficient; given subcutaneously it is inefficient and dangerous, because of the difficulty of sterilization and owing to the necessity of puncture, with the risk of starting fresh bleeding. Wright recommends in desperate cases the administration of CO_2 gas, which promotes rapid coagulation, and the employment of a "physiological" styptic, which should be made from fresh thymus gland with the addition of calcium chloride. These two measures will, he claims, nearly always control the bleeding for the moment, but when the clot formed by this means is removed fresh hæmorrhage is likely to take place. Lastly, the subcutaneous inoculation of normal serum is advocated, with a view to supplying to the body the substance the lack of which causes the delay in coagulation.

In bleeding from the internal organs a subcutaneous injection of pituitary extract, which has a most powerful effect in contracting the bloodvessels, might be of advantage. In bleeding from the kidneys this drug is to be avoided, for, unlike adrenalin, it causes dilatation of the renal vessels.

Summary of Treatment.—Place the patient under the most advantageous conditions possible in regard to climate, surroundings, and general hygiene. Administer thyroid extract in doses of 2 to 3 grains, and of calcium lactate in doses of 15 to 50 grains, two or three days in each week. Avoid any necessary surgical operation until the patient has been prepared in this way, and has been given subcutaneously a dose of 25 cubic millimetres of serum. Lastly, when hemorrhage is actually in progress, give subcutaneously $\frac{1}{4}$ cubic centimetre of pituitary extract, representing 0.2 gramme of infundibular extract. Use compresses soaked in adrenalin 1 in 2,000, or in calcium chloride solution, and employ the additional aids recommended by Wright, of the inhalation of CO_2 gas and the use of the "physiological" stryptic.

Avoid altogether the use of ferric chloride or of the caustery; both are inefficacious and make matters worse.

REFERENCE.

BULLOCK AND PILSON: *Treasury of Human Inheritance*, parts v. and vi. University of London, Biological Laboratory Memoirs, 20.

This work contains a complete bibliography to 1909, with pedigree charts of all the known hæmophilic families.

LYMPHATISM (STATUS LYMPHATICUS).

INTRODUCTION.—In 1889 Paltan drew attention to cases of sudden death occurring in infants, who were found post mortem to have a general hyperplasia of the lymphatic structures throughout the body. During the last ten years many articles on the subject have been published in America, England, and on the Continent dealing with the subject, but it cannot be said that any light has been thrown upon the causes which lead to the condition. In the absence of this knowledge, a definition of what is meant by the term "lymphatism," or "status lymphaticus," must of necessity be based upon the appearances met with at the post-mortem examination. Lymphatism is said to be present when the lymph glands and the lymphatic structures of the tonsils, pharynx, and alimentary tract, are obviously enlarged, and when the spleen and the thymus gland are hypertrophied. In marked instances of the condition there is no difficulty in its recognition, but the dividing line between what is physiological and what is pathological is obviously obscure, depending upon the individual experience and judgment of the pathologist. (See also Chapter X., p. 579.)

Ætiology.—The chief sufferers from the condition are children between the ages of one and four years, but lymphatism has been said to exist in persons considerably older, past the age of puberty, and even in adult life. There does not appear to be any sex or race predominance, nor any seasonal or geographical factor common to the cases. The condition has been found in children who have died in the course of other diseases, and also where no other sufficient cause of death could be traced. Of late years it has acquired considerable importance

both from the medico-legal point of view and from the liability of patients affected to death while under the influence of an anæsthetic.

SYMPTOMATOLOGY.—Lymphatism is often quite unsuspected during life, the symptoms which occur in these patients who are found post mortem to suffer from the condition being of a general character, which may easily be attributed to other causes. Thus, the patients are often markedly anæmic in appearance, fat and flabby; the spleen may be felt to be enlarged; there may be a history of attacks of dyspnoea, and the palpable groups of lymphatic glands may be moderately hypertrophied. But any or all of these symptoms are constantly observed in children who cannot be described as suffering from lymphatism. The feature which is the most markedly characteristic of status lymphaticus is undoubtedly the sudden failure of vitality, sometimes occurring in the course of administration of an anæsthetic to a child who is apparently in good health, sometimes occurring with dramatic rapidity in children lying quietly in bed, who die without uttering a sound or with merely a few struggles. In such cases the general hypertrophy of the lymphatic structures of the body, including the spleen and the thymus gland, are the marked and often the only pathological appearances. The names "thymus death" and "thymic asthma" sufficiently indicate the association.

Generally it is not possible to detect the enlargement of the thymus gland during life with any certainty. In a few instances it can be felt as a distinct tumour in the suprasternal fossa; and, again, the area of impaired resonance which normally is found over the mammary sterni, from the sterno-clavicular articulations to the upper border of the third rib, may be demonstrably enlarged to either side. When the condition has been suspected, a radiographic picture has occasionally shown the presence of the enlarged gland.

PATHOLOGY.—In a child who has died in the manner above-described, the principal morbid anatomical appearances are the presence of an abnormally bulky and heavy thymus gland, and the enlargement of the Peyer's patches and solitary follicles of the intestines. These two features are always present. Less constant are the enlargement of the various lymphatic glands, cervical, axillary, mesenteric, and inguinal; the presence of subpleural and subpericardial hæmorrhages; the dilatation and muscular degeneration of the heart; and the empty condition of the great veins and cerebral sinuses.

The thymus gland has of recent years been carefully weighed in a large number of children of all ages, and the net result would appear to be that, while, as the text-books of anatomy state, it attains its greatest weight about the age of two years,



FIG. 51.—HYPERTROPHY OF THE THYMUS GLAND FROM A CHILD AGED THREE MONTHS WHO DIED ATGENEV. NO THYMIC SYMPTOMS.

(From the Museum of St. Bartholomew's Hospital.)

this weight has been much exaggerated, and seldom reaches more than 9 grammes instead of the 25 to 50 ordinarily stated (Dudgeon, Thorsfield). Hence any weight above 10 grammes must be regarded as abnormal. The gland itself, both macroscopically and microscopically, appears normal, and there is no evidence on record to show that there is any chemical alteration in its constitution. Experimentally, the juice of the thymus gland injected into the peritoneal cavity of guinea-pigs produced paresis of the hind-legs, followed by sudden death (Hart).

The other marked features at the autopsy are the general enlargement of the lymphatic tissue in the nose, fauces, tonsils, pharynx, alimentary tract, and lymphatic glands. In these situations, also, the enlargement appears to be due to a simple hyperplasia. In a few rare instances compression of the trachea has appeared to play a part in the causation of death, but far more often the trachea is normal. The liver is nearly always in a condition of fatty degeneration. Bacteriological examinations have hitherto failed to reveal any infections either of the blood or tissues of the body. The cause of death, therefore, in these cases remains undetermined; but the prevailing tendency is to regard them as due to a toxæmia of unknown origin.

DIAGNOSIS.—In a child who is of markedly pale or sallow complexion, of fat or flabby habit of the body, and in whom there is a history of laryngeal spasms or attacks of dyspnoea, the possibility that lymphaticæ is present should always be held in mind. In such a patient careful percussion over the upper 2 inches of the sternum and neighbouring spaces will sometimes reveal an abnormal area of impaired resonance, and whenever possible a radiograph should be taken in the endeavour to confirm the diagnosis. In the majority of instances, however, there is no suspicion of the real condition of the child.

PROGNOSIS AND TREATMENT.—Where the condition is suspected to exist, the first consideration is to avoid any occasion for the induction of anaesthesia. If this is unavoidable, it is recommended by Buxton that a low-strength vapour of chloroform in the presence of oxygen should be employed. In the absence of any knowledge of the cause, treatment of the condition must be symptomatic, directed to the removal of the anaemia and the improvement of the muscular tone by massage, open air, and abundant diet.

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LYMPHADENOMA.

INTRODUCTION.—In 1832 Hodgkin, of Guy's Hospital, described a peculiar disease characterized by a progressive enlargement of the lymph glands in various parts of the body, associated with an enlargement of the spleen, often of great degree. At the autopsy the spleen was found studded with firm white deposits, of different shapes and sizes, giving to the organ the appearance of the almond-studded cake which is known by the name of "hardtack." For many years the subject was entirely neglected, and, though from time to time writers both in England and on the Continent described cases of the disease, there was no general

recognition of the condition until the decade 1870 to 1880. The publication of Droschfeld's and Sternberg's papers in 1892 and 1898 did much to draw attention to its peculiarities, and of late years the investigations into its histology and ætiology have led to a recognition of the characteristics which separate it from a number of conditions which clinically resemble it closely. It is not, however,



FIG. 52.—LYMPHADENOMATOUS SWELLING FROM A BOY AGED SEVEN YEARS.
The drawing shows the typical "hardlike" appearance. Weight, 3½ ounces.

easy even now to frame a definition which will be an adequate description of the disease, and at the same time differentiate it from a number of somewhat similar conditions. The nature of the difficulty will be best appreciated by a consideration of the variety of names allotted to it: lymphadenoma, lympho-sarcoma, malignant lymphoma, pseudo-leukæmia, the favourite name hitherto on the Continent, and, lastly, lympho-granulomatosis. It is a disease of a chronic character,

beginning with an enlargement of the lymphatic glands, usually in the neck, and progressing steadily, with enlargement of the spleen and hypertrophy of much of the lymphoid tissue of the organs, to a fatal termination. After death the histological changes which can be demonstrated in the structure of the glands are typical of the disease, and in nearly all cases the macroscopic characters of the spleen are unmistakable. Anæmia is usually present in the later stages of the disease, but in the beginning there is often no abnormality in the blood, and there is never at any time a characteristic alteration which would indicate the nature of the disease, with as we find, for example, in leukaemia.

Ætiology.—Distribution.—The disease is widely distributed wherever accurate reports of patients are kept in Europe, Asia, and America, but is nowhere common. It is said to occur in animals—dogs, horses, and pigs—but the records are unsatisfying.

Frequency.—It is not a common disease, but probably more so than was formerly supposed. Thus in ten years at St. Bartholomew's Hospital over 100 cases have been admitted to the wards, medical and surgical and of these 30 were children under twelve years of age. Similarly, in ten years at Great Ormond Street, in the medical wards alone, there have been 13 cases in which the diagnosis was definitely established, besides a few others in which it was, while not certain, in the highest degree probable. These 13 cases occurred among 15,000 patients admitted, representing a percentage of all diseases of 0.1.

Sex.—The disease is far more frequent in male children than in female. Only two of the thirteen Great Ormond Street cases were females.

Age.—The youngest patient in this series of forty-three cases was aged just over three years; but the average age was seven and a half years, and it is certainly much more common after the period of second dentition.

Predisposing Causes.—The condition sometimes appears to follow directly upon some infective disorder, especially of the throat or nose; and since the cervical glands are usually the first to become enlarged, it is *a priori* reasonable to suppose that their enlargement is due to the entrance of an infective agent from these regions; but so long as the infective agent is unknown, such a supposition is incapable of proof, and there are too many exceptions in the clinical records to permit us to speak of local inflammation as a predisposing cause.

There is no hereditary or family influence recognizable, though a few instances have been placed on record of more than one case in a family.

The *essential cause* is quite unknown. Bacteriology has failed to find it, unless the recent discovery by Fraenkel and Much of an acid-fast organism in lymphadenomatous tumours be confirmed. These authors believe that the organism is a specialized form of the tubercle bacillus.

Relation to Tuberculosis.—A patient may suffer at one and the same time from lymphadenoma and from tuberculosis; but in such cases the lesions of the two diseases can be definitely distinguished from each other, and the association of the two is by no means so frequent as was at one time believed. Sternberg in 1895 tried to establish the position that Hodgkin's disease was a form of tuberculosis. His views have occasionally found favour, but a bulk of histological evidence has been accumulated against this hypothesis, and Sternberg himself no longer holds his former opinion, but believes that lymphadenoma is an infective granuloma of the same class as tuberculosis, but not identical with it.

Experimental inoculations have resulted in the development of tuberculosis, but only rarely; and, in spite of the claims of various authors, it may be confidently

stated that no method of experimental inoculation has as yet produced in an animal a disease in the least resembling lymphadenoma.

The relation between lymphadenoma and syphilis is certainly not close; the histological resemblances are few, and the general course of the disease is quite unlike any form of syphilitic infection.

SYMPTOMATOLOGY.—The first symptom to attract attention is nearly always an enlargement of the cervical glands. Out of thirty-three cases which the writer has collected, this was the earliest symptom in twenty-eight. In one the femoral glands were enlarged before any others; in two jaundice and in two abdominal swelling due to the enlargement of the spleen, were the causes of complaint. In those early stages as a rule there is nothing else to attract the attention. The patients are not exceptionally anæmic or unduly ill. The swollen glands at first grow slowly and quite painlessly. They are soft, discrete, not attached to the skin or to the deeper structures, and cause no distress to the patient. As a general rule the swelling occurs first in the superficial cervical glands above the clavicle or behind the ear, and the submaxillary group is involved later. When first seen, careful examination can seldom detect enlargement in any other groups of glands, and in the majority of cases it is months, or even years, before the disease has progressed to a general lymphoid hypertrophy. The swelling may be present without impairment of the general health for some months before any other signs present themselves; or, less commonly, other groups of lymphatic glands may rapidly be involved. In the more slowly progressive cases the glands become harder, and inflammation in the capsule tends to mat them together and to the surrounding tissues. The disease appears to spread by direct extension from its original site. Thus, hypertrophy in the cervical region is followed by hypertrophy in the axillary and in the mediastinal glands, passing downwards along the aorta to the retroperitoneal group. This appearance, however, is no doubt deceptive, because in many cases the spleen and the liver become enlarged before the glands are notably involved.

When the disease is fully established, the patient is usually markedly anæmic in appearance, with masses of glands on both sides of the neck, with an enlarged abdomen, and often with œdema of the legs. If the mediastinal glands press upon the trachea, there is great respiratory distress, and in some instances tracheotomy becomes imperative. The spleen at this stage is always markedly enlarged, but seldom reaches the enormous size present in leukaemia. It is hard, and in some cases the irregularities of its surface can be detected by palpation. The liver is bulky, and its edge can be felt rounded and firm 2 inches or so below the costal margin. Both the liver and spleen are as a rule free from tenderness on palpation.

Pressure symptoms which may be met with include dyspnoea, cyanosis, tachycardia, jaundice, ascites, and paraplegia from compression of the spinal cord.

The *fever*, which is a marked feature of the disease, is of three types: (1) Continuous, which, when considered in conjunction with the splanic enlargement and the swollen abdomen, may produce a marked resemblance to a case of typhoid fever; (2) intermittent, a frequent type; or (3) remittent. The intermittent fever is periodic, the fever lasting ten to twelve days, followed by an afebrile period of variable duration, which is again succeeded by pyrexia. The fever in these attacks often reaches 103° or 104° F. Such a chart is characteristic of the more advanced stages of lymphadenoma.

The *blood* in the early stages shows no alteration of any kind. The red corpuscles

are well up to the normal number, the white corpuscles are unchanged either in number or character, and the percentage of hæmoglobin is about the average. When anaemia begins to assert itself, the change takes place first in the hæmoglobin, which becomes diminished. The red corpuscles also quickly diminish in numbers, and often exhibit variations in shape, size, and staining reaction. The white corpuscles at the same time remain unaltered. In twenty-five counts, twelve times the count was below 10,000, eleven times between 10,000 and 15,000, and only twice above that number. The lowest count was 1,500. The average of twenty-five counts made at all stages in the disease, and at all ages under twelve, among thirty-three cases, was 10,400; and the only marked alteration in the proportionate numbers of the different varieties was that occasionally the polymorphonuclear cells were increased. Some authors have stated that a relative increase in the numbers of the small lymphocytes is common, but that is certainly not the case in children. Cases are on record, also, where there was a definite eosinophilia, but there was no such case recorded in this series. It must be repeated, and with emphasis, that there is no alteration of the blood-picture which affords any aid in the diagnosis of lymphadenoma, except, of course, in so far as it serves to exclude the presence of leukaemia.

Blood-cultivations, even in the febrile periods, are sterile.

Other symptoms of inconstant occurrence are a slight degree of albuminuria; oedema of the legs, scrotum, or back, in the later stages; pleural, or more rarely peritoneal, effusions; and, lastly, a symptom of great interest, but of great rarity, the occurrence of true lymphadenomatous deposits in the skin. Such tumours are small, shotty nodules lying sometimes in, sometimes just beneath, the true skin, which in a few cases have become, in the late stages, purpuric, owing to extravasated blood. Pigmentation of the skin is usually due to the arsenic which has been prescribed, but has been seen in cases where this drug was not employed.

COURSE.—If we now construct a picture of the usual course of the disease, we shall find that at or after the period of the second dentition there appears a painless swelling of the cervical lymphatic glands. This swelling is at first accompanied by no other symptoms of disease, and the patient is brought for advice more from the æsthetic point of view than from any idea that the complaint is a serious menace to life. In some cases, however, it is not the cervical glands alone that are involved; examination reveals other glandular enlargements, or perhaps the spleen may be felt unduly prominent. If, as often happens, such glands are excised, as a general rule even before the healing of the wound fresh hypertrophy has occurred in neighbouring glands. At this period there may be a remission of the disease; with or without treatment the glands become smaller, and there is no visible deterioration of health. After a period more or less prolonged, but seldom exceeding eighteen months to two years, the disease, which has in the meantime insidiously progressed, becomes widely generalized, and the patient presents a picture not easily forgotten. Immense masses of enlarged glands deform the neck, and often the axillæ and groins; the respiration is difficult owing to the pressure in the mediastinum; the abdomen is swollen by the size of the spleen and liver, and often of the mesenteric glands; the axilla is marked, and the fever often high; there may be in addition distended veins on the chest, abdomen, or thighs, from pressure on the great veins. Death follows soon from exhaustion, respiratory difficulties, or failure of the circulation. Hemorrhage from any source is extremely rare.

PATHOLOGY.—The disease affects the lymphatic glands, the spleen, liver, and lungs, in the order named; and in addition lymphoid tissue, wherever it normally occurs, may be hypertrophied. Thus, there have been found masses and deposits in the stomach, intestines, pericardium, pleura, bones, and skin. On section of a gland, the surface is homogeneous, smooth, and pinkish-grey, or in some cases traversed by firm bands of connective tissue. Suppuration or necrosis is extremely uncommon. Microscopically the normal appearance of a lymphatic gland is lost; the section appears homogeneous, no longer showing the germinal follicles or the distinction between cortex and medulla. The number of the lymphocytes, which usually obscure the reticular endothelial structure of the gland, is apparently much diminished; and, conversely, the endothelial reticulum is greatly exaggerated, and in the finer glands interspersed with abundant and irregular fibrous tissue. The endothelial cells are large and numerous, and in a typical instance many of them are multinucleate, the nuclei apparently piled one upon another near the centre of the cell. These cells are most numerous in the soft glands of rapid growth, but can be found even in the most chronic cases. Eosinophil cells are said to be present in great excess, but on this point there is considerable divergence of opinion; and it is certain that they are by no means a constant feature. The microscopic histology of the spleen is of the same character, and the deposits in other organs are very similar. In the liver the deposits are most apparent in the neighbourhood of the portal canals and beneath the capsule, in the lungs around the bronchi and beneath the pleural membrane.



FIG. 32.—SECTION OF A LYMPHADENOMATOUS GLAND, SHOWING THE EXAGGERATION OF ENDOTHELIAL CELLS AND RETICULUM.

Hyaline degeneration of the enlarged lymphatic glands is not uncommon, and is of importance because such a change is extremely rare in any other disease.

DIAGNOSIS.—The diseases with which lymphadenoma may be confounded are tuberculosis, lympho-sarcoma, leukaemia (especially of the lymphatic type), typhoid fever, and acute inflammatory conditions of the lymphatic glands. The chief difficulty is to distinguish the early stages of lymphadenoma from tuberculosis of the lymphatic glands. The main distinctive feature of tuberculous glands is that they are as a rule matted together, and form a nodular firm mass, whereas the glands of Hodgkin's disease are soft, discrete, and movable. But in some cases these characteristics are ill-defined, and there are instances in which clinical examination will fail to distinguish the two diseases. Again, the stage of more generalized distribution of the glandular enlargement, with splenomegaly, is occasionally simulated by miliary tuberculosis or by the rapid disease degeneration of tuberculosis. In both these conditions there may be rapid enlargement of the lymphatic glands all over the body, with swelling of the spleen and liver, and continuous or remittent fever. Such cases can be diagnosed only by the course

of the disease; or, better, by excision of a superficial gland, when the histological structure will at once establish the diagnosis.

Next to tuberculosis, the greatest difficulty is to recognize the rare cases of *sarcoma of the lymph glands*. Here swelling of the spleen is rare; the liver is, on the contrary, often considerably enlarged; jaundice is an early, not a late, symptom; and it is usual for the disease to run a far more rapid course. The tumour masses themselves infiltrate surrounding tissues, and therefore quickly become fixed and immovable.

Lymphocytic leukaemia, which presents as its chief features enlargement of the lymphatic glands associated with swelling of the spleen, is quickly differentiated by a blood-count; and besides has other features, especially the tendency to hæmorrhage, which mark it off sharply from Hodgkin's disease.

The diagnosis from *typhoid fever* and from *acute milium tuberculosis* is seldom doubtful—only in those rare cases of lymphadenoma where the superficial lymphatic glands are little, if at all, enlarged. Widal's reaction is the first, and the history is both, are the best guides to accuracy.

The relation of Hodgkin's disease to the so-called "lymphatomas" is much discussed, but it is probable that the increasing knowledge of the histology of the various swellings of the lymphatic glands will diminish the number of the alleged varieties. It is at least doubtful whether there are any diseases except lymphadenoma, tuberculosis, lymphocytic leukaemia, sarcoma, and inflammatory conditions, which produce a large and persistent tumour of the lymphatic glands. Such a diagnosis as "lymphoma" is no longer permissible.

In the diagnosis of microscopic sections of the excised glands, stress must be laid (1) on the presence of the typical multinucleate cells, (2) on the heterogeneous character of the section, (3) on the presence of hyaline degeneration. The chief difficulties are to distinguish the lymphadenomatous gland from the gland of chronic inflammation, and from the gland which is the seat of the type of tubercle characterized by endothelial proliferation. In a few cases, also, the new growths which originate in lymphatic glands may lead to error, owing to the presence of large lymphoid cells arranged in masses. On the whole, however, mistakes are infrequent.

Blood-examinations, except in so far as they serve to exclude leukaemia, are not helpful.

PROGNOSIS.—From the earliest swelling to the fatal termination is usually a period of two to three years. Instances are on record of longer duration, up to seven years; and, on the other hand, there are cases which may fairly be called "fulminating," the whole disease lasting but a few weeks. The shortest duration in the present series was fifteen weeks.

In the more chronic cases there occur periods in which the disease makes no progress, and false hopes are raised, soon to be dispelled, for these periods seldom last more than a month or two. Probably no case of true lymphadenoma has recovered. None of the reported cures rests on an undoubted diagnosis.

TREATMENT.—No treatment has hitherto proved of any avail. Arsenic has been the drug most often employed, and with its use temporary improvement in the size of the glands often occurs. Surgical treatment is of no avail. In most cases it does not even prolong life. X-ray applications undoubtedly cause the glandular swelling to diminish, but only temporarily. It is possible that they

may be of avail to hold in check the pressure effects which produce such terrible results. Of the newer remedies, such as radium and the Ehrlich-Hata drug, it is too soon to speak, but the outlook is not at present hopeful until we can obtain some knowledge of the nature of the causative agent.

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DISEASES OF THE LYMPHATIC VESSELS AND GLANDS.

INTRODUCTORY.—The relation of the lymphatic vessels and glands to the other structures of the body is such that the affections to which they are liable are in nearly every case secondary to disease elsewhere. The few primary pathological conditions are rare, and for the most part clinically unimportant. It will be sufficient briefly to consider (1) affections of the thoracic duct, (2) of the smaller lymphatic vessels, and (3) of the lymphatic glands.

1. Disease affecting the Thoracic Duct is rare at any age, but exceptionally met with in children. The symptoms are obscure, the cardinal sign being the development of chylous ascites or pleural effusions, or more rarely of chylouria, due to obstruction of the duct. It is agreed, however, that in cases where the obstruction develops slowly a collateral circulation may be established, and these cardinal signs never appear. Further, milky effusions into the pleural and peritoneal cavities are not all chylous; the majority seem to be pseudo-chylous, and seldom or never associated with disease of the duct. The distinction between a true chylous and a pseudo-chylous effusion is often very difficult, and in any case must be determined by an exhaustive chemical examination.

The chief causes of obstruction to the thoracic duct are involvement in new growths or pressure from tumours, involvement in fibrous tissue, and thrombosis in the left subclavian vein; and to these must be added the rare cases of rupture or injury in the course of operation.

Schöberg and Wallis in an extensive research on chylous and pseudo-chylous effusions record only three cases, in children below twelve years of age, in which the thoracic duct was definitely involved. In two of these sarcomatous growths blocked the main trunk of the thoracic duct, and in the third an injury to the chest wall had caused a rupture of the duct. Pseudo-chylous effusions, on the other hand, they found to be much more common; but in these there was no evidence that the thoracic duct was injured or diseased.

Thrombosis in the left subclavian vein blocking the orifice of the thoracic duct has been several times recorded, but not in a child.

Lastly, there are a few cases recorded in adults in which an acute purulent inflammation of the main duct has followed an intestinal infection; but the writer cannot find that such a condition has been observed in children.

2. Affections of the Smaller Lymphatic Vessels.—(a) **LYMPHANGITIS.**—Acute inflammation of the lymphatic vessels is never a primary affection, but always due to the presence of an infected focus in the area drained by the affected vessels,

It manifests itself by the appearance of red lines in the skin running from the infected focus towards the nearest lymphatic gland, and in the severer cases is accompanied by cellulitis. Tuberculosis and malignant disease are often propagated by the lymphatic vessels, and in some cases the thickened cords can be felt during life.

(b) Diffuse dilatation of the lymphatic vessels is called LYMPHANGIOMATOSIS; circumscribed dilatations forming tumours composed chiefly of dilated lymph spaces are called LYMPHANGIOMATA.

These dilatations may be either congenital or acquired. A diffuse lymphangiomatous condition is the pathological basis of the congenital enlargements of the tongue and lip known as "macrochelia" and "macroglossia," and of some of the congenital tumours of the neck and floor of the mouth. In some instances, however, the tissues are neuro-fibromatous.

When the condition is acquired, the dilatation is due to obstruction. In tropical countries the lymphatic channels are blocked by the parasitic worm *Filaria bancrofti*, and the consequent obstruction to the flow of lymph leads to the development of elephantiasis, lymph-scrotum, and chylaria. In this country the obstruction is most often the result of injury—e.g., deep burns—or follows operation; but the examples are rare, and the obstruction is sometimes relieved after a time by the development of collateral channels.

3. Affections of the Lymphatic Glands.—(a) LYMPHADENITIS.—Acute inflammation of the lymphatic glands is most often seen as a local swelling of a gland or group of glands which drain a portion of the tissues infected by bacteria. In children the group most frequently affected is the cervical, in connection with infections of the fauces. The constant association of enlargement of this group with the presence of a retropharyngeal abscess should be noted.

Lymphadenitis in the specific fevers is treated in the appropriate sections.

Chronic lymphadenitis, especially of the cervical group, is very common in children. The causes are various. Among them may be mentioned otitis media, carious teeth, enlargement of the tonsils, the presence of adenoids, and the presence of sores upon the scalp (impetigo, pediculosis).

Tuberculous lymphadenitis is by reason of its great frequency the most important cause of enlargement of the glands. Tuberculosis of the bronchial and mesenteric glands is considered in the sections on Pulmonary and Mesenteric Tuberculosis (see pp. 391 and 242); that of the cervical and axillary groups comes under the notice of the surgeon; but the physician usually sees the rare cases of general enlargement, and often has to debate the diagnosis of cervical enlargements.

Generalized glandular tuberculosis is very rare, and always difficult of diagnosis. It may attack a child who has previously enjoyed good health, or it may be the terminal outbreak of a previously localized infection. In either case the disease sets in with all the symptoms of an acute infection—fever, malaise, anorexia, and rapid wasting—together with an acute enlargement of many groups of glands. An instance of the first type recently came under the writer's notice. The child, previously healthy, became acutely ill, and the cervical, axillary, and inguinal groups of glands rapidly enlarged. In addition, both pre-auricular and both submental glands were affected. The glands formed masses filling both sides of the neck and the axillary fossae, and stood out as definite tumours in the groin and on the face. In all these situations the skin was red and inflamed, and seemed

about to give way. The temperature was raised and the general health impaired. In this case no visceral lesions were detected, and improvement took place rapidly, with disappearance of the glandular swellings. The diagnosis was established by the excision and microscopical examination of a gland.

An example of the other type occurred several years ago: A child, the subject of tuberculosis of the lungs, suddenly became acutely ill with swelling of the superficial glands. After death, which occurred in two weeks, all the lymphatic glands, superficial and deep, were found enlarged and the site of tuberculous lesions.

Such cases must be diagnosed from lymphadenoma, lymphocytic leukemia, and sarcoma. Leukemia is easily excluded by a blood-examination. Sarcomatous conditions, while they may attack more than one group of glands, never involve many widely-separated groups at the same time. The real difficulty is the distinction from lymphadenoma. The chief stress must be laid upon the absence in that disease of the signs of inflammation, the absence of the matting together of the glands, of the signs of softening and of involvement of the skin.

With regard to the localized tuberculous of glands, it is sufficient to remark here that it is probable that it is often diagnosed when it does not exist. The tendency is to set down as tuberculous those cases in which there is a firm enlargement of a chronic type, where the individual glands are adherent to each other and the skin. That many of these are tuberculous is certain from the results of operation, but Wangh has recently shown that not a few are examples of a simple lymphadenitis due to a chronic focus of infection. He points out that no merely clinical diagnosis is sufficient proof of the presence of tubercle, and that this fact invalidates almost all the evidence in favour of the use of tuberculin in the treatment.

Syphilitic lymphadenitis is so rare a manifestation of congenital syphilis that the possibility is sometimes overlooked in the presence of a mass of glands of a chronic inflammatory nature. The writer has seen several cases in which this glandular swelling occurred, all in children at or after the period of the second dentition. The glands were moderately enlarged and firm, and their mass deformed the neck on either side. They slowly disappeared with antisyphilitic treatment.

(b) **TUMOURS OF LYMPHATIC GLANDS.**—*Primary* tumours of the lymphatic glands are rare. They are generally sarcomata of the small round-celled type. They occur most often in connection with the mediastinal and cervical glands, and tend to invade the surrounding tissues rapidly.

An uncommon type of growth occurs in the cervical glands, in which the endothelial cells grow into masses of large oval cells. Clinically this type of tumour, which must be considered an endothelioma, produces masses which deform the neck and invade the deeper structures. It does not, however, attack bone. It is of slow growth, and eventually kills by dissemination.

Secondary tumours, owing to the rarity of carcinoma, are uncommon in children. The diagnosis of such tumours can be made only by the microscope, and even with this aid it is often difficult to decide between malignant growth, lymphadenoma, and the chronic proliferative inflammation which occasionally is found in tuberculous infections.

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WANGH: *Ibid.*, 323.

CHAPTER X

DISEASES OF THE DUCTLESS GLANDS

A. E. GARROD

INTRODUCTION.

DISEASES OF THE THYROID GLAND:

ACUTE THYROIDITIS.

SIMPLE GOITER.

ENDOMETRIC GOITER.

THYROID ADENOMATOSIS:

CYSTIC, ENDOGENIC AND SPURIOUS.

MYXEDEMA.

DISEASES OF THE PARATHYROID GLANDS.

DISEASES OF THE PITUITARY BODY:

ACROMEGALY.

DYSTROPHIA ADIPOSO-GENITALIS.

DISEASES OF THE PINEAL GLAND.

DISEASES OF THE SUPRARENAL GLANDS:

MAALIGNANT MEDULLARY HYPERTHYROIDISM.

CORTICAL HYPERNECROSIS.

HÆMORRHAGE INTO THE SUPRARENAL GLANDS.

ADDISON'S DISEASE.

DISEASES OF THE TESTES GLANDS.

INFANTILISM.

INTRODUCTION.

At present we can claim to stand only upon the threshold of a wider knowledge of the functions of the ductless glands, and of the whole subject of internal secretions. Many facts of much interest have been already learned concerning their action and interaction; but up to now the known facts serve rather to indicate how wide a field remains to be explored than to afford a firm basis for generalization and theory.

It is obvious that these organs exert potent influences upon bodily development and nutrition, and that morbid changes in them lead to widespread pathological changes, which are attributable in some instances to undue activity and hypersecretion, in others to impaired function and hypo-secretion. Moreover, there are indications of an interdependence of the several glands of internal secretion, ductless and other, and the working of influences which are in some cases adjuvant, in others inhibitory, so that it may well be that disease of some one gland of the group may produce its morbid effects, not merely in virtue of excessive or impaired activity of the gland in question, but also by disturbing the functions of other glands which are in sympathy with, or in antagonism to, it.

It is a noteworthy fact that a particular symptom may result from disease of various glands of internal secretion, as, for example, glycosuria or peccorumis abesity. On the other hand, we do well to remember how scanty our knowledge of such influences still is, and that our science is likely to be hindered rather than advanced by the too ready acceptance of hypotheses which, as yet, rest upon but frail foundations, and by the assumption that, because lesions artificially

produced in certain glandular structures in animals have given rise to certain symptoms, such symptoms, when observed in man, are necessarily due to disease of the gland in question. Owing to the influence exerted by the glands of internal secretion upon growth and development, their diseases acquire special interest and importance in the study of diseases in children; for a malady which, when it develops in adult life, may produce changes which are capable of being counteracted and controlled by treatment may, when present in children, result in irreparable mental and bodily arrest.

DISEASES OF THE THYROID GLAND.

Some degree of enlargement of the thyroid gland may occur as a temporary phenomenon, and apart from any symptoms which are obviously referable to disturbance of its secretory functions. Thus, the swelling which is not uncommon in women during pregnancy is a well-recognized physiological occurrence, and somewhat analogous thereto is the slight enlargement of the gland which is seen in not a few girls at the onset of puberty.

Acute Inflammation of the Thyroid Gland.—*Acute thyroiditis* is a rare affection at any period of life, but not more so in children than in adults. It may occur as an isolated isolated event which cannot be ascribed to any recognizable underlying cause, but more often its development is an incident in the course of an infective fever, such as scarletina, measles, typhoid fever, or mumps. It has also been met with as a complication of malarial fever. Acute rheumatism is among its recognized causes, and Barlow has described its occurrence in a child of three years with an attack of erythema nodosum.

Pain and swelling in the region of the thyroid gland are its salient symptoms. The swollen gland is very tender when touched, and in order to minimize pressure upon it the head is held inclined forward, and all lateral movement is avoided. Any movement of the neck causes pain, and swallowing is difficult; but symptoms due to pressure exerted by the swollen gland are seldom observed. The shape of the swelling and its movements in deglutition serve to indicate the organ affected, but when at its height the swelling is wont to involve the surrounding parts, and may extend from the chin to the sternum. One lobe is usually the more enlarged, and this is more often the left than the right lobe. There is considerable febrile and other constitutional disturbance. The skin covering the inflamed thyroid is apt to be tense and shiny, but is seldom reddened, save in the rare cases in which suppuration occurs. As a rule the inflammation reaches its height in three or four days, and afterwards subsides rapidly. Recovery is usually complete, but some enlargement of the gland may persist, and may even be permanent. More rarely atrophy of the gland follows as a sequel to the acute inflammation.

The treatment usually adopted is the application of an ice-bag to the inflamed gland, and this affords considerable relief. In Barlow's case the application of a leech, when the swelling was at its height, had a very beneficial effect. When suppuration occurs, surgical measures are of course called for.

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Goitre.—In districts in which goitre is endemic many children are affected, and the enlargement of the thyroid gland may even be present from birth.

The greater liability of females is manifested in childhood as in adult life. Among 642 goitrous children, under fifteen years of age, who came under Denon's care in the Children's Hospital in Bern, during a period of thirteen years, 345 were girls and 297 boys. The ages at which the patients came under observation were as follows:

Within a few hours or days of birth	37
Between 2-12 months	59
" 2-4 years	35
" 5-7 "	83
" 8-10 "	94
" 11-13 "	150
" 14-15 "	184

In Denon's series were fifty-three cases in which the goitre was present from birth, and in this number were not included cases of mere hypoxæmic swelling of a transitory nature. In congenital cases the entire gland is usually involved in a true hyperplasia, with or without cyst formation.



FIG. 34.—CONGENITAL GOITRE IN A CHILD AGED EIGHT YEARS.

In some instances the tumour has been so large as to prove an obstacle to the birth of the child. In infants the thyroid lies high in the neck, and substernal goitre is rare. Nevertheless, the shortness of the neck, the vascularity of the tumour, and the pliability of the trachea, all conduce to the production of respiratory difficulties. Continuous or paroxysmal dyspnoea, which may culminate in asphyxia, is the most conspicuous symptom. Pulmonary oedema, atelectasis, and broncho-pneumonia are common complications.

The subjects of congenital goitre are often the offspring of goitrous parents. Of Denon's fifty-three patients, both parents had goitre in fourteen cases, the mother alone

in twenty-three. In later childhood goitre tends to develop as puberty is approached. The enlargement of the thyroid gland is very often partial, and the right lobe is specially apt to be affected. The goitres are usually of the follicular (parenchymatous) or cystic varieties. The colloid variety is rarely met with.

Not a few children affected with endemic goitre are cretins, whose thyroid glands have been rendered functionally inactive, but others show no signs of

hyperthyroidism. The symptoms produced by goitre in young subjects do not differ from those met with in adult patients, but respiratory difficulties are commoner.

Treatment with small doses of iodine has given the best results. When signs of hypothyroidism are present, thyroid extract should be given. It may, indeed, prove of service as a means of administering iodine in very small doses. In the case of infants at the breast, iodine or thyroid extract may be conveyed to the child through the medium of the mother's milk.

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SPRENGER: Haendler und Schlossmann, Handbuch der Kinderkrankheiten, 2te Auflage, 1910, III., p. 564.

Graves' Disease (Exophthalmic Goitre).—It is now generally conceded that the striking and characteristic symptoms of Graves' disease, or exophthalmic goitre, result from undue activity of the thyroid gland, and are the effects of continuous overcharge with its internal secretion. Thus Graves' disease may be looked upon as the converse of myxœdema.

ARE INCIDENCE.—Exophthalmic goitre is undoubtedly a very rare disease in children, and, of the few cases which fall within the accepted limits of childhood, the great majority occur in the latter part of that period of life, the incidence increasing as puberty approaches. Thus, of 39 cases brought together by Barret, 5 were in children under five years of age, 11 in children between five and ten years, and 23 in children between ten and fifteen.

The only case of congenital exophthalmic goitre known was recently recorded by Clifford White. The mother, who was herself suffering from disease at the time, gave birth to a male infant who exhibited all the features of the parental malady. The eyes were prominent and staring, the thyroid gland was uniformly enlarged, the heart's beats could not be counted, and a loud systolic murmur was audible over the precordial area.

The child was cyanosed, and died thirty-four hours after birth. The thyroid was found to be of twice the normal size, and sections showed overgrowth with proliferation of cells. The pituitary was not enlarged. The thymus was normal.

SYMPTOMS.—The symptoms as seen in children do not differ in any very material respect from those presented by adult patients. In some even of the youngest sufferers the clinical picture has been complete; in others one or other cardinal sign has been wanting. In some instances the disease is suspected rather than diagnosed; in others the diagnosis admits of no doubt. In prostrated examples the staring exophthalmos, and the facies as a whole, are at once recognized. Retraction of the upper eyelids (Stellwag's sign) and lagging of the lids (von Graefe's sign) may be well marked. The thyroid swelling is obvious, and pulsation of the gland is usually observed. Cardiac murmurs of the functional kind, so familiar in adult patients with Graves' disease, and tachycardia with palpitation, complete the picture. Tremor, which is one of the most constant symptoms in later life, has been absent in many of the juvenile cases, in some of which coarser and more inco-ordinate movements, suggestive of a chorea of

limited range, have been observed. Some children have been described as suffering from true chlorea in association with Graves' disease. Enlargement of the spleen has been met with in a few cases.

The following notes of a case in a boy aged four years may serve to illustrate the features of the malady as seen at that period of life.

The child, a twin aged four years, is the son of healthy parents, and there is no history of exophthalmic or simple goitre, nor of any grave neurosis, among his kindred. The other twin, also a boy, was weakly in infancy, but is now the stronger of the two. At about the beginning of his fifth year the mother noticed that the child had become nervous and excitable, and had a strange look in his eyes. He was also noticed to limp in walking. No assignable cause for the limp could be detected. During the following three months the symptoms developed considerably, and when he was seen by the writer, four months after the onset of the symptoms, his condition was as follows:

A well-nourished child and of healthy complexion. There was conspicuous



FIG. 55.—EXOPHTHALMIC GOITRE IN A BOY AGED FOUR YEARS.

exophthalmos, which at once suggested the diagnosis. Stellwag's and von Graefe's signs were present. The movements of the eyes in convergence were natural, and the skin of the forehead was raised when he looked upwards. The thyroid gland was enlarged and felt remarkably hard. The upper part of the lateral lobes felt pointed and horn-like. Pulsation was well felt. The area of cardiac dulness was not enlarged, but a loud systolic murmur was audible all over the precordial area, and was loudest at the apex. It was little, if at all, conducted into the axilla. The pulse was regular, 140 beats to the minute. There was no tremor of the extended hands, and no choreiform movements were observed. The knee-jerks were lively, but not exaggerated. There was no pigmentation of the skin. The urine contained neither sugar nor albumin. There was no wasting and no tendency to diarrhoea. No cause could be assigned for the onset of the disease.

Other movements were observed. The knee-jerks were lively, but not exaggerated. There was no pigmentation of the skin. The urine contained neither sugar nor albumin. There was no wasting and no tendency to diarrhoea. No cause could be assigned for the onset of the disease.

Ætiology.—The ætiology of exophthalmic goitre in children shows no special peculiarities. In some cases its onset has been ascribed to injury or to fright, and in some cases the influence of heredity has been conspicuous.

The greater liability of the female sex holds good for children as well as for adults. Of the thirty-nine cases collected by Barron (which include those previously brought together by Steiner), twenty-eight occurred in girls and eleven in boys.

There are few records of examinations of the thyroid gland in children who have succumbed to exophthalmic goitre.

PROGNOSIS.—The prognosis appears to be favourable. In a large proportion of the cases concerning which information is forthcoming, recovery has resulted, or recovery so nearly complete that a slight prominence of the eyes or slight enlargement of the thyroid has alone persisted.

In one recorded case (Söhring), a child of eight years exhibited the acute form of the disease, a very rapid development of the symptoms followed by an equally rapid recovery, the total duration being not more than ten days. In Droschfeld's case, a little girl of three years, who exhibited the symptoms in a most characteristic form, gradually recovered, and in her eighth year showed no trace of the malady save a slight prominence of the eyes, was in good health, and attended school. In two cases recorded by Baldwin, the hyperthyroidism has been followed by hypothyroidism, and myxodema has ensued upon the disappearance of exophthalmic goitre. Of Barret's thirty-nine cases only two proved fatal within the period of observation, which was, however, very short in many instances.

DIAGNOSIS.—In incomplete cases the diagnosis may remain open to grave doubt, and cases are met with in which children exhibit symptoms suggestive of Graves' disease concerning which the prudent observer will prefer to suspend judgment. However, we may well suppose that very various degrees of hyperthyroidism are present in different patients, and that the disease may at its time pass beyond the slighter grades which produce the less pronounced symptoms seen during the gradual onset of the more characteristic forms.

Children develop exophthalmos as the result of various causes, such as infantile strychny, malignant hypercæmia, and chloroma. Exophthalmos is one of the outstanding features of oxycephaly, but in practice such conditions are little likely to be mistaken for Graves' disease, the diagnosis of which is never made upon this symptom alone.

TREATMENT.—The degree of mental and bodily rest which is so essential for the successful treatment of the disease under discussion is more difficult to obtain for a child, and especially for a young child, than for an adult. Nevertheless, it should be our chief aim to obtain such rest, by change of air and scene, apart from other children, and, if possible, in company of a judicious nurse of whom the child is fond.

Medicinal treatment is of much less importance, for drugs do not exercise any great influence on the course of the malady. As in adult cases, gentle application of the galvanic current may be tried.

The apparently benign course of the malady in most children affords little encouragement to surgical measures.

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Thyroid Inadequacy—Cretinism and Myxodema in Children.—In children, as in adults, the symptoms of thyroid inadequacy vary in degree, from slight defects of mental and bodily development coupled with certain peculiarities of appear-

since which, in the slighter grades, suggest rather than posit their causation, to the most pronounced signs of cretinism which result from complete arrest of thyroid secretion. Such arrest may be due to advanced disease, atrophy, or absence of the thyroid gland. Some authorities, especially in France, attribute to thyroid inadequacy a very important share in the production of varieties of infantilism of which the causation is not obvious.

The name of "cretin" is applied to individuals of two classes who agree in exhibiting the signs of athyroidism from a very early age. Those known as "endemic cretins" are met with in goitrous districts, and in families so affected, and in them the thyroid gland is often enlarged, although functionally inactive; whereas in *sporadic cretins* the gland is atrophied or wholly absent. Sporadic cretins are met with in all lands, and are not uncommon in our own.



FIG. 56.—SPORADIC CRETINISM.

Sporadic Cretinism.—Although the thyroid defect is present from birth, the new-born cretin does not show the characteristic features of its condition until some time later, and up to the age of some six months there may be nothing to suggest cretinism, unless it be an undue placidity and slowness of movement. This delay in manifestation is probably due to the presence of a certain amount of thyroid secretion in the milk which forms the diet of the infant, and it is stated that the symptoms of cretinism are longer postponed in breast-fed than in bottle-fed children.

CLINICAL FEATURES.—Once developed, the symptoms and features of cretinism are highly characteristic. In build the child is stumpy and thick-set, and the limbs are short in comparison with the trunk. The skin is harsh and dry, and has a yellowish, sallow tint, whereas the extremities are cold, as a rule, and exhibit a distinct cyanosis. The hair of the head is coarse, brittle, and scanty, and usually reddish-brown in colour. The individual hairs appear to be set too widely apart, but the body may be covered in parts, as also may be the forehead, with a coarse down. The head is large, and usually

delicho-cephalic. The fontanelle remains widely open long after the period at which it closes in a normal child. The eruption of the milk-teeth is often, although not always, much delayed, and these teeth persist much later than is usual, whereas second dentition tends to be correspondingly postponed.

The facies is distinctive. The palpebral fissures are narrow and slit-like, and set far apart; the nose is broad and flat; and these peculiarities, coupled with the coarse thick lips and broad thick tongue, which is often protruded between the lips, as if too large for the mouth, gives to the face a peculiar ugliness. These characteristics of appearance are chiefly due to thickening of the subcutaneous tissues, skin, and mucous membranes. The toes share in the general enlarge-

ment, and the nasal passages tend to be narrowed. The expression is dull and apathetic, and reflects the underlying mental state. The peculiar smile of the cretin, the long-drawn-out muscular contraction, and the broad and widely-spreading folds into which the redundant skin is thrown, are diagnostic of the condition.

The myxedematous infiltration affects the trunk and limbs also, and renders the neck broad and squat, an appearance which is accentuated by the presence of conspicuous pads of subcutaneous fat above the clavicles. The abdomen is protuberant, and its prominence usually increased by lordosis. The umbilicus almost always projects, and there is often a true umbilical hernia. The cold, blue, stumpy hands, with their broad squat fingers which do not exhibit the usual differences of length, are also characteristic of cretinism.

As age advances, the child falls more and more behind his fellows in mental and bodily development, and when adult age is reached the cretin may not be so much developed as a child of five or six. The infantile characters persist, there is no sexual development, and little or no gain of intelligence. Speech is usually limited to a few words, indistinctly articulated. Power of standing and walking is usually acquired very late. The disposition tends to be placid and apathetic. The child sleeps much, and is with difficulty stirred to any display of emotion. The movements are slow and reptilian, and cleanly habits are acquired late, if at all.

The metabolic fires, lacking the stimulus of the thyroid secretion, burn low. The body temperature tends to be subnormal, and the child is unduly sensitive to low temperatures, in striking contrast to the undue activity of the metabolic processes which is a striking feature of hyperthyroidism, as seen in exophthalmic goitre. The pulse is usually infrequent. Constipation, often of a very obstinate kind, is a common symptom. There is usually a more or less pronounced degree of anemia, with a low percentage of hemoglobin.

DIAGNOSIS.—The diagnosis of cretinism in its more pronounced forms should present little difficulty. The condition most likely to be mistaken for it is the Mongolian form of idiocy; but, apart from a certain superficial likeness, the cretin and the Mongol have little in common. The vacant expression, the narrow eyes, and the protruded tongue, constitute the chief resemblance; but the eyes of the cretin have not the Mongolian slant, and his large square tongue has little resemblance to the pointed mobile tongue of the Mongol, which is so often inserted beneath



FIG. 57.—SEVERE CRETIN. GIRL, AGED SEVEN YEARS.

the lower lip. The large dolicho-cephalic head of the cretin is quite unlike the small short head of the Mongol, with its flattened back; and his squat plump hands have nothing in common with the hands of the Mongol, with their tapering, separated digits and incurved little finger.

Still less should be the risk of confusion between the slowly-moving and listless cretin and the active and alert achondroplasia, whose intelligence, when not above the average level, is seldom below it. The chief superficial resemblances between dwarfs of these two kinds lie in the relative shortness of the limbs, the large head, and the flattened bridge of the nose.

MORPH ANATOMY.—The changes in the skin, mucous membranes, and subcutaneous tissues resemble those seen in the myxedema of adults. The bones show abundant evidence of defective development, and retain the features of childhood at all ages. In the epiphyses of the long bones the centres of ossification are very late in appearing, and complete ossification is indefinitely delayed. In the diaphyses both the periosteal and internal development of bone is, to a great extent, inhibited. Some authors have described the formation of a thin layer of bone at the edge of the epiphyseal line, and Siegart states that this layer is shown as a sharply-defined linear shadow in X-ray pictures.

No trace of the thyroid gland may be discoverable, or only a completely atrophied vestige of that structure.

Acquired Myxedema in Children.—It is probable that acquired, as distinguished from congenital, myxedema is commoner among children than has usually been supposed. The diagnosis of such cases rests in part upon the history, which indicates that up to a certain age, three or four years or more, the child exhibited no abnormal features, and learned to walk and to speak at the usual ages; and in part upon the evidences of such signs of development in early years as are wanting in congenital cretins, such as closure of the fontanelle, a greater degree of growth, some power of speech beyond that expected in the cretin, and somewhat better mental faculties.

From cases of incomplete thyroid inadequacy, on the other hand, such children are distinguished by the evidences of complete athyroidism which they exhibit. They differ from cretins in that their growth and mental development have been cut short at a more advanced stage; and the older the child when the disease develops, the more obvious will be the evidences of an antecedent advance.

In some cases the atrophy of the thyroid gland has followed an acute inflammatory process in the organ; a few cases of cachexia strumipræva have been observed in children; but in the majority of instances the origin of the thyroid inadequacy is no less obscure than it is in most cases of myxedema in adults.

TREATMENT.—It is of the utmost importance that cretinism should be early diagnosed, seeing that we have at our disposal an efficient treatment of the condition, and time lost in initiating such treatment can never be fully regained. Each year which passes without thyroid administration involves a retardation of development which is to some extent irreparable. If, after the child has been kept supplied with the required amount of thyroid extract from earliest life throughout the whole period of growth, treatment should be omitted for a time, a relapse will occur, but arrest of development will have been averted. If treatment be resumed, such a patient will respond, as does an adult sufferer from myxedema.

The effects of thyroid administration are somewhat unequal. In some cases

a cretin who has been treated from a very early age may after a time be quite unrecognizable as such, may develop as does a normal child, and hold his own in school with other children of like age. In most cases, however, some of the cretin marks remain, perhaps because they have already become impressed before treatment was initiated. Other factors, such as the degree of thyroid inadequacy and the sufficiency or otherwise of the dose of thyroid extract given, may also come into play. In cases in which the diagnosis presents any doubt thyroid treatment should be tried, and its effects will sometimes clinch the diagnosis, whilst giving the patient the benefit of the doubt. The earlier the treatment is begun, the better the ultimate result, whereas it is permissible to doubt how far it is desirable to initiate thyroid treatment in the case of a cretin who has reached adult years, seeing that the improvement in the physical condition which is brought about is often not attended by any corresponding mental gain, or the patient may be merely rendered conscious of his deficiencies of mind and body.

The administration of thyroid extract to young children should be carried out with caution. Overdosage may cause febrile and other constitutional disturbances, and is not unattended with danger. It is better to begin with a dose of $\frac{1}{4}$ to $\frac{1}{2}$ grain, given once or twice a day, and to increase the quantity gradually, as age advances, until such a quantity as 5 grains is given in the day. The administration of thyroid must, of course, be continued throughout life, and the optimum dose will be such as will be equivalent to the natural secretion of the gland. The means best calculated to afford an accurate dosage is that suggested by Cushing for the estimation of the required dose of pituitary extract in cases of pituitary inadequacy. Patients with hyperthyroidism usually excrete sugar after a single dose of glucose smaller than is needed to excite alimentary glycosuria in a normal individual, and, conversely, patients with myxedema or cretinism can deal with far larger doses of glucose than can normal persons of like age and weight without excreting sugar. Administration of thyroid extract reduces the dose of glucose required to produce glycosuria, and we may suppose that a patient whose glucose tolerance has been so reduced to the normal limit is taking the exact dose which he requires. However, this mode of estimating dosage has not yet come into practical use.



FIG. 58.—SPORADIC CRETINISM. Treatment with Thyroid Extract begun late, and caused but little improvement.

DISEASES OF THE PARATHYROID GLANDS.

It is necessary to refer to diseases of the parathyroid glands in this chapter, because it must be regarded as an established fact that symptoms of tetany result from removal of these structures, and the tetany which sometimes follows thyroidectomy is to be attributed to simultaneous removal of the parathyroids. It is held by some authorities that the tetany of children is due to parathyroid lesions, and in not a few cases of fatal tetany these glands have been found to be the seats of old or recent hemorrhages. But it has been objected, on the other hand, that lesions of the parathyroids are to be detected post mortem in many cases in which there have been no symptoms of tetany during life. It must be remembered, however, that there are several parathyroid glands, some of which may be destroyed, whereas others may escape and may suffice to prevent the development of symptoms of parathyroid inadequacy. Moreover, it must be borne in mind that the symptoms of tetany vary widely in severity, so that in cases, in which carpo-pedal contractions are absent, latent indications, such as facial irritability, may be excited by suitable stimuli (see Chapter XIII., p. 684).

Other groups of symptoms which have been attributed by some to lesions of the glands under discussion, such as paralysis agitans, do not concern us here.

DISEASES OF THE PITUITARY BODY.

Recent years have brought large additions to our knowledge of the functions of the pituitary body, and the effects which follow upon disease of that organ. There is reason to believe that, of the symptoms observed, some depend upon overactivity of the gland (hyperpituitarism) and others upon reduction or arrest of its internal secretion (hypopituitarism). Again, we are gaining an insight into the respective functions of the several lobes.

Acromegaly is believed to be a manifestation of hyperpituitarism, although in the latter stages of the malady diminution of the functional activity probably occurs. It occupies a very small place among disease of early life. There are a few recorded cases of acromegaly in older children, at or after puberty, as, for instance, that described by Franchini and Giglio, of the fourteen-year-old daughter of an acromegalic father, who had, from the age of puberty, exhibited unequivocal signs of the parental malady, including enlargement of the nose, lips, and jaw, and abnormal development of the hands and feet.

There is good reason to believe that hyperpituitarism, when developed in childhood, before ossification is complete, tends to manifest itself in a different manner, and that child patients exhibit the earlier stages of that variety of gigantism which has been shown to be associated with pituitary tumours. The bones increase in length in a manner which is no longer possible after the junction of the epiphyses with the shafts. On the other hand we may suppose, with Brocaud, that, when the morbid process continues active into later life, the changes characteristic of acromegaly are grafted on to the gigantism which began at an earlier period.

Dystrophia Adiposo-Genitalis.—This condition, commonly known as "Frölich's syndrome," after the observer who first described it, is now generally attributed to a converse disorder—hypopituitarism. If this view be correct, Frölich's syndrome stands to acromegaly in a similar relationship to that in which myxedema stands to exophthalmic goitre. A considerable proportion of the observed cases of this syndrome have been in children of about fourteen years of age.

The most conspicuous signs are obesity and hypoplasia of the genital organs, which acquire or retain infantile characters. With these are nearly always associated signs of cerebral tumour, localized in the region of the hypophysis, headache, and bitemporal hemianopia. The distribution of the subcutaneous fat is chiefly on the abdomen and breast; the mammae are usually well developed, even in male patients, and there is in addition a general increase of fat on the trunk and limbs. In some instances, as in myxedema, a raised tolerance for glucose has been demonstrated. Doses much larger than are required to induce glycosuria in normal individuals could be taken by the patients without giving rise to any excretion of sugar in the urine.

A priori there would seem to be no necessity that the pituitary lesion should be of the nature of a tumour—a simple atrophy of the gland should be sufficient to produce the symptoms; but as yet we have no definite knowledge of such lesions. Cases which were apparently of this nature have been described in which no marked changes were found in the pituitary body, even on microscopical examination. Madelung has recorded a remarkable case of a girl of nine years, in whom a bullet, which lodged in the sella turcica, induced the generalized adiposity which is a most conspicuous factor in Frölich's syndrome.

In some instances treatment with pituitary gland preparations has had beneficial results.

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FRANKEL: *Deutsche med. Woch.*, 1888, xiv, 451.

FRÖLICH'S SYNDROME.

- FRÖLICH: *Wiener klin. Wochenschr.*, 1901, xv, 883.
T. FRANKEL-HOCHWALD: *Sixteenth International Congress of Medicine (Buda-Pest)*, 1906, Sect. vi., p. 89.
MADELUNG: *Archiv f. Klin. Chirurgie*, 1904, 1906.

DISEASES OF THE PINEAL GLAND.

Our knowledge of the influence of the pineal gland upon bodily development is almost entirely derived from the peculiar changes which have been associated with tumours of this structure in a small number of children. One of the earliest descriptions of such a case was given by Cyril Ogle, and our knowledge of the subject has been greatly advanced by the observations of Marburg and v. Frankl-Hochwald. In addition to the symptoms pointing to the presence of a cerebral tumour affecting the corpora quadrigemina (see p. 365), there have been present certain more special signs which indicate the organ involved. In such cases there have been found after death tumours of the pineal gland, usually teratomata,

a form of neoplasm to which this organ appears to be especially liable. These special signs are abnormal growth, obesity, which is usually not so pronounced as in the adrenal or pituitary cases, luxuriance, precocious development of the genital organs, with growth of pubic hair, mental precocity, and sometimes extreme sleepiness. Nearly all the cases have been boys between four and eight years of age. Marburg's case was in a girl of nine years.



FIG. 59.—PRECOCIOUS PUBERTY OF UNCERTAIN ORIGIN IN A BOY AGED FIVE YEARS.

(From a Photograph in the Museum of St. Bartholomew's Hospital.)

These cases present resemblances to those of cortical hypersecretion, on one hand, and the pituitary cases on the other. From the former they are distinguished by the signs of the cerebral tumour; from the latter by the localizing cerebral symptoms, and by the fact that pituitary obesity tends to be associated with arrested development of the genital organs, in conspicuous contrast to the precocious development seen in the pineal cases.

Frankl-Hochwald regards it as probable that the functions of the pineal gland are only exercised in early life, since its involution commences before puberty. This will explain the fact that the symptoms described have only been observed in children. He suggests, moreover, that, as regards sexual development, the functions of the pineal are to some extent antagonistic to those of the pituitary gland, and that loss of function of the former leads to hypertrophy of the genital organs, whereas when there is hypopituitarism, as in Prödl's syndrome, a contrary effect is produced.

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 [MUSKIEWICZ: *Wiener med. Wochenschr.*, 1908, p. 2617.
 [OULE, C.: *Pathological Society's Transactions*, 1899, l. 4.
 [BAILEY AND JEFFERSON: *Tumours of the Pineal Gland*. *Archives of International Medicine*, 1917, viii, 803.]

DISEASES OF THE SUPRARENAL GLANDS.

Although the cortex and medulla of the suprarenal glands form parts of a pair of circumscribed glandular organs, the study of their development and of the structure of the corresponding organs in the lowest vertebrata shows that, in reality, cortex and medulla are distinct organs. Moreover, the suprarenals as present in man do not embody the whole of the medullary and cortical substance. Chromaffin tissues such as constitute the medulla are somewhat widely distributed outside the suprarenals, and accessory glandules having the structure of the cortex are frequently present upon the surfaces of the kidneys and elsewhere.

The discovery of adrenalin, the special secretory product of the chromaffin

times, a substance of comparatively simple chemical composition and structure, has given a great stimulus to the study of the functions of these glands. Moreover, there are indications that the cortex also has important influences upon bodily development, as witness the symptom-complex which is associated with cortical hypernephromata. This lends a peculiar interest to the study of diseases of these organs in children.

There is reason to believe that complete absence of the suprarenals, a rare congenital anomaly, is intimately associated with gross cerebral defects, such as anencephaly.

The diseases of the suprarenals which call for special description in a work dealing with diseases of children are hemorrhagic lesions and tumours, which latter give rise to widely different groups of symptoms according as they originate in the medulla or cortex. Addison's disease is very rarely seen in children.

Malignant Medullary Hypernephromata.—Malignant growths originating in the medullary portion of the gland are not very rare in young children, and the clinical picture which the cases present is singularly uniform in character, although it differs according as the right or left suprarenal is involved. As R. Hutchison first pointed out, such cases constitute a well-defined disease of childhood. Their interest lies rather in the distribution of the secondary growths than in any effects of disordered secretion, and also in the fact that the diagnosis is based as a rule upon the nature of the metastases, and is, not infrequently, correctly made before the primary suprarenal growth can be detected by palpation. Indeed, it may happen that the primary growth is only found after the patient's death. This is more particularly true of the cases in which the left suprarenal is the seat of the tumour. The majority of the cases occur in children of two or three years, and the great majority of the patients are less than six years old. Of forty-six cases collected by Frew, twenty-seven were in male and nineteen in female children.

Symptoms.—The earliest symptoms may be wholly misleading, and suggest rheumatism rather than a malignant growth. Pains in the lower limbs, sometimes referred to a knee, may be complained of, and in some cases a systolic murmur has been audible over the heart, which is not explained by any valvular lesion to be found after death. In not a few cases the earliest characteristic sign is the development of a hemorrhagic streak upon one upper eyelid, or both. This is soon followed by protrusion of the eyeballs, first one and then the other, and the eye first affected is usually that on the same side as the primary suprarenal growth. Experience of similar cases will enable a provisional diagnosis to be made at this stage, although it is unlikely that any abdominal tumour will yet be palpable. Before long tumours develop upon the outer surface of the skull, often in the temporal fossæ, and the diagnosis then becomes highly probable. The exophthalmos increases, and optic neuritis develops, which often attains an extreme degree and may culminate in blindness. The exposed corneæ are apt to become ulcerated. The general symptoms are increasing secondary anemia, progressive weakness and headache, and other signs of intracranial pressure. The temperature may be slightly raised at times, but there is no conspicuous fever. Pigmentation is not observed, the blood-pressure is not raised, and there is no leucocytosis.

In the right-sided cases the suprarenal tumour is more apt to be felt at an early stage, and the growths upon the skull are less prominent features. Exophthalmos

if present, is usually first observed in the right eye, and a group of enlarged glands is usually palpable behind the sternal end of the right clavicle.

The disease is always fatal, usually in a period of from one to six months from the appearance of the early signs.

DIAGNOSIS.—As has been already mentioned, diagnosis is usually impossible until exophthalmos develops, and in some cases the symptoms are suggestive of rheumatism. Later on the condition may closely resemble that seen in cases of infantile scurvy with orbital hemorrhages, in which the exophthalmos is usually preceded or accompanied by hemorrhage into the eyelids. The absence of other signs of scurvy, and especially of the characteristic general tenderness, the failure of antiscorbutic treatment, and the appearance of cranial tumours, will serve to correct such a diagnostic error, although subperiosteal hemorrhages upon the cranial bones are occasionally seen in cases of infantile scurvy.



FIG. 68.—METASTASES OF MALIGNANT HYPERCEPHALIA.
(E. Hutchinson, Quarterly Journ. Med.).

Again, the syndrome here presented is very closely simulated in some cases of chloroma, for chloromatous growths commonly invade the orbits and give rise to exophthalmos. However, examination of the blood serves to distinguish between the two diseases, for the conspicuous blood-changes in chloroma stand in marked contrast to the normal leucocyte count in the cases of hypercephalæmia.

HISTOLOGY.—The primary tumours are enclosed in firm fibrous capsules, from which fibrous trabeculae extend into the tumour substance. A portion of unaffected cortex is usually to be seen upon the surface of the tumour. The tumours consist of round or oval cells, with large nuclei and a granular protoplasm. They have usually been described as "round-celled sarcomata," but opinions differ as to their nature, and by some they are regarded as belonging rather to the class of carcinomata.

As the result of careful examination of several cases, Frey arrived at the conclusion that the spread is by the lymphatics, and that the clinical differences between the right- and left-sided cases is to be explained by the fact that the

lymphatics of the left suprarenal run to the lumbar glands and thoracic duct, whereas those from the right suprarenal are tributaries of the right lymphatic duct. This opinion he bases upon the distribution of the enlarged lymphatic glands in the cases of the two classes. Thus, in the left-sided cases the lumbar glands were involved, the mesenteric, and those in the posterior mediastinum around the thoracic duct. A group of enlarged glands lay behind the sternal end of the left clavicle, and there were some affected glands in the neck. In the right-sided cases affected glands were present on the upper surface of the liver, on the surface of the right half of the diaphragm, on the pericardium, and in the anterior mediastinum. In such cases there were also enlarged glands at the root of the lungs and pulmonary metastases. When the growth was in the left suprarenal the lungs were not invaded.



FIG. 31.—MALIGNANT MEDIASTINAL HYPERNEPHROMA.
(H. Hitchcock. Quarterly Journ. Med.)

Growths are found in the medulla of bones, and subperiosteal swellings upon the inner and outer tables of the skull. The exophthalmos is due to implication of the sphenoid, causing it to bulge forward into the orbital cavity.

REFERENCES.

- HITCHCOCK, H.: *Quart. Journ. of Medicine*, 1897-98, i, 33.
PHEW, R. S.: *Ibid.*, 1911, iv, 123 (with bibliography).

Cortical Hypernephroma.—Cases in which tumours of the suprarenal cortex exist present a wholly different aspect. With them there tend to be associated precocious obesity or muscular development, hirsuties or actual precocious puberty. As these developmental anomalies are presumably connected with the functions of the structures involved, and not with the nature of the new growth, it is not surprising to find that the tumours in question have not been of uniform nature. In the majority of cases they have been described as carcinomata; but it is notorious that malignant tumours of the suprarenals are especially difficult to classify, and

form a group apart, so that it is safer, as Rolleston suggests, to apply to them the neo-oncological name of "malignant hypernephroma." In some few cases the growths have been classed as sarcomata, and in some others they appear to have been simple adenomata, devoid of malignancy.

The developmental changes also appear to differ in different cases, and fall under the heads of precocious puberty, to which Bullock and Soqueira have specially called attention; hirsuties, including growth of pubic hair, apart from true premature sexual development; obesity; and in some instances excessive muscular development apart from any obesity. Cases of this last variety, for which Parkes Weber has proposed the name of the "infant Hercules" type, have hitherto been observed in male children only, whereas precocious obesity occurs in both sexes.

The form of obesity so designated is of a different kind from that seen in ordinary fat children, and the distribution of fat resembles that met with in middle life. To quote Guthe and Emery: "In precocious obesity the features are bloated, the cheeks swell with fatness, the complexion is dusky and congested. The skin may be studded with stellate venules; bluish-white striae resembling the *lineae gravidarum* are seen about the abdomen and thighs. The fat is usually distributed evenly throughout the body, but the upper and lower extremities may remain thin. Rolls or pads of fat are situated about the neck, breasts, and flanks; a lipomatous mass or lump is sometimes seen between the shoulders. The mammae may be large and well developed; the belly is pendulous and bulging."

The hirsuties manifests itself in the premature development of pubic hair; but it may be widely distributed over the body, in the axilla, or even on the face. Actual sexual precocity is not a common feature in the obese cases, but is constant in those of the muscular type. Hirsuties may occur apart from obesity. In one remarkable case recorded by R. Hutchinson, the obesity was unilateral, and the suprarenal of the same side was enlarged and hyperplastic. However, the remaining paired organs—*e.g.*, the kidney and testicle—were also much larger on the affected side.

It should be mentioned that cases resembling those under discussion have been recorded in which there was no suprarenal tumour. In some of these there have been tumours of the testes or ovaries, in others tumours of the pituitary gland (p. 572), and in others, again, no obvious lesion of any organ.

REFERENCES.

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GUTHE AND EMERY: *Clinical Society's Transactions*, 1907, xl, 175.



FIG. 62.—DR. GUTHE'S CASE OF PRECOCIOUS OBESITY AND HIR-
SUTIES IN A BOY AGED FOUR AND
A HALF YEARS: HYPER-
NEPHROMA.

Hæmorrhage into the Suprarenal Glands.—Hæmorrhage into the suprarenals, or more often into only one of them, has been found post mortem in a considerable number of infants still-born, or who have died during the first days of life. Still found, among the records of 3,731 autopsies on children less than twelve years of age, at the Hospital for Sick Children, only four cases of suprarenal hæmorrhage, one of which was in a child aged three, and the remainder in infants. Among still-born infants such lesions are probably less rare. Thus, Spencer found 23 cases of hæmorrhage in 106 such cases. The right suprarenal is more often affected than the left.

Still groups the cases in three classes: (1) Those in which death occurs within a few hours or days of birth. In some of these actual rupture occurs, with escape of blood into the peritoneal cavity. (2) Those in which death occurs later, and the suprarenal lesion is a complication of some disease, usually in the respiratory tract, such as tuberculosis or broncho-pneumonia. (3) Those in which, after an acute febrile illness, lasting only two or three days, often with a purpuric eruption, suprarenal hæmorrhages are found post mortem.

The cases of the third class form a group apart, and the suprarenal hæmorrhage, which is bilateral, appears to be a part of the fatal illness. Such cases are almost exclusively met with in patients under one year of age, and they are wont to occur in groups, although the victims may come from widely scattered homes, and have never been in contact with each other. Such a series of cases came under observation at St. Bartholomew's Hospital in 1902, when the writer made the autopsies on several of them; and C. Riviere has described a similar series at the Staswell Hospital, where three cases were brought in within a period of twenty-three days. Most of the affected children have been unvaccinated, but there are no adequate grounds for supposing that the disease in question is a form of variola.

Symptoms.—A child, usually some five or six months old, is suddenly attacked with a febrile illness, which may be ushered in by vomiting. Localizing abdominal symptoms are as a rule absent, but there may be diarrhoea. The skin often shows a purplish mottling, and petechiæ or vibices may appear. There is considerable fever, which may reach the grade of hyperpyrexia. Convulsions are as a rule prominent symptoms, and collapse, with coldness of the extremities and rapid pulse and respirations, closes the scene. The whole duration of the disease is usually not more than twenty-four hours.

MORPHOLOGY.—At the autopsy areas of engorgement or hæmorrhage may be found in other structures, but the most striking appearance is that of the suprarenal glands, which may be somewhat enlarged, but preserve their form and have a deep purple colour. On section the division between medulla and cortex is not obliterated, but the medulla has a deeper purple hue. Under the microscope the structures of the gland are seen to be infiltrated with blood throughout, and especially the medulla. In the more advanced cases the cells proper of the gland appear to have been destroyed, and are replaced by extravasated blood-corpuscles. In some parts glandular cells are seen in considerable numbers; their nuclei stain well, but the cell substance appears to be partly destroyed. Cultures from the affected organs, and also from the spleen and liver, have proved sterile.

When series of cases have occurred, the correct diagnosis has been made in some of the later ones, but the symptoms suggest the onset of one of the acute exanthemata.

No TREATMENT has proved of any avail hitherto.

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 ANDREWS, F. W. : *Pathological Society's Transactions*, 1908, lxxx, 253.
 GARDIN, A. E., and DRYSDALE, J. H. : *Ibid.*, p. 257.
 SYLL, G. P. : *Ibid.*, p. 252.
 EATON, F. E. : *Ibid.*, p. 258.

Addison's Disease.—Addison's disease is rare at all periods of life, but appears to be even rarer in children than in adults. Thus, Monti, writing in 1878, had only been able to find 11 recorded cases in children under fifteen among 250 cases, and nearly all of these were in older children, between twelve and fifteen years of age. In four cases there was pulmonary tuberculosis in addition, and in another there were caseous mesenteric glands.

The symptoms as observed in children resemble those seen in adults, including progressive debility, wasting, loss of appetite, and digestive disturbances, such as vomiting, diarrhoea, and singultus. The bronzing of the skin has been slight in some cases, as among adults, but is usually well marked, and has the ordinary distribution, affecting the mucous membrane as well as the skin. The pulse is small and of very low tension.

Netter describes the case of a girl of thirteen who was suddenly seized with diarrhoea, fever, prostration, and severe abdominal pain, which suggested a diagnosis of peritonitis. The only sign which pointed to Addison's disease was a slight pigmentation of the skin. Death occurred three days after the onset, and there were found at the autopsy old tuberculous lesions of the suprarenal capsules. The spleen was very large, and Peyer's patches were swollen, but not ulcerated. From the spleen pulp streptococci were obtained in pure culture.

In the case of another girl of thirteen, whose case is recorded by Desrochelles, the symptoms exhibited were general bronzing, with some pale areas on the limbs, feebleness, and widespread cramps. Here also there was abdominal pain, diarrhoea, and dilatation of the pupils. The temperature was not raised; the pulse was rapid, thready, and intermittent; the urine was scanty and albuminous. The patient sweated profusely and wasted rapidly. At the autopsy the suprarenals were found to be enlarged and to contain many tuberculous foci. The thymus was unduly large, and weighed 25 grammes.

Monti has described a case, in a boy of eleven, in which the lesion found after death was atrophy of the suprarenals.

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 MONTI : *Gesundh's Handbuch der Kinderkrankheiten*, 1878, iv., Abth. G., 200.
 NETTER : *Sanation Méd.*, 1900, ix, 154.

DISEASES OF THE THYMUS GLAND.

INTRODUCTION.—Save in connection with thymic asthma, and lymphatism, which is discussed elsewhere (p. 585), diseases of the thymus gland occupy but a very small place in clinical medicine. This is somewhat remarkable, seeing that it can hardly be doubted that an organ so conspicuous has important functions, which are mainly exercised during the period of maximum development—namely, in early life. Of the nature of these functions we know hardly anything.

It is now generally conceded that the thymus is a gland of internal secretion. There are evidences of a correlation of its functions with those of the genital glands, and the enlarged thymus of patients with exophthalmic goitre suggests that it also has relations to the thyroid. On the other hand, the thymus tends to be involved in abnormal or diseased conditions of the lymphatic structures, as witness its enlargements in the subjects of lymphatism.

Experimental removal of the thymus from young animals, and injection of extracts of the gland, have not produced results nearly so striking as in the cases of other glands of internal secretion, such as the thyroid and pituitary. Of experiments of the former kind, those of Basch have yielded the most important results. In the animals from which the thymus had been removed, and especially in puppies, a conspicuous softening and pliability of the bones was observed, as compared with those of control puppies of the same litter. The processes of repair after fracture were seriously interfered with, and a markedly increased excretion of lime salts was at the same time observed. In the young of herbivorous animals the changes in the bones were far less conspicuous, and it may well be that the conflicting testimonies of different observers may be due in part to the choice of animals experimented upon, and in part to technique and more or less complete removal of the gland. In addition to the changes in the bones, Basch observed more general effects, such as delayed growth, diminished activity, and change of disposition.

The injection of thymus extracts has taught us very little. Suchla observed a conspicuous fall of blood-pressure and quickening of the pulse, but similar effects have followed the injection of extracts of many tissues.

It may be stated generally that the symptoms observed in cases in which the thymus has become the seat of gross disease, such as tuberculosis, have little or no connection with variations in the quantity of an internal secretion of the gland.

Hypertrophy of the Thymus.—The thymus is subject to such wide variations of bulk and weight that it is difficult to estimate with certainty whether it be unduly large in any given case. The weight of the gland in normal children has been estimated as high as 20 or even 30 grammes; but the observations of Dodgson and Thursfield show conclusively that such estimates are excessive, and that the average weight is some 10 grammes. The weight and bulk are said to increase up to the age of two years, and thenceforward to remain stationary up to the age of eight to twelve years. After this the gland undergoes atrophy, and its substance becomes largely replaced by fat.

In most cases conspicuous enlargement is due to a simple hyperplasia, but inflammation, oedematous swelling, and extravasation of blood, play an important part in this connection.

The clinical recognition of hypertrophy of the thymus is by no means easy. Occasionally the upper portion of the enlarged gland may be felt projecting above the sternum, but as a rule the detection of its increased size rests upon the presence of an area of dullness behind the manubrium sterni, which dullness is described by Blumenreich as of an irregular triangular shape. The base of the triangle connects the sterno-clavicular joints, and its apex extends downwards to the level of the second chondro-sternal junctions. The evidence afforded by this impairment of the percussion note may be greatly strengthened by the demonstration, by means of the X-rays, of an area of opacity above the cardiac opacity in the middle line.

Thymic Asthma or Stridor; Kop's Asthma; Millar's Asthma.—The condition designated by the above names is a respiratory stridor, which is due apparently to compression of the trachea. It may be present at birth, or may develop during the early weeks of life. The stridor is more pronounced during inspiration, but both inspiration and expiration are stridulous. It may be continuous or paroxysmal. The signs of laryngeal obstruction are wanting; the cry is natural. In some cases there may be some associated dysphagia. During a paroxysm of dyspnoea, the obstruction to respiration may be so great that cyanosis supervenes, with lividity of the lips and tongue. Death may occur during the paroxysm, and it is probable that such a paroxysm is the cause of sudden thymic death in not a few cases. In the milder cases the stridor tends to diminish, and ultimately ceases as the child grows older.

The diagnosis of the condition will rest upon the character of the dyspnoea and the presence of other indications of thymic enlargement; and it must not be forgotten that other forms of respiratory stridor are met with in young infants, including a congenital stridor of laryngeal origin (Chapter VI., p. 292).

The nature of thymic asthma has been the subject of much discussion. While some hold that, unless very grossly enlarged, the thymus is incapable of exerting such pressure upon the trachea as would be required to produce the observed effects, others—and these, perhaps, the majority of observers—incline strongly to a mechanical explanation. In support of such an explanation is adduced the rigid girdle in which the thymus and trachea lie, the narrowness of the upper opening of the thorax in infants, and the tendency for attacks to be provoked by the throwing back of the head. In some cases the trachea shows indications of compression after death, and it is not to be expected that slighter degrees of pressure will produce permanent alterations in the shape of so elastic a structure. Lastly, the mechanical theory gains powerful support from the favourable results which have followed, occasionally, upon complete or partial removal of the peccant gland in cases in which life has been threatened.

Lymphatism.—The condition known as "lymphatism" or "status lymphaticus" is dealt with in connection with diseases of the lymphatic structures (p. 548).

Atrophy.—The thymus is found to be conspicuously atrophied in infants who have succumbed to wasting diseases, and this is the case whether the wasting be secondary to some gross disease, such as tuberculosis, or is due to mal-assimilation of food or to other causes which produce no definite changes which can be detected after death. Dodgeon agrees with other observers in describing the atrophied gland as white, soft, and indurated; and when sections are observed with the microscope, the fibrous stroma is seen to be markedly increased, and the lymphoid corpuscles, which are abundantly present in the normal gland, are replaced to a

large extent by cells with spindle-shaped nuclei, and endothelial cells. Giant cells with several nuclei are also seen in considerable numbers.

Tumours.—It is probable that only a small proportion of the malignant tumours which occur in the region of the thymus actually originate in that gland, rather than in the lymphatic glands in its vicinity. However, cases are met with in which tumours belonging to the class of sarcomata, carcinomata, and malignant teratomata, have apparently arisen in the thymus. Such an origin is suggested in some cases by the shape and position of the tumours, and in others by the presence in them of Hassall's corpuscles.

The clinical symptoms due to such tumours are pressure symptoms, whereas the physical signs are swelling above the sternum, dulness in the thymic area, and occasionally bulging of the manubrium. Of the pressure symptoms, respiratory stridor and enlargement of superficial veins are the most conspicuous.

In a case recently reported by Sheen, Griffiths and Schödlberg, of sarcoma of the thymus, in a boy aged seven years, the urgent dyspnoea due to pressure upon the trachea was relieved by division of the manubrium and partial removal of the tumour.

Acute Thymitis.—Acute inflammation of the thymus with suppuration has been met with occasionally, but it is probable that in not a few cases the collections of pus-like fluid described by Dubois, which are normally present in enlarged thymus glands, have been mistaken for true abscesses.

Dudgeon found a large abscess cavity in the right lobe of the gland of a rickety child who died suddenly, and in whose body no other pathological changes were present. The pathogenic organism in the pus was the pneumococcus.

Tuberculosis and Syphilis.—As regards the affections of the thymus gland due to tuberculosis and syphilis little need be said, save that in young children who have died of tuberculosis the thymus may contain milary tubercles, or be converted for the most part into a mass of fibro-caseous material (Dudgeon); and that small-celled infiltration and fibrosis, and more rarely gummata of the thymus, have been met with in children who succumb to congenital syphilis. The far wider influence which has been ascribed to congenital syphilis in the production of thymic lesions is almost certainly based, for the most part, upon misinterpretation of the morbid changes described. Neither tuberculosis nor syphilitic lesions of the thymus reveal their presence by any characteristic signs or symptoms.

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INFANTILISM.

The subject of infantilism may be dealt with appropriately in this place, seeing that some at least of its varieties are due to disturbances of the functions of glands of internal secretion.

By infantilism is meant a retention of the characteristics of childhood up to an unduly advanced age, or a retardation of the rate of development. Although it is manifested in its most pronounced form in individuals who, having reached the age of maturity, retain the contours and features of childhood, it may be observed at any period of childhood, as when a child of ten years has the development to be expected at the age of three. Infantilism is not necessarily associated with dwarfism, but may show itself less by arrest of growth than by retention of the childlike build and plumpness, by delayed ossification and absence of sexual development. Moreover, the structural delay may not be accompanied by any arrest of mental advance, and the child may resent being treated in accordance with its apparent age.

The study of infantilism is rendered more difficult by the adoption of classifications which cross each other. Thus, infantile individuals may be grouped according to their appearance, as into the well-known classes of Brissaud and of Lorisin. Those conforming to Brissaud's type exhibit a rounded fullness of the soft parts and large heads. Their appearance suggests hypothyroidism, which is probably the underlying cause, whereas those who belong to Lorisin's type have smaller heads, slim extremities, and slender bones.

A more satisfactory plan of classification takes account of the underlying cause of the infantilism rather than the outward appearance of the patients. Some such causes are now clearly discerned; others are suspected rather than established. In some cases, as in those classed by Hastings Gilford under the name of "osteosis," the underlying cause is as yet wholly obscure.

Of the varieties of infantilism classed as "symptomatic," that which develops in association with intestinal disorder is the best known. To it Herter was the first to call attention. The patients who exhibit it are, for the most part, sufferers from the colic complaint (see Chapter IV., p. 166), show protuberance of the abdomen, and pass large, pale, unformed motions which are unduly rich in fat. The resulting retardation of growth may be of any degree. The development of the child may only be retarded to a slight extent, or it may have the appearance of a child many years its junior.

Closely allied to the intestinal variety, if not identical therewith, is the pancreatic form of infantilism of Byrom Bramwell. Bramwell's patient was a youth of eighteen years, who in size and build resembled a child of eleven. He had suffered for years from chronic diarrhoea, with fatty stools. The chief evidence of a pancreatic lesion was afforded by the effect of treatment by pancreatic extract, which brought about a conspicuous diminution of the diarrhoea and an advance in stature and general and sexual development. Sahli's capsule test indicated a pancreatic defect, but further investigation of similar cases, with application of other tests of pancreatic efficiency, and especially estimations of the relative amounts of split and unsplit fats in the stools, are much to be desired. J. L. Rentoul has described a similar case in which pancreatic extract was given with an equal measure of success.

In a further small group of cases, infantilism has been observed in association with *hypertrophic cirrhosis of the liver*.

That deficiency of thyroid secretion is a cause of infantilism admits of no doubt. All cretins are infantile unless treated, and, as has been mentioned already, it is probable that most infantile subjects who conform at all closely to Basedow's type have some degree of hypothyroidism. Here again the strongest evidence of such origin is afforded by the effects of treatment by thyroid extract.

Pituitary infantilism, as manifested in Frolich's syndrome, is a well-defined variety, and, as we have seen, the failure of the pituitary secretion is capable of causing retrogression of sexual development in adult patients, an actual reversion to infantile conditions.

It has been suggested that in some cases of infantilism the *suprarenals* are at fault, and that their secretion is insufficient. The writer has met with a case in which a marked advance in development followed, and apparently resulted from, the administration of suprarenal tablets. This plan of treatment is worthy of trial in cases of obscure origin, but the evidence as yet available does not appear sufficiently cogent to justify the recognition of a definite suprarenal variety.

Considerable interest attaches to the association of infantilism with *polyuria*. Conspicuous arrest of development is not an uncommon symptom in cases of diabetes insipidus commencing in early life, and, seeing that polyuria is a result of some affection of ductless glands, it is not unlikely that such influences are at work in cases in which infantilism and excessive polyuria exist side by side. However, there appears to be another group of cases in which these symptoms coexist, in which the primary lesion is interstitial disease of the kidneys. H. Morley Fletcher recently described such a case, and others have since been placed on record by Leonard Parsons, R. Miller, and others. In some instances the clinical diagnosis of renal disease has been confirmed by post-mortem examination. (See also Chapter XII, p. 625).

Arrest of development is also seen in some cases of acquired heart disease in children, and is still more pronounced even in not a few cases of congenital morbus cordis. Nor must mention be omitted of infantilism as an accompaniment of mental defects, such, for example, as Mongolism and microcephaly.

The cases classed by Hastings Gifford under the name of *osteolosis* are characterized by the very conspicuous arrest of development which they exhibit, by their tendency to appear in successive generations of a family or in several members of a single generation. In them there are no indications of the seat or nature of the underlying lesion, and Hastings Gifford looks upon such individuals as freaks rather than as subjects of acquired disease. The subjects of osteolosis form the greater number of those who may be styled "professional dwarfs." Like the victims of achondroplasia, they present remarkable resemblances of feature, and agree in perpetuating into adult life the facies of babyhood. The head is relatively large, the face broad and flat, the limbs short, and trunk and limbs are infantile alike in dimensions and relative proportions. Gifford divides them into two subclasses—the sexual and asexual respectively. In members of the sexual class, puberty, although delayed, is reached eventually, the sexual organs mature, and the union of the epiphyses puts a limit to the tardy and incomplete growth. In the asexual cases puberty and the attendant bone changes are indefinitely postponed, and slow growth progresses up to the age of thirty years, or even later.

To the same observer we owe the description of a still more remarkable anomaly,

prognosis—even rarer—in which a child exhibits side by side the signs of infantilism and of premature senility, such as baldness, atrophy of tissues, arterial sclerosis, and general decrepitude.

Infantilism in its various aspects has much more than an academic interest, for, as we have seen, the condition is in some instances due to underlying morbid conditions which are more or less amenable to treatment. Whether or no these lesions be recognized in early life, and whether or no suitable treatment is applied, are matters of immense importance to the patients.

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CHAPTER XI

METABOLIC DISORDERS

A. E. GARROD

OBESITY.

DIABETES MELLITUS.

DIABETES INSIPIDUS.

GOUT.

EXCESSIVE EXCRETION OF METABOLISM:

ALKALOTONIA.

EXCESSIVE EXCRETION OF METABOLISM—CON-

STINUED: CYTOSINYL.

ACETONURIA—ACIDURIA.

LEUCURIA.

URICURIA.

PROSTHATIA.

OBESITY.

It is hardly possible to draw any sharp dividing line between ordinary fat children and those in whom the accumulation of fat is so excessive as to amount to an actual morbid phenomenon, for in respect of fatness children differ as widely among themselves as do adults, and examples of all intermediate degrees are met with. Nevertheless, for practical purposes it is not difficult to effect a grouping, if we take as our standard of excessive obesity a degree of fatness which incapacitates the patient seriously, hampers his activities, and renders him an object of general remark. Such obesity is rare, but is occasionally seen in children of all ages, more often in later than in early childhood. Very rarely it has been observed at the commencement of life, as in the case of a breast-fed child described by Heubner, whose weight at birth was 7.5 kilos (16½ pounds), and who when eight months old had attained to the enormous weight of 18.5 kilos (40½ pounds), which was obviously due to excessive deposition of fat. The obesity disappeared shortly after the child was weaned, and was attributed by Heubner to excessive secretion of milk by the mother, whose average daily supply was estimated at no less a value than 200 calories.

In children the deposits of fat tend as a rule to be more evenly distributed over head, trunk, and limbs, than in adults, but in certain rare morbid conditions a so-called "precocious obesity" is met with in children, whose contour resembles that of elderly obese individuals, and abdominal adiposity is a pronounced feature.

The factors which are concerned in the regulation of fat deposition are, on the one hand the intake of food, and on the other the expenditure in muscular exertion and heat production. Thus, one individual whose activities are normal may accumulate fat because his intake of food, and especially of fats and carbohydrates, is excessive, whereas another, whose appetite is normal, may become fat because his expenditure is lowered by a sedentary mode of life. In many fat children excessive intake is the responsible cause of obesity, and fat overfeeding either

greediness or the misdirected zeal of the parents, and their efforts to promote his nutrition, may be responsible.

Obesity, when once established, male ruin and; for by their hampering effects, and the increase of body-weight which results from them, the copious deposits of fat tend to restrict muscular exercise, and to diminish the expenditure of heat by conduction and also by radiation from the surface. Thus a vicious circle becomes established.

It is still an open question whether or no there be a variety of obesity which has its origin in an actual vice of metabolism—a *rectification de nutrition*, to employ Bouchard's term. In other words, whether there are individuals in whom the metabolic processes are so sluggish that, owing to that fact alone, the income outstrips the expenditure, even when the diet taken would not tend to induce obesity in normal persons. If so, their obesity is to be regarded as the outcome of an inherent morbid tendency rather than of erroneous habits of life. The doubt which still surrounds this question is mainly attributable to the difficulty and tediousness of the elaborate metabolic investigations required to establish the existence of such a metabolic defect. The observations available as yet are too few in number to serve as a basis for generalization, but they show clearly that such an explanation does not apply to all cases of obesity. The most elaborate investigations yet published are those of Rubner upon two brothers, aged ten and eleven years respectively, who were living under like conditions. The elder boy was thin and weighed only 26 kilos (57 pounds), whereas the younger, who was fat, weighed 41 kilos (90½ pounds). The elder was of an active temperament, whereas his fat brother was lazy and went to lie about. The observations showed that in the case of the fat boy the metabolic processes were not less, but actually were, active than those of his lean brother. However, other observations of a less complete kind, which have been carried out upon other obese persons, suggest that not a few fat persons, if subjected to similar rigid tests, would not respond as Rubner's patient did. Nor must it be forgotten that a slackening of metabolic activity of a slight degree might, if continuous, lead in the course of time to a conspicuous accumulation of fat.

It is obvious that members of certain families tend to obesity, and this suggests that they inherit an inborn anomaly of metabolism; but we must allow for the possibility that what the members share in common are habits of life which conduce to fatness.

It is difficult to avoid the conclusion that certain rare varieties of obesity which appear to be the direct results of disease of glands of internal secretion have their origin in disorders of metabolism, and it may well be that in the future a far wider influence in the production will be ascribed to such glands. As examples of such forms of obesity may be quoted that which follows the removal of the testes, with which may perhaps be associated that which develops at puberty or at the menopause in women, and the more extreme forms met with in patients whose thyroid or pituitary secretion is deficient, in children with tumours of the pineal gland or of the suprarenal cortex. Such obesity is wont to be associated with a conspicuously increased tolerance of glucose—at any rate, in cases of hypothyroidism and of hypopituitarism.

TREATMENT.—The treatment of obesity in children needs to be carried out with caution, for it must be remembered that during the period of active growth a certain degree of overnutrition is essential to such growth.

In many cases it is sufficient so to regulate the diet that any undue excess is avoided, but such a rigid dietary as is not infrequently prescribed for adult patients is likely to prove more harmful than beneficial to a child. Some observations by Hellesen upon a fat girl of the age of twelve years afford valuable guidance in this matter. His investigations showed that in the case of the growing organism it is difficult to guard against loss of nitrogen when the patient is placed upon a restricted diet. The loss of nitrogen is the outward sign of a breakdown of protein in excess of repair. Hellesen found that when the diet which maintained an even weight was reduced by two-fifths of its caloric value it was not possible to protect the organism from protein loss. When the caloric value of the food was reduced by one-fifth only, very different results were obtained, according as the reduction was made in the fat or carbohydrates. On a diet of protein and fat there was a loss of nitrogen, whereas on one of protein and carbohydrate there was an equal loss of weight, but at the same time a retention of nitrogen. Hence it would appear that reduction in weight by diet is best effected in a child by a moderate cutting down of fats, and this plan has the additional advantage that a diet of protein and carbohydrate satisfies hunger more than one of protein and fat. The restriction of diet should not be maintained over any long period.

Judiciously regulated exercise is also of much service in controlling obesity in older children.

Treatment by drugs is to be avoided, except in that special class of cases in which excessive accumulation of fat is due to thyroid insufficiency. In such cases the cautious administration of thyroid extract is indicated. In all other cases it is to be avoided.

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DIABETES MELLITUS.

It has been said of diabetes mellitus occurring in children, that "it gives no quarter," and this expression fairly describes the rapid and relentless course of the malady as seen in the earliest years of life. Diabetes is indeed one of the most grave of the diseases of children, but, fortunately, by no means common.

ETIOLOGY.—It has usually been reckoned that children below the age of eleven years contribute some 1 per cent. of all diabetic patients. Of 506 cases collected by Morley Fletcher from the records of St. Bartholomew's Hospital, 25 per cent. were in children of twelve years or under, and von Noorden found that, of 8,000 cases under his own observation, 28 per cent. had been in-patients under the same age limit.

Diabetes may have its onset in early infancy, and there are even records of children born with the disease. It is possible that such infantile cases are not rarely overlooked, and the diagnosis has doubtless been made on insufficient grounds in some instances.

The sex incidence among adults and children is remarkably different, for whereas amongst adult patients a considerably larger proportion are males, in childhood the two sexes suffer almost equally.

There can be little doubt that the group of symptoms to which the name "diabetes mellitus" is given has different pathological bases in different cases, and that we are in the habit of including under the name a group of maladies rather than a single disease. It is a familiar fact that cases of the more benign kind, in which glycosuria persists for years apart from any serious detriment to health, are chiefly met with among individuals who have entered upon middle life, and the elimination of the milder types of the disease is largely responsible for its gravity as seen in children.

Chronic changes in the pancreas, and especially fibrosis, are seldom met with among the child victims, in many of whom this gland shows no obvious signs of disease to the naked eye, and may even be passed as normal on microscopic examination.

LACTOSURIA.—It is not so widely known as it should be that milk-fed infants may excrete lactose, in quantities sufficient to yield the ordinary reduction tests. The tolerance of infants for that sugar varies within somewhat wide limits; and whereas in one case such tolerance is merely overtaxed by the too free addition of milk-sugar to the contents of the bottle, in others, and especially in those in whom gastric or intestinal disturbances are present, the tolerance is so far lowered that the comparatively small quantity contained in the milk suffices to overtax it.

It is important, therefore, to make sure, in breast- or bottle-fed infants, that the sugar excreted in the urine is actually glucose. Lactose reduces less readily, and, although dextro-rotatory, does not undergo fermentation with yeast within the period of twenty-four hours usually allowed. More positive evidence of the presence of lactose is afforded by Rubner's test. Lactosazone is more soluble than glucosazone, and, unless the quantity of lactose present be exceptionally large, no deposition of crystals occurs when the phenylhydrazine test is employed.

HEREDITY.—In a considerable proportion of diabetic cases hereditary tendency is well marked, but it is open to question whether such influences are specially conspicuous among children sufferers. It sometimes happens that several children of healthy parents succumb to diabetes, and von Noorden records instances among the offspring of consanguineous marriages. In a remarkable instance which has been placed on record by Langaker, no less than four children of a family of eight succumbed to the disease within a period of four years, of whom three were at the time of their death less than five years old. Five years later a fifth child of the same family fell a victim at the age of seven years. In this last case, the only one of the series in which an autopsy was made, conspicuous morbid changes were found in the pancreas. Langaker is inclined to ascribe this family *diabète* to infection from patient to patient rather than to hereditary influences. More often the child or children of a diabetic parent develop diabetes.

MODE OF ONSET.—The onset of the symptoms of diabetes in children not uncommonly dates from an injury, a febrile illness, or some trifling surgical operation, and, once manifested, the malady makes very rapid progress. Thus the writer has met with two cases during the past few years in which the whole period, from the development of the earliest symptoms noticed to death in coma, did not exceed three weeks. However, it must be remembered that diabetes is frequently overlooked in its early stages if the urine has not been examined for any cause, as witness the frequency with which an adult who believes himself to be in perfect health is rejected for life insurance on account of glycosuria. Undoubtedly, such

an initial period of freedom from symptoms, and apparent well-being, may occur in children also. Hence the actual date of onset of diabetes usually remains unknown, and it may well be that in not a few instances the apparent provoking cause really brings about an aggravation, rather than the development, of the disease.

The evil prognosis which attaches to diabetes in children is not materially modified by the fact that transient glycosuria is perhaps a more common event in childhood than in adult life, and is liable to be overlooked. Such glycosuria has been observed in the course of pneumonia, scarlatina, and some other infective maladies, possibly as a result of transient affections of the pancreas. For its recognition, daily examinations of the urine may be necessary, or, at least, examinations at short intervals. In the often-quoted cases of R. Schmidt, children, for the most part of diabetic parents who were under close observation, excreted sugar on a few successive days, were strictly dieted, quickly recovered their tolerance for carbohydrates, and retained it over long periods of observation. Häter, too, has recently recorded the case of a girl, aged ten years, in whom a temporary diabetes accompanied an intestinal catarrh with urticaria. After a short period the patient recovered completely, and passed no sugar even after a large dose of glucose. There may also be mentioned a variety of glycosuria, hardly to be classed as temporary, which is not uncommonly observed during the last few days of life in cases of tuberculous meningitis.

On the other hand, most of the reported recoveries of children from diabetes, a long series of which was collected by Stern, are found to rest upon evidence wholly insufficient, either as regards the nature of the sugar excreted or the transitory character of the condition.

SYMPTOMS.—The symptoms of diabetes as seen in children do not differ in any conspicuous respect from those exhibited by adult patients. The differences are rather of degree than of kind. Thirst and polyuria may be the first signs which attract attention, and in young children the large excretion of urine may lead to incontinence. This fact, that diabetes is an occasional, though rare, cause of enuresis, should not be lost sight of; for the earlier the glycosuria is recognized, the better is the chance of amelioration under treatment. In other cases the early symptoms are less suggestive of their true origin. The child is noticed to be listless, disinclined for play, and easily tired by any exertion. Later on there is conspicuous muscular weakness, wasting is common, and, as the disease advances, may proceed to emaciation. The skin is dry, and the tongue may acquire the "beefy," glazed appearance so familiar in adult diabetics. The *hæmorrhæ* may be lost at a comparatively early stage. Digestive disturbances are common, constipation is the rule, and diarrhoea, unless due to some definite independent cause, gives cause for anxiety, as it is a well-recognized precursor of coma.

In spite of the liability of children to tuberculous diseases, and of diabetic patients to phthisis pulmonalis, such accidents are not at all common among child sufferers. Some accidents met with among adults, such as gangrene, are practically unknown among diabetic children, but cataract has been occasionally observed in them. On the other hand, the special liability to pyogenic infections plays an even more conspicuous part in the diabetes of children than in that of older patients. A trifling prick from an apparently clean instrument may be followed by abscess formation, and extensive and widespread suppuration of subcutaneous tissues is sometimes the actual cause of death.

In the great majority of cases death occurs in coma, nor is this to be wondered at, seeing how specially liable children are to develop acetonaemic acidosis (p. 527). Indeed, this liability probably accounts, to a large extent, for the rapid termination and special gravity of diabetes in children. The onset of coma may be heralded by vomiting, and is often marked by severe abdominal pain, with or without diarrhoea. Such pain should always be regarded as of grave import in these cases. Drowsiness and air-hunger soon make their appearance, and the patient passes into complete coma. In adults the body temperature is usually not raised during diabetic coma, but rather tends to be subnormal; but in not a few cases of children there is con-



FIG. 63.—CHARTS SHOWING THE OCCURRENCE OF HYPERPYREXIA IN TWO CASES OF DIABETIC COMA IN CHILDREN.

spicious febrile disturbance, apart from any special complication to which it can be ascribed, and this may even attain to the grade of hyperpyrexia. The remarkable fall of eyeball tension during the stage of coma is as pronounced in children as it is in adults. It is possible that the group of symptoms which is associated with diabetic coma is not wholly due to a single cause, such as acetonaemic acidosis. In the case of a boy recently under the writer's care, death was attributable to widespread subcutaneous suppuration. The fatal ending was preceded by the abdominal symptoms mentioned above, but the urine had ceased to yield the iron reaction, and contained very little β -oxybutyric acid. There was no true air-hunger, and the patient remained conscious up to the end.

PROGNOSIS.—The gravity of the outlook of diabetes in children has already been sufficiently emphasized, but its course differs widely in different cases. In the earlier stages the glycosuria may be quite amenable to treatment, and in cases of older children, in whom dietetic measures can be carefully carried out, sugar may disappear from the urine as the diet is restricted, a conspicuous degree of carbohydrate tolerance may be recovered, and it may be possible to keep the urine free from sugar over periods of months. In other cases, even in the earlier stages, when no definite symptoms have yet developed, the scanty output of sugar may be little influenced by diet; and, as with adults, in cases of the grave kind, dietetic restrictions may have but little controlling action. Sooner or later the disease tends to assume the grave form, often with remarkable suddenness, and a child whose parents find it difficult to realize the gravity of his state, and chafe at the interference with his education, may pass into coma, with but little warning, from a condition of comparative well-being.

TREATMENT.—The principles of treatment are, of course, the same in children as in adults, but the special circumstances make it far more difficult to carry them into practice. The restrictions of diet which prove so irksome to adult sufferers are doubly unattractive to children, who wholly fail to realize their aim or utility. Moreover, a strict diabetic dietary, if persisted with over long periods, is apt to upset the digestive organs, especially of children. In dealing with infants, any rigid elimination of carbohydrates is out of the question, and in older children our treatment needs to be adapted to the individual case rather than to conform to strict rules. Further, in view of the special liability of children to acetonaemia, restriction of carbohydrates needs to be cautious and tentative, and to be regulated by the condition of the urine. A child in a very early stage of the disease, who exhibits no symptoms and excretes but little sugar, may develop a very pronounced acidosis when the diet approaches the limit of strictness, and may even become drowsy; whereas relaxation of diet may at once bring about the disappearance of the acetone bodies from the urine, in striking contrast to the effect of such relaxation in cases in which a true diabetic acidosis exists. In such cases the acetonaemia is merely such as always results from carbohydrate starvation, but reaches a greater degree because the patient is a child.

Langsten advocates the use of butter-milk in the feeding of diabetic infants, on the ground that it is poor in fat, which is probably the chief parent substance of the acetone bodies. A number of observers, including von Noorden, have found oatmeal a valuable article of diet for diabetic children. It should be pointed out that, when oatmeal is employed, it should be given from time to time for a few days or for a week or two, to the exclusion of other carbohydrate foods. Von Noorden advises that a child of seven should receive, during the oatmeal periods, some 150 grammes of oatmeal, 150 to 200 grammes of butter, 60 to 70 grammes of Roboust, and four or five eggs, daily. The better utilization of oatmeal than of other carbohydrate foods by diabetic patients is as yet an unexplained fact, accidentally found out, which rests on observation and experience. It is only manifested when no other carbohydrate is given. As part of a mixed carbohydrate diet oatmeal has no advantages. The chief advantage of such a diet is an increase of carbohydrate tolerance, often conspicuous, and a diminution of acetonaemia.

In cases of older children who are so circumstanced that an efficient diabetic dietary can be given, with beneficial results as regards recovery of tolerance and disappearance of sugar from the urine, the lines indicated are those followed with

adults. Our aim should be to allow an amount of carbohydrate food well below the maximum quantity which can be taken without provoking glycosuria.

Sodium bicarbonate is the drug chiefly required in the treatment of diabetic children. It should be given freely as a safeguard whilst reduction of carbohydrate food is being carried out, and still more freely by the mouth or rectum when coma threatens. There are even a few cases on record in which intravenous injection of a 3 per cent. solution of this salt has restored the patient, although death in coma has occurred sooner or later. The evil day has been merely postponed, and not averted.

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DIABETES INSIPIDUS.

The cases which are classed together under the name of "diabetes insipidus" do not belong to one definite category. Nor is this to be wondered at, seeing that they are characterized only by a pair of closely related symptoms—namely, polyuria and excessive thirst. Although the cases in which these symptoms occur bear a superficial resemblance to each other, they may own widely different pathological causes.

Ætiology.—Speaking generally, diabetes insipidus is a rare condition, alike in childhood and adult life; but the proportion of children sufferers is far larger than in diabetes mellitus.

Age.—About half of all cases are in patients between the ages of ten and forty years, but of 366 cases of diabetes insipidus, embodied in the published tables of four observers reproduced by D. Gerhardt, no less than 20 were in patients between five and ten years of age, and 30 were in children under five years old.

Sex.—Many more males than females exhibit the disease, but among children there is little, if any, preponderance of males. Of twenty-nine cases in children under fifteen, which Eichhorn collected, and in which the sex is stated, fourteen were in girls and fifteen in boys.

Heredity.—In some families the malady is strongly hereditary, and may reappear frequently in members of successive generations.

The quantity of urine excreted may be extremely large, even as much as 20 to 40 litres (35 to 70 pints) in the twenty-four hours; but smaller volumes, such as 5 to 10 litres (8 to 16 pints), are more common.

Owing to its extreme dilution, the urine is almost colourless, and has a very low specific gravity, usually 1.003 or 1.004, occasionally not above 1.004. Nevertheless, the output of solids is not below the average, and may even be in excess thereof. In severe cases the output of urine may be considerably in excess of the intake of liquid over long periods, the excess representing the fluid contained in solid foods. The output by the skin and lungs is naturally diminished. *Albumin* may be present in the urine in small amount, but usually is absent. *Isonit* has been

found in some cases, but its presence has probably no importance, and perhaps results from the abundant flushing of the tissues.

The *thirst*, which is the more distressing symptom, is proportional to the polyuria, as far as can be judged.

Primary and Secondary Cases.—A distinction must be drawn between secondary or symptomatic cases, in which the polyuria and polydipsia are dependent upon a definite lesion of the central nervous system, usually of the brain, and primary cases, in which there is no evidence of any organic lesion to which they can be ascribed. From the clinical standpoint this line is often not easy to draw; the boundary between the two varieties is somewhat blurred, so that differentiation may hardly be possible during life.

Of the lesions met with in symptomatic cases, gummatous meningitis calls for special mention. Amongst others are tumours in various situations, and especially in the neighbourhood of the fourth ventricle. Such lesions usually manifest themselves by revealing or localizing signs, such as paralysis of cranial nerves, optic neuritis, or bitemporal hemianopia.

The onset of the symptoms may be sudden or gradual. Sometimes it dates from an injury to the head or from some grave emotional disturbance, such as a severe fright. In not a few cases of children the onset has followed a specific fever; in others no exciting cause can be traced.

In one group of cases, persistent diabetes insipidus is associated with conspicuous arrest of bodily development, a form of *dysplasia*; so that a boy of sixteen or seventeen may present the appearance of a child of seven. As a rule there is no corresponding mental arrest. It appears probable that such arrest of development is rather the associated result of a common cause than a consequence of the urinary anomaly; for the polyuria may persist for years, and yet no such arrest occur.

Neurotic or neurotic-like symptoms may be present; there may be conspicuous anorexia or the appetite may be excessive. Progressive emaciation may occur both in primary and secondary cases. There may be no obvious constitutional disturbance. The skin is dry, but the saliva is not necessarily scanty. Optic atrophy has been observed in a few cases.

Polydipsia and True Diabetes Insipidus.—There can be little doubt that a further subdivision of the so-called "primary" cases is necessary—into those in which polyuria is the chief symptom, and those in which it is secondary to undue thirst or a morbid habit of taking liquid in excess. Patients of the one class are thirsty because they pass so much urine; those of the other class pass so much urine because they drink so much.

The work of Erich Meyer throws important light upon this distinction. He found that, whereas in some cases the administration of a large dose of sodium chloride is followed by a depression of the freezing-point of the urine, indicative of increased molecular concentration, in others the dose of salt causes no depression of the freezing-point, but a conspicuous increase in the output of urine. These latter he classes as examples of true diabetes insipidus, the essential error in which he holds to be inability on the part of the kidneys to excrete urine of more than a certain very low concentration. In order to insure the excretion of the solid waste, enormous volumes of water are required. The others he regards as examples of primary polydipsia. Some observations carried out by J. E. Roberts on children under the writer's care tended to confirm Erich Meyer's distinction, which was

based upon observations of adult cases; and clinical experience shows that, whereas in some cases a judicious limitation of the liquid intake can be carried out without causing distress, and with only good results, in others such restriction is not possible, in cases of the former class the polyuria is usually less in degree.

PROGNOSIS.—In the symptomatic cases the prognosis is much more grave than in those of the primary group, and death not infrequently results from the cerebral lesion. A few instances are on record in which death has occurred, apart from intercurrent disease, in cases of the primary class. The symptoms preceding the fatal ending have been progressive wasting and anæmia, diarrhoea, and finally coma.

In neurotic subjects, and when the disease follows some definite exciting cause, rapid improvement may occur, and even recovery, in the course of weeks or months. In other instances the polyuria persists for many years, it may be from childhood on into adult life, without any serious detriment to health. Primary polydipsia is the form more amenable to treatment.

Speaking generally, the prognosis of diabetes insipidus is good as to life, unless it be secondary to a serious cerebral or meningeal lesion, but less favourable as regards recovery.

MORBID ANATOMY.—In secondary cases the lesions found in the brain are of the various kinds already referred to, but in primary cases the only lesions found are those of a fatal intercurrent disease or such as are attributable to the polyuria, including hypertrophy and dilation of the bladder, and in some cases hypertrophy of the kidneys.

TREATMENT.—The treatment of diabetes insipidus is not attended with any conspicuous success. A cautious limitation of liquid intake may be tried, but the reduction should be very carefully watched. If the restriction causes any real distress, and is not responded to by a corresponding diminution of the polyuria, it should not be persisted with. It is better that no such attempt should be made than that it should be injudiciously carried out. In some cases, which are probably of the polydipsia variety, satisfactory improvement is effected. Erich Meyer advised a diet poor in meat, so as to restrict excretory products. A salt-free diet may be tried, and has given encouraging results in some cases (von Noorden). General tonic treatment, fresh air, and warm clothing, are indicated. Some of the drugs (e.g., opium) which have been recommended are such as one hesitates to give continuously to children. Valerian may be tried, but too much should not be expected from medicinal treatment.

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GOUT.

Although the diagnosis of gout in a child will only be arrived at with great caution and circumspection, there can be no doubt that the disease does occur. The best evidence forthcoming is derived from the fact that in a certain number of cases the first of a long series of definitely gouty attacks has occurred during the patient's childhood. In such cases the disease has usually manifested itself in one parent

or both. The occurrence of a single attack of inflammation of a great toe joint in a child does not justify the diagnosis, even although the affected joint may present the appearances usually seen in a gouty joint.

Sir A. Garrod narrates the case of a girl of ten who, when she came under his care, was suffering from a fourth attack. In the first attack, which occurred when she was seven years old, the joint of the right great toe suddenly swelled, was red and painful, and the trouble was ascribed to a sprain. Two years later there was a slight return in the same joint, and this was followed, after a further interval of nine months, by a third more severe attack, which spread to the dorsum of the foot. When seen the fourth attack was commencing, the great toe was hot and shining, whereas the rest of the foot was cool. Desquamation occurred later. The urine contained no albumin, was often thick on cooling, especially at about the time of the attacks. Heredity was not strongly marked, and the child had never taken alcohol in any form. During the following year she had several attacks, which involved one knee and one wrist. The same author refers to a second case in a girl of seven.

Duckworth records the case of a man of fifty who had had many attacks of gout, the first of which had occurred when he was eleven years of age. Soudastaceo, who expresses some scepticism in this matter, quotes a man whose mother suffered from gout, who "narrates, with such precision as to demand my assent to the fact, that he was attacked in the great toe at eight years of age." He gives a table showing the period of the first attack in 515 cases. In one case the age was eight, in one twelve, and in one fifteen.

The evidence available is not sufficient to permit of any generalization as to the character of gout as seen in children, nor of the formulating of any special rules as to its treatment.

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INBORN ERRORS OF METABOLISM.

There are certain rare disorders of metabolism, anomalies rather than diseases, which, since they are in almost all cases congenital, and persist throughout the patient's life, are as often met with in children as in adults. Therefore it is desirable that a brief account of them should be included in a work on diseases of children. Such anomalies are very rare, and it is probable that we are only acquainted with a few out of many examples, and only with those which advertise their presence in some conspicuous way. They are wont to occur either in several children of normal parents, or may be traced through several generations of a family.

Those which call for mention here are alkaptonuria and cystinuria. *Pentosuria*—the condition in which arabinose, a pentose sugar, is continuously excreted in the urine, and which has no obvious connection with glycosuria and diabetes—is probably a member of the group, but has not hitherto been detected in young children.

The writer has recently had under observation a boy of eight who has suffered from *abosterilia* from birth. His motions contain liquid fat, which solidifies

on cooling, but he shows no other signs of disease of the pancreas or other organ. Another child of the family showed the same peculiarity from birth, and there is reason to believe that we have here another metabolic anomaly of the class under consideration. We may expect that in time many other such anomalies will be recognized.

It is obvious that an error in chemistry, which may persist throughout a long life must be in itself little harmful, but in the case of cystinuria the tendency to the production of urinary calculi brings real dangers in its train.

Alkaptonuria.—This anomaly is characterized by the excretion of urine which, although it appears normal when freshly passed, soon darkens on exposure to air, passing through various shades of brown to actual blackness. Fabrics moistened with such urine become deeply stained, especially when dried at the body temperature, a peculiarity which calls attention to the condition even in young infants. Darkening occurs much more quickly in the presence of an alkali, and is attended by absorption of oxygen. As the urine stands, it tends to darken from the surface downwards. The urine reduces Fehling's solution readily when the mixture is heated, and reduces an ammoniacal solution of silver nitrate in the cold. With Nylander's reagent, darkening occurs from the action of the alkali, but no black precipitate falls. Hence alkaptonuria is apt to be mistaken for glycosuria, and its subjects, on that account, to be rejected when applying for insurance. When a very dilute solution of ferric chloride is added to such urine drop by drop, a deep blue colour appears for a moment as each drop falls. If the reagent be too concentrated, oxidation occurs too quickly, and this very characteristic reaction is not obtained. The reducing substance is not a carbohydrate, but an aromatic acid, homogentinic or hydroquinone-acetic acid, and its presence is due to a defect in the katabolism of the tyrosin and phenyl-alanin fractions of the proteins of the food and tissues. The large amount of investigation which has been devoted to this subject has thrown much light upon the nature of the metabolic error and upon the katabolism of proteins.

Alkaptonuria may make its appearance in several members of a family in which no such peculiarity is known to have occurred previously. In a large proportion of such cases the parents are cousins, and this and other peculiarities of its incidence are best explained by the Mendelian theory of heredity, and on the assumption that this error of metabolism is a recessive characteristic.

Alkaptonuric children grow and thrive like normal children, with no symptom save occasionally a slight dysuria. In later life they tend to develop the peculiar staining of cartilages and other tissues known as "ochronosis"—a phenomenon which may also result from applications of carbolic dressings to indolent ulcers over long periods of years. Moreover, chronic changes in joints, of the osteo-arthritic variety, have been observed in elderly alkaptonurics often enough to suggest a causal connection. No special treatment is called for, and although the output of homogentinic acid can be conspicuously reduced by limitation of the protein intake, such dietetic treatment, if long continued, would be calculated to do more harm than the anomaly itself.

Cystinuria.—It is probable that in the great majority of cases cystinuria is a congenital anomaly, in virtue of which a portion of the sulphur-containing fraction of proteins, cystin, is excreted unchanged in the urine. The condition is recognized by microscopical examination of the sediment, which consists of

characteristic hexagonal plates, the crystals of cystin, and by the calculus troubles to which the victims of cystinuria are unfortunately so liable.

The first cystin calculus, recognized by Wollaston in 1810, was removed from the bladder of a young child, and instances are known in which cystinuria has been transmitted through several generations, and has occurred in several children of a family. In a family observed by Cohn, no less than seven of a family of twelve children of a cystinuric father presented the anomaly; and Abderhalden has recorded the case of a child dying at the age of twenty-one months, a member of a family in which cystinuria had shown itself in three successive generations, in whose tissues and viscera abundant deposits of cystin were found. In some cases of cystinuria other protein fractions besides cystin have been excreted in the urine intact, such as tyrosin and leucin; and in not a few instances the urine has contained in addition the diamines cadaverin and putrescin, derived from the protein fractions leucin and arginin respectively.

No satisfactory treatment of cystinuria has been devised. Some have advocated the administration of alkalis, in the hope of retaining the cystin in solution, and so diminishing the tendency to calculus formation, and there is evidence that restriction of protein foods tends to diminish the output of cystin in the urine. Save for the risk of calculus formation, this metabolic anomaly appears to be harmless, but the inveterate tendency to form calculi which many of its victims exhibit, which is usually, if not always, associated with infection of the urinary tract, gives it a very real and grave clinical importance.

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ACETONÆMIA—ACIDOSIS.

The disturbance of metabolism which is spoken of as "acetonaemia," and which is manifested by the excretion in the urine of acetone, aceto-acetic acid, and β -oxybutyric acid, is much more readily induced in children than in adults.

In adults acetonaemia is induced by abstinence from, or deprivation of, food, or by elimination of carbohydrates from the diet, and may be regarded as an effect of carbohydrate starvation. It is not with in its most pronounced form in severe cases of diabetes. Diabetics suffer from carbohydrate starvation in virtue of the diminution of their power of utilizing carbohydrates, and the acetonaemia is increased when they are placed upon a strict diet, owing to withdrawal of such carbohydrate as even they are able to utilize.

In children acetonaemia is induced by very trifling causes, and in its slighter forms may result from apparently insignificant changes of dietary. Thus, R. S. Frew, who has recently published the results of the examination for acetone of the urine of more than 600 children admitted to the wards of the Hospital for Sick Children, found acetone, in larger or smaller quantities, in the urine of no less than 61·6 per cent. of the patients during the three or four days following their admission. The maximum reaction was as a rule obtained in specimens passed some thirty-six hours after the child was admitted, and after that time the acetonaemia tended to disappear. An analysis of his observations showed that the

acetonæmia following admission to hospital been no obvious relation to the diseases from which the patients were suffering; that it was more readily induced in younger than in older children, but in children under one year it was relatively less frequent, except in cases in which a change from breast feeding to artificial feeding had occurred on admission to hospital. Few attributes the temporary acetonæmia to change of diet, and to a temporary loss of power of assimilating carbohydrates due to such change. After some three or four days the child's digestion becomes adapted to the change of diet, the more readily the less the change, and this explains the relative immunity of infants, whose diet consists almost wholly of milk. In older children the acetonæmia cannot be ascribed to any reduction in the quantity of carbohydrate food given, but to a change in its quality.

Among the morbid conditions in which the more severe forms of acetonæmia are observed in children are those in which vomiting is a conspicuous symptom, such as that obscure affection to which the name of *cyclic vomiting* is applied, and the very grave state known as *delayed chloroform-poisoning*, which may follow the administration of other anesthetics besides chloroform. It were easy to ascribe the effect to starvation resulting from the rejection of all food, but there are serious difficulties in connection with such an explanation, not the least of which is presented by the fact that in some cases of persistent vomiting in children acetonæmia is absent, whereas in others its appearance is simultaneous with, or may even precede, the onset of the vomiting.

Acetonæmia is a prominent symptom in some cases of pneumonia and bronchopneumonia in children. Occasionally the odour of acetone is detected in the breath, and it has appeared to the writer that there is a close association between the acetonæmia and the drowsiness and apathy which the patients not uncommonly exhibit.

In diabetic children acetonæmia is usually pronounced, and one is inclined to believe that the specially grave course of diabetes in children, and the shortness of the period which often elapses between the appearance of the diabetic symptoms and the superintention of coma in young subjects, may be due in great part to the readiness with which acetonæmia is induced in them.

The administration of large doses of drugs of the *salicylic* group may be mentioned as another important cause of acetonæmia in children.

THE DETECTION OF ACETONÆMIA.—As has been mentioned already, the odour of the patient's breath may proclaim the presence of acetonæmia, but the examination of the urine reveals much slighter grades of the condition.

For the detection of acetone in urine Rothemann's test is to be preferred to others. It is carried out as follows: 5 c.c. of the urine are saturated with ammonium sulphate by shaking it with the crystalline salt; 2 c.c. of a solution of ammonia are added, and afterwards a few drops of a 5 per cent. solution of sodium nitroprusside. If acetone be present, the liquid acquires a rich purple colour, and the length of the interval before the colour appears, and the intensity of the tint, afford some indication of the quantity of acetone present. It is more convenient to add to the urine an equal quantity of a saturated solution of ammonium sulphate, instead of saturating with the solid salt, a simplification which does not impair the delicacy of the test.

For the detection of aceto-acetic acid the well-known test of Gecharlt is employed—namely, the appearance of a brown or purple colour when a solution of

ferric chloride is added to the urine. It must be remembered that the salicylic drugs yield a similar reaction; however aceto-acetic acid is destroyed if the urine be heated for a few minutes in a water-bath, whereas salicylic acid is not affected by such treatment.

No clinical test for the detection of β -oxybutyric acid is available, and recourse to somewhat complicated laboratory methods is necessary.

In the slightest cases evidence of the presence of traces of acetone alone may be obtained, and the iron reaction may fail. In the most severe cases all three members of the group are present, abundance of β -oxybutyric as well as aceto-acetic acid. Adults with severe diabetic acetonaemia may excrete as much as 30 to 70 grammes of β -oxybutyric acid in the course of twenty-four hours.

SYMPTOMS.—It is believed that the symptoms of acetonaemia are not due to any specific toxic effects of the members of the acetone group, but rather to the action of the aceto-acetic and β -oxybutyric acids as acids, causing depletion of the fixed alkalies of the tissues. The organism averts this drain of alkalies to some extent by supplying ammonia for neutralization, a well-known protective mechanism which is also called into play when any acid is administered in excess. Hence a greatly increased excretion of ammonia in the urine accompanies acetonaemia, and its amount affords some measure of the degree of acidosis.

The symptoms which accompany the more severe degrees of acetonaemic acidosis, whatever its cause, are those which are seen in diabetic coma, with conspicuous loss of eyeball tension, and that peculiar form of slow, deep breathing to which Kussmaul gave the appropriate name of "air-hunger." Except in diabetic cases, the stage of actual coma is very seldom reached.

In most conditions in which fatal acetonaemia is met with, the liver is found at an autopsy to be the seat of a pronounced fatty change, and this is a particularly prominent feature in "delayed chloroform-poisoning." It is still very uncertain in what relationship the changes in the liver and the acetonaemia stand to each other. It has been held by some that the change in the liver is the exciting cause of the metabolic disturbance, and of the resulting acidosis; others maintain that the change in the liver and the acetonaemia are alike results of toxic influences exerted upon the tissues; and, lastly, it is suggested that the hepatic lesion is a result of the acidosis. Our knowledge does not yet suffice to enable us to decide between these several explanations.

TREATMENT.—Two methods of treatment are available in cases of acetonaemia—viz. the administration of carbohydrates in easily assimilable form, such as glucose or levulose, and the administration of alkalis to neutralize the excess of acids. In cases of persistent vomiting, glucose may be given in enemas. In diabetic cases a relaxation of diet is indicated, but the result of such relaxation is usually disappointing.

Alkali is best administered in the form of sodium bicarbonate, either in large doses by the mouth or per rectum, or by the infusion of a 3 per cent. solution of the salt. Such treatment is more effectual in averting the symptoms of acidosis than in combating them when developed, and, accordingly, when there is any cause to anticipate the development of acetonaemia, as during reduction of diet in a diabetic case, sodium bicarbonate should be freely administered as a safeguard. Large doses of salicylates, especially when given to children, should be guarded by still larger doses of sodium bicarbonate. Steps should also be taken

to correct a tendency to constipation, which appears to favour the development of acidosis. Similar measures may be adopted as a preparation for the administration of an anæsthetic to a child, in the hope that the risk of delayed anæsthetic poisoning will thereby be diminished.

The best results are obtained in cases in which the acidosis is due to an extraneous toxic substance, such as a salicylic drug, the administration of which can be stopped. In cases of delayed anæsthetic poisoning the effects of treatment are wont to be very disappointing.

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LITHURIA.

Deposits of urates and crystals of uric acid are common in the urine of children. Even infants often excrete minute crystals of uric acid which may be grouped into wart-like collections or may form soft calcareous masses.

R. A. Gibbons (*Med-Chir. Trans.*, 1896, lxxix, 31) has described attacks of renal colic in infants, mostly boys, followed by the passage of large numbers of crystals of uric acid, and of soft masses of such crystals held together by mucus. The symptoms observed were paroxysms of abdominal pain, recurring at short intervals, with tenderness over one kidney and retraction of the testicle of the same side. In addition to the crystals of uric acid, the urine contained small quantities of albumin and blood-corpuscles. Gibbons gives the notes of six cases in all, but the condition must be a very uncommon one, and it will be necessary, when confronted with such cases, to exclude infection of the urinary tract by *Bacillus coli*, which may produce somewhat similar symptoms.

The writer has met with a few cases of leucaturia in infants, associated with, and probably due to, excretion of crystalline uric acid. In the cases in question there was no reason to suspect infantile scurvy, which usually is accompanied by hæmaturia of greater or less degree.

In cases of infants who pass crystalline uric acid, dietetic treatment is out of the question, and milk is practically purin-free; but older children may be placed on a diet poor in purin substances, with a view to reducing the exogenous uric acid. Of drugs, alkalis, such as potassium bicarbonate or citrate, are indicated.

OXALURIA.

The familiar octahedral crystals of calcium oxalate are frequently present in the urine of children. They may form an opaque white layer upon the surface of the tubercula, and cling to and emphasize the grease marks upon the inner surface of the containing glass.

It is uncertain whether such oxaluria is ever due to a metabolic error, manifested by an increased excretion of endogenous oxalic acid. It is certain that the deposits may occur apart from excessive output, as the result of conditions

unfavourable to the holding of calcium oxalate in solution, such as diminished acidity, or the abundant presence of calcium salts; and scanty presence of salts of magnesium. It is probable that in most instances the deposition of oxalate crystals is due to dietetic causes; and when rhubarb has been freely eaten, the abundant excretion of calcium oxalate which results may be attended by conspicuous hæmaturia. The possibility of such an origin should be thought of in connection with any obscure case of hæmaturia, either in a child or an adult. In some persons the eating of rhubarb is always followed by hæmaturia.

However, the chief importance of oxaluria is due to the fact that it is a common, if not the commonest, component of urinary calculi.

Treatment, when called for, should be directed to the limitation of the intake of oxalic acid, mainly by reduction of vegetable foods, and to the increase of magnesium and decrease of calcium in the urine. The vegetables chiefly to be avoided are rhubarb and spinach. Tea and cocoa also are rich in oxalate, whereas coffee has the advantage of being poor in oxalate and lime, and rich in magnesium. Some vegetables, such as cauliflower and cabbage, which contain little oxalate, are rich in lime and poor in magnesium. Peas, beans, potatoes, and apples, on the other hand, fulfil both requirements. In the treatment of children, a difficulty is created by the fact that milk and eggs are, of all foodstuffs, those which contain most lime. Meats of all kinds fulfil the required conditions, and may be taken freely. However, it is only in exceptional cases that children need to be dieted on account of oxaluria, save by the exclusion of rhubarb from the diet.

PHOSPHATURIA.

When a patient passes urine which is alkaline from excess of fixed alkali, and not as the result of ammeniacal decomposition, and turbid from precipitation of earthy phosphates, he is said to suffer from phosphaturia. The taint is an unfortunate one, because there is no excessive output of phosphoric acid in such cases; indeed, such an excess would render the urine unduly acid, and effectually prevent precipitation of earthy phosphates.

Healthy persons frequently pass alkaline urine some hours after a meal, during the period of the so-called "alkaline tide," and vegetivorous animals habitually pass such urine. A like effect is produced by an increased intake of alkali in mineral waters or medicines, and, again, may result from a relative diminution of acids, as when the hydrochloric acid of the gastric juice is lost by vomiting.

When the phosphaturia is continuous, and is not to be explained in any of the above ways, some underlying cause is to be sought for. It is generally conceded that the influence of the nervous system has an important share in the causation of many cases, and the phosphaturia of neurasthenic individuals, and of those who suffer from continued worry or overwork, is a well-recognized variety. The apprehension which this relatively unimportant symptom causes to such patients tends to aggravate the primary morbid condition, for phosphaturia is wont to assume a wholly undue importance in their minds.

Calcaruria.—There is a variety of phosphaturia, met with in children, which calls for special attention. This form, originally described by Sendtner, has been thoroughly investigated by Soetbeer, who showed that the cause of the alkalinity of the urine is an increased excretion of calcium by the kidneys, in virtue of which

the urine is alkaline and throws down an abundant deposit of normal calcium phosphate, on which account medical advice is sought. In a comparative investigation carried out upon a normal and a phosphaturic child, the urine of the latter was found to contain nearly three times as much calcium as that of the former. In normal persons the bulk of the calcium is excreted by way of the intestine, and only a comparatively small fraction by the kidneys. In the cases in question this ratio was disturbed, and although the daily output of calcium was not increased, a relatively large proportion of it was excreted by the kidneys. Soetbeer ascribes the impairment of the intestinal excretion to a colitis, and regards the urinary abnormality as merely a secondary result of the intestinal disorder. However, as Langstein has shown, it is by no means possible to class all cases of phosphaturia in children in this group, and he has met with "calcururia," to employ the uncouth name suggested by Soetbeer, in cases in which there were no clinical evidences of intestinal disturbance. Nor is calcururia met with in children alone.

TREATMENT.—In cases of phosphaturia of the nervous varieties, tonic drugs and hygienic measures directed to the improvement of the general health, and to increase of nervous stability, are called for; but the effects of treatment are often disappointing. Acids may be given, and, as R. Hutchison has pointed out, the most efficient of these is acid sodium phosphate. A drachm or two of the salt may be dissolved in a pint of water, and the solution may be given in frequently repeated small doses.

The best results of treatment are obtained in cases of calcururia, in which the urine may usually be restored to its natural condition by limiting the intake of calcium by regulation of diet. The foodstuffs richest in calcium are milk and eggs, whereas meats, farinaceous foods, and those vegetables which are poor in calcium, such as potatoes, and apples, peas, beans, and carrots, may be taken freely. If there be any symptoms indicative of intestinal disorder, treatment should also be directed to restoring a normal condition of the bowel.

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CHAPTER XII

DISEASES OF THE GENITO-URINARY SYSTEM

J. S. FOWLER

THE URINE IN HEALTH AND DISEASE:	NEPHRITIS—continued:
POSTURAL ALBUMINURIA.	CHRONIC INTERSTITIAL NEPHRITIS.
HÆMATURIA.	NEPHRITIS IN OLDER CHILDREN.
HÆMOGLOBINURIA.	URÆMIA.
PYELIA.	BACTERIAL INFECTIONS OF THE URINARY TRACT.
ENTERIA.	TUMOURS OF THE KIDNEY.
MALFORMATIONS AND MALPOSITIONS OF THE KIDNEYS.	URINARY CALCULI.
FLOATING KIDNEY.	TUBERCULOSIS OF THE KIDNEY.
CYSTIC KIDNEY.	MALFORMATIONS OF THE BLADDER AND THE GENITAL ORGANS.
HYDRONEPHROSIS.	ORCHITIS, EPIDIDYMITIS.
NEPHRITIS:	VELLO-VAGINITIS.
NEPHRITIS IN INFANCY.	

THE URINE IN HEALTH AND DISEASE.

THE kidneys are functionally active during the later weeks of intra-uterine life, and the bladder has been found distended with urine at birth. According to Ballantyne, it contains little urea (0.15 per cent.), uric acid is relatively large amount, chlorides, creatinin, and sometimes albumin. During the first two or three days of life the secretion of urine is scanty, and none may be passed during the first twenty-four hours. The urine which is passed immediately after birth is usually pale-coloured and clear; from the first day onward, however, until the eighth or tenth day, it is darker and somewhat turbid. Thereafter it again becomes clear, and has a pale yellow colour, which slowly darkens as childhood advances. The turbid darker urine of the first week or ten days corresponds with the period of urtic infants and albuminuria of the new-born (see Chapter I., p. 76).

Quantity of Urine at Different Ages.—After the first few days of life the quantity of urine secreted is relatively greater than in the adult. The variations are, however, considerable, and most of the figures given are only approximations to an average. In a series of eleven children from twelve to twenty-four months, none of whom were suffering from any disease calculated to affect the kidneys, and all under the same conditions in hospital, the urine was carefully collected for a period of forty-eight hours. The daily quantity varied from 7½ to 19 ounces, with an average of 11½ ounces. In some older children the results were nearly as variable.

The following table gives an idea of the normal limits of variation :

Age.	Minimum.		Maximum.	
	C.C.	Os.	C.C.	Os.
1st day	2	0.70	41	2.1
2nd	11	0.38	145	5.1
3rd	15.5	0.47	171	6.0
4th	17.5	0.60	179	6.3
5th	22.5	0.80	222	7.5
6th	70	2.30	240	9.8
7th	93	3.30	338	11.9*
1st month	150	5.30	400	14.0
2nd to 5th month	210	7.40	500	17.5
6th .. 12th	250	8.70	600	21.0
2nd .. 4th year	300	11.50	800	28.0
5th .. 7th	600	27.00	1200	42.0
8th .. 14th	1000	35.00	1500	52.5†

* Boiling.

† Boil.

While the quantity of urine depends to a considerable extent on the amount of fluid taken, the ratio to the intake of milk is less in breast than in artificially fed infants. Still has worked out a formula for calculating the average quantity of urine passed by a child from the fourth year onwards : Multiply the age in years by 2.5 ; the product is the daily amount in ounces. This formula gives a somewhat lower average than the above table.

Frequency of Micturition.—The younger the child the more frequent is urination. During the first two years a child may pass urine every hour when awake, and retain it for from two to six hours during sleep ; during the third year it is passed every two or three hours during the day, and may be retained eight or nine hours at night. Normally, control of the act is gained about the end of the second year.

Reaction.—In the newly-born infant the reaction is nearly always acid, and often markedly so, especially during the infarction period—(acid 108, neutral 11, alkaline 0—Fleisberg). Throughout the rest of infancy it is faintly acid or neutral.

Specific Gravity.—After birth the specific gravity rises ; by the sixth day it has fallen, but then rises steadily until puberty. Holt's collected table shows this :

1 to 3 days	1.010 to 1.012
4 to 10 days	1.004 — 1.008
10 days to 6 months	1.004 „ 1.010
6 months to 2 years	1.006 „ 1.012
2 to 8 years	1.008 — 1.014
8 to 14 years	1.012 — 1.020

Urine of the Infarct Period.—What may conveniently be called the "infarct urine" is that which is excreted during the first eight or ten days of life. It differs considerably from the urine of other age periods, and these differences are due to the adjustments which are now taking place between the foetal organs and their extra-uterine environment. The urine of this period has been studied by many investigators, who, though they have demonstrated its main features

have not succeeded in fully explaining them. Infarct urine is somewhat dark-coloured, leaving a yellowish stain and slight sandy deposit on the napkins; it often contains albumin, and sometimes casts.

Albuminuria in the New-born.—Traces of albumin are very frequently present in the urine of young infants. Flenburg examined the urine of 155 infants daily for the first fortnight, and found albumin constantly present during the first four days, and occasionally up to the fourteenth. The occurrence of albuminuria seems to be uninfluenced by the nature of the labour, the sex of the child, the stage of development, or the parity of the mother. It coincides generally with the occurrence of infarcts, and is usually most abundant if there is a copious deposit of urates, but infarcts occur without albuminuria, and vice versa. Albumin persists after the infarcts are gone. The nature of the albuminous substance is unknown; some state that it is mucin, but Flenburg regards it as a nucleo-albumin, and Langstein as albumin or globulin. It has, however, no clinical significance.

Casts.—Hyaline casts are not uncommon during this period. It seems that during foetal life an albuminous substance accumulates in some of the renal tubules, and is excreted as casts. These are not signs of nephritis, and apparently form the basis of the urate infarcts as mentioned below. Granular casts may also occur.

Infarcts.—On splitting open the kidney of a newly-born infant, yellowish-brown radiating lines are often seen, and sometimes also yellowish granular masses in the kidney pelvis. These consist of ammonium urate and uric acid, chiefly the former. They were formerly thought to be proof of live birth, but the fact that they occasionally occur in stillborn infants has robbed them of all medico-legal importance. The uratic infarcts begin to appear in the urine towards the end of the first day, and are not usually present after the sixth day, although they may still be found in the kidneys during the second week or later. Flenburg has shown that the urates forming the infarcts do not encrust the epithelium lining the tubules, but are deposited on albuminous or hyaline casts lying free in the lumen. The urine secreted towards the end of the first day is concentrated, and contains in particular a large quantity of uric acid, and it is supposed that the hyaline casts, mechanically or otherwise, cause a precipitation of ammonium urate from the concentrated urine. The excessive output of uric acid is perhaps associated with the transient leucocytosis which normally takes place after birth.

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Urine of Later Infancy and Childhood.—After the first few days of life the urine does not differ greatly from that of the adult. The excretion of nitrogen cannot be profitably studied except as a part of the general metabolism, and for this reason statements as to the average excretion of urea, etc., are valueless, especially when not accompanied by any particulars as to the quantity or quality of the food. Most of the nitrogen (80 to 85 per cent.) is excreted as urea; the amount of ammonia-nitrogen (ammonia coefficient) rises in many acute nutritive disturbances, on account of the liability of children to acidosis. Apart from the excess of uric acid in the early days of life, there is no "normal" uric acid output in children; in any given case the quantity has to be considered in relation to the

diat. The excretion of phosphoric acid is considerably greater in hand-fed than in breast-fed infants. In older children phosphaturia (q.v.), characterized by milky urine, sometimes occurs. Oxalic acid is excreted in absolutely greater quantity than in the adult. Ethereal sulphates are present in very small amount in the urine of infants; phenol is more abundant in artificially-fed than in breast-fed children; indican and urobilin are also said to be absent from the urine in breast-fed children, and to be present in that of the artificially-fed.

The urine of infants contains a reducing substance. Langstein found lactose and galactose in breast-fed children suffering from gastric disorder. Other sugars are said also to have been detected. Glycosuria of this nature is of no clinical importance.

To the practitioner the chemistry of the urine is not of much assistance. The principal metabolic disturbances leading to anomalies of the urinary secretion are treated of elsewhere (Chapter XI.).

Collecting Urine for Examination.—In infants and young children micturition can often be induced by applying a cold sponge to the pubic region, or by pressing firmly over the bladder. To collect all the urine of an infant is not easy. In little boys the barrel of a small glass syringe, or a test-tube, may be fixed over the penis with a piece of strapping; for little girls a specially-shaped funnel, devised by Grossmann, is sometimes used. The method of collecting urine in a pad of absorbent wool placed over the genitals, and subsequently squeezed out into a glass, is not satisfactory. If aseptic precautions are taken, there can be little objection to obtaining a specimen of urine by means of a catheter.

Albuminuria.—It is not uncommon to find a trace of albumin now and again in the urine of infants and young children who are ailing. It may occur in the course of any febrile affection or in gastro-intestinal disturbance. The amount is usually small, casts are absent, and no serious import attaches to its occurrence. In addition to this, cases of so-called "functional" or "paroxysmal" albuminuria occur, in which, however, albuminuria continues for long periods. The most important variety of this disorder is called "postural albuminuria," and probably most of the other types—albuminuria after cold baths, after severe muscular exercise, etc.—are allied to it.

Postural, Cyclic, or Orthotic Albuminuria.—That albuminuria may occur apart from nephritis is a well-established fact. It was recognized by Gull, and later by Moxon, who associated the condition with adolescence. In 1885 Pavy described it as "cyclic albuminuria," and in 1887 it was more accurately defined by Stirling as "postural albuminuria." From these three designations we perceive its leading characters: it occurs in youth before and during the age of puberty, and tends to disappear as adult life is attained; the urine is intermittently, not constantly, albuminous; the appearance of the albumin depends on the position of the patient—it is absent when he is lying down, present when he is up. As the last peculiarity is the most striking, the term "postural albuminuria" is that generally employed in this country—"orthostatic," or "orthotic" in Germany. The epithet "orthotic" (from *ὀρθός*, to erect) was coined by Heubner to convey its nature more precisely, because, although under certain circumstances the albuminuria disappears while the patient remains on his feet, it is invariably produced by the transition from the lying to the erect posture—by the act of rising. More recently the term "lordotic albuminuria" has been applied to it.

Ætiology.—Little or nothing is known as to the cause of postural albuminuria. In some cases it is a family peculiarity. As many of the subjects are naturally delicate or below par, a constitutional weakness has been blamed, but of this we have no real knowledge. Postural albuminuria does not occur in early childhood, and, although isolated cases have been reported during the third and fourth years, the condition is rare before the fifth and sixth years, after which it gradually increases in frequency up to the age of puberty. Girls are somewhat more frequently affected than boys. Postural albuminuria is of relatively frequent occurrence. Clement Dukes found 157 cases of albuminuria among 1,000 schoolboys between the ages of thirteen and fifteen years. Götsky, among 4,189 children between five and fourteen years attending Heubner's clinic during two consecutive years, found 5.7 and 4.4 of cases respectively.

Symptomatology.—Apart from the presence of albumin in the urine, the symptoms which these patients present are decidedly indefinite; there may, indeed, be none of any description. In some cases the children are robust and in good general health, but as a rule they are delicate or below par. They are usually pale, and often a little puffy about the face; examination of the blood, however, shows no anemia. They are languid, readily tired, and disinclined for work or play. Headaches are not infrequent. Sutherland describes a group of symptoms, corresponding to neurasthenia in the adult, as being often associated with albuminuria. The children show signs of overstrain; lessons are a burden, play causes fatigue, and there is lack of control of the emotions. The heart sounds are feeble, and the pulse is weak. There may be a tendency to chillblains or fainting attacks. Headaches and epistaxis also occur, and there are often digestive disturbances and constipation. The most characteristic symptoms are connected with the circulation. In children of school age who are ailing slightly it is not uncommon to find a snapping apex beat, together with somewhat sharp or impure heart sounds and irregular cardiac action. Along with this the child is easily tired, and perhaps breathless on exertion. In many, but not all, cases of this kind, albumin can be found in the urine. The above signs suggest some slight cardiac dilatation, but it is doubtful whether this exists, and in any case it is temporary. Reyher, indeed, found that in most cases of postural albuminuria the heart (by the orthodiagraph) was small. Dukes made the interesting observation that boys who were liable to faint while standing at morning prayers at Rugby School were almost without exception albuminuric and had high-tension pulses. Heubner has found that the systolic and diastolic pressures are high in these patients, and also states that the electro-cardiogram of an eleven-year-old girl showed the so-called "infantile notch," which normally disappears towards the end of infancy, and is found in adult life only in neurasthenic persons. There is thus a considerable amount of clinical evidence that in these cases the circulation is abnormal.

Urine.—The essential feature of the urine is that, while a specimen passed in the morning before rising is free from albumin, that which is passed after rising invariably contains albumin. The urine has generally a rather high specific gravity (1.010 to 1.030); it is acid, often markedly so, or occasionally neutral, and frequently deposits oxalates. Uric acid crystals and phosphates are sometimes also found. The amount of albumin varies from day to day, and also at different times of the day. It may reach its maximum in the morning hours, or not until evening. It may be a mere trace, or as much as 4 to 5 per 1,000. A few

hyaline casts have also been found in some cases, but their presence always suggests the possibility of nephritis, which fails to be considered in connection with the diagnosis. During the albuminuric period the urine is lessened in quantity. The albumin diminishes after exercise and after taking food.

More than one proteid is usually present in the urine. The most characteristic is a body which is precipitated by acetic acid in the cold; serum albumin and globulin are also usually present. The nature of the body which falls down on adding acetic acid is disputed; it has been regarded as *uracin*, as *nucleo-albumin*, and as *euglobulin*. Whatever it be, it is generally more abundant than the others, which is not the case in Bright's disease. Langstein, who has specially studied the urine in these cases, describes three types: (1) Those in which the acetic acid precipitate alone is present; (2) those in which there is also true albumin; (3) those in which both albumin and globulin are also present. He recommends the following method of testing: Two specimens of urine are taken, and to each is added a few drops of acetic acid; after thorough shaking the urine is diluted with three or four times its bulk of water, whereupon a cloud appears. The precipitate does not form immediately on the addition of acetic acid. To one specimen is then added solution of potassium ferrocyanide, which precipitates the albumin. The condition can also be recognized by the ordinary tests for albumin.

PATHOLOGY.—The hypotheses in explanation of postural albuminuria fall under four heads: (1) That it is due to nephritis; (2) that it is a metabolic disease; (3) that it is of vasomotor origin; (4) that it is of mechanical origin.

Nephritic Hypothesis.—As the disease is not directly fatal, few cases have been studied post mortem. Heubner, however, was able to examine thoroughly the kidneys of a typical case which had been under prolonged observation during life, and found not the slightest evidence of nephritis. It is therefore certain that this form of albuminuria can exist without nephritis, and it is extremely unlikely, considering the clinical course of the disease, that there is any organic lesion of the kidneys in true orthotic albuminuria.

Metabolic Hypothesis.—According to this hypothesis the albumin of the urine is a blood-albumin which has lost the power of being assimilated, and is excreted by the kidneys (diabetes albuminosis—*van Noorden*). The comparatively frequent occurrence of exhalosis in these cases is brought forward in support of this hypothesis, but except for this there is little or no evidence of faulty metabolism.

Vasomotor Hypothesis.—The most widely accepted hypothesis is that postural albuminuria depends on some disturbance of the renal circulation, brought about by vasomotor influence. The circulatory symptoms shown by many patients are suggestive of some cardio-vascular instability. The most exact work in this direction has been done by Erlanger and Hooker, on an albuminuric and a normal subject, in connection with their study of blood-pressure. By varying the conditions—*e.g.*, by compressing the legs and abdomen in Crile's pneumatic suit—they showed that the erect posture was not in itself responsible for the appearance of albuminuria, and the upshot of their experiments is the demonstration that the only circulatory factor influencing the output of albumin is the pulse-pressure (ratio of systolic to diastolic pressure). When this diminishes (as it normally does with the assumption of the erect position) albumin appears. When the subject was placed erect, yet (owing to the experimental conditions) did not excrete albumin, the pulse-pressure was found to remain high. Acts which raised the pulse-pressure, such as eating or moderate exercise, checked or diminished the albuminuria. The

output of albumin varied inversely as the pulse-pressure. From these facts it seems that orthostatic albuminuria may originate primarily from a disturbance of the vasomotor mechanism. The act of rising from the lying to the standing posture tends to cause the blood to gravitate into the lower part of the body, and this is normally compensated by vasomotor influence. In orthostatic albuminuria this regulating mechanism is apparently at fault.

Mechanical Hypothesis.—Jehle makes the ingenious suggestion that postural albuminuria is due to lordosis of the lumbar spine, which causes pressure on the vessels of the kidney. Like Erlanger and Hecker, he showed that bending the body forward checked the albuminuria. By fixing the spine to prevent lordosis, he enabled albuminuric subjects to stand erect without excreting albumin, while, conversely, by fixing the spine in a position of lordosis, he caused albuminuria even though the subject lay down. It is not clear whether the lordosis of which Jehle speaks is physiological or pathological. His observations have been repeated by a number of other observers, and there is apparently little doubt that it is possible to produce albuminuria in the way he describes in some persons. The hypothesis is attractively simple, and would explain some features of the condition. Thus orthotic albuminuria does not occur in quite young children, in whom the spinal curves are not properly developed. Both lordosis and albuminuria are disorders of the period of most rapid growth. Albuminuria occurs for the most part in weakly children, in whom slight degrees of lordosis are common. Lastly, albuminuria is most readily produced by standing, in which passive lordosis is more likely to occur than when the patient is moving actively about. It seems probable that lordosis in some cases plays a part in the production of albuminuria, perhaps in association with vasomotor instability. It cannot be the sole cause of the condition, because in many cases placing the patient in a position of lordosis does not bring on albuminuria.

The **DIAGNOSIS** of postural albuminuria requires care, because in nephritis, especially during the stage of recovery, albumin may be absent from the morning urine, though present during the day. We have accordingly (1) to establish the orthostatic nature of the condition by a sufficient number of examinations of the urine, and (2) to exclude nephritis. It is therefore imperative to search carefully for casts. If none are present in several specimens of centrifugalized albuminous urine, the case may be looked on as one of postural albuminuria. If even few casts are found, there must always be a fear lest we have to do with a nephritis. No doubt an occasional cast may occur in the urine without much significance, but their frequent presence is suspicious. Strictly speaking, albuminuria is merely a symptom, and, while pure postural cases nearly always recover, some, apparently of this nature, pass on to a nephritis. In coming to a diagnosis, the result of a search for casts has more weight than examination of the heart and vessels, because in many cases of nephritis in children the cardio-vascular changes are slight, while in postural albuminuria there may be thumping action, irregularity, and even a suspicion of dilatation.

PROGNOSIS.—The prognosis in pure postural albuminuria is good. The abnormality nearly always disappears when growth is complete. If casts are present the prognosis must be guarded.

TREATMENT.—Postural albuminuria is not influenced by treatment. Prolonged rest in bed only prevents the occurrence of albuminuria for the time being.

does not arrest it permanently. Children who suffer from the disorder should not be unreasonably restricted as to diet or exercise. Excessive quantities of nitrogenous food should not be taken; chills, wetting the feet, and unnecessary standing, should as far as possible be avoided. It is undesirable to coddle these children on account of the albuminuria; but if the general health is poor, or if they are neurotic, it will probably be desirable to take them from school for the time being. No drug has any effect on the albuminuria, but the writer has seen benefit as regards the circulatory symptoms in these cases from calcium lactate in doses of 5 grains thrice daily.

While postural albuminuria is the best known and most clearly defined type of functional albuminuria, it must not be supposed that every patient who from time to time shows traces of albumin in the urine is suffering from this disorder. On the contrary, occasional albuminuria arising from other little-known causes is common. Transient albuminuria of this kind, unaffected by posture and occurring intermittently, is probably scarcely abnormal, particularly if the protein is that which is precipitated by acetic acid in the cold. The frequency with which it is found largely depends on the diligence with which it is sought. In a careful examination of the urines of 129 normal children, McHamill and Blackfan obtained traces in 88.7 per cent. of the specimens, and in 32.5 per cent. of the children casts were occasionally present. It may be concluded that the presence of the acetic acid protein is scarcely outside physiological limits, and that occasional hyaline casts and traces of serum albumin are not indicative of damaged kidneys.

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Hæmaturia.—In the newly-born child hæmaturia may occur as part of the hæmorrhagic disease of infants; scæbæ, or hæmatæmæsis, or hæmorrhage from the umbilicus, is usually also present. When an infant under one year old has blood in the urine, it is probably suffering from scæbæ, and other characteristic signs of the disease can usually be found, although in some cases hæmaturia is the only manifestation. It is a very constant symptom, and if, in a difficult case, the microscope shows even a few red blood-corpuscles in the urine, it is practically conclusive. Hæmaturia also occurs in childhood or infancy in some, but not all, cases of renal tumour.

Traces of blood sometimes accompany a deposit of uric acid crystals in the urine. Children also suffer from hæmaturia from the same causes as adults—tubercle of the urinary tract, calculus, or some forms of cystitis, for example. In acute nephritis, hæmaturia is sometimes the first warning that anything is amiss. Hæmaturia may also be caused by the absorption of such poisons as carbolic acid and cantharides.

The hæmorrhagic diseases constitute another group of rarer; Herxol's purpura

is the most important of these. Hematuria may be the first sign of hæmophilia. Among the rarer cause of hæmaturia is thrombosis of the renal veins in cachectic children. I have, however, known this occur without there being the slightest trace of blood in the urine. Bleeding from the kidney may also be caused by a violent paroxysm of hooping-cough (Still). Very profuse hæmaturia, lasting only for a day or two, is sometimes met with in apparently healthy children. I have seen two cases of this kind, and in one there had been several previous attacks. Hæmaturia may result from an excessive excretion of calcium oxalate crystals, following a surfeit of certain fruits, especially rhubarb or strawberries. Guthrie has described an hereditary familial form of idiopathic hæmaturia. It is said that in some such cases there is varicosity of the capillary plexus of the kidney. Another form of hæmaturia which is relatively common in children who have lived in South Africa or Egypt is bilharziosis. In the only case I have seen the urine was smoky, and a few drops of bright red blood were passed at the end of each act of micturition. The ova are easily found in the urine.

As a rule hæmaturia is not a symptom calling for special remedies, apart from the treatment of its cause. The patient should be kept in bed, on a milk diet, and, if the hæmorrhage is severe, any of the usual astringent drugs (turpentine, gallic acid, hæmefine) may be tried. Two other remedies are sometimes of value—subcutaneous injections of gelatine and subcutaneous or rectal injections of horse serum. From 10 to 20 c.c. of 3 per cent. solution of specially sterilized gelatine, guaranteed free from species of the *tetanus bacillus*, should be used. The dose of horse serum is the same.

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Hæmoglobinuria.—Hæmoglobinuria occurs in children either as the result of a toxæmia or in the form of paroxysmal hæmoglobinuria. An epidemic form of hæmoglobinuria, associated with icterus and cyanosis, in newly-born infants has been described (Winkler's disease; vide Chapter I, p. 91). It is a form of septicæmia and excessively rare. Hæmoglobinuria occasionally also occurs in acute infectious diseases, particularly scarlet fever, and in poisoning with chlorate of potash, quinine, salicin, carbolic acid, etc.

Paroxysmal hæmoglobinuria is relatively not uncommon in childhood. In the great majority of cases there is evidence of hereditary syphilis, and instances of two members of a syphilitic family being affected have been reported. It is therefore advisable to subject all patients suffering from paroxysmal hæmoglobinuria to a course of antisyphilitic treatment.

Pyuria.—Pus occurs in the urine as the result of a bacterial invasion of the urinary tract (vide p. 639). In infections with *Bacillus coli*, which are the most common, and in tuberculous infections, the urine is generally acid; in other infections, generally alkaline. The urine may be contaminated by pus in cases of vulvo-vaginitis. Urethritis is rare in little boys. Pus may also enter the urine from an abscess situated in some neighbouring part—perinephric abscess, appendicitis, etc.

Quantitative Variations in the Secretion or Excretion of Urine.—*Oliguria and Anuria.*—Temporary suppression of urine is common in healthy infants and

young children. Many newly-born infants pass little or no urine for the first twenty-four or even forty-eight hours after birth, but this does not signify that anything is amiss, and need cause no anxiety. Congenital malformations causing obstruction are rare. Children two or three years old may pass no urine for many hours without being inconvenienced in any way. In these cases catheterization might not be thought of, because the bladder is not overdistended.

Pathological diminution of the quantity of urine occurs under much the same circumstances as in the adult—in cardiac disease, especially with dropsy; in some forms of nephritis; in conditions of inanition—e.g., following pyloric stenosis or persistent vomiting from any cause. In acute nephritis the urine may be completely suppressed. In the later stages of tuberculous meningitis, also, complete anuria is not uncommonly met with.

Polyuria occurs in diabetes mellitus, diabetes insipidus, and granular contracted kidney, all of which, however, are rare in childhood. I have once or twice seen cases of polyuria which seemed to depend on the child having acquired the habit of drinking excessive quantities of water. A girl aged two and a half years was brought to hospital because for the past six months she had been "thirsty night and day," and was constantly passing water. When she was first admitted she was constantly demanding "drinks," and consumed from 5 to 6 pints of water in the twenty-four hours, and passed a correspondingly large quantity of urine, normal except for its low specific gravity (1.002). There was no evidence of any cardio-vascular changes, and after she was allowed out of bed and began to play with the other children the polydipsia ceased, and she left the ward in three weeks, neither drinking nor passing more water than normal. Polyuria may occur in hydronephrosis, when for any reason the dilated pelvis of the kidney suddenly empties itself. The disappearance of an effusion of fluid or of oedema is accompanied by an increase in the output of urine.

Pollakiuria, or frequent micturition, is not uncommon. It must be distinguished from incontinence. A child may be driven to pass urine repeatedly at short intervals, yet never lose control of the sphincter; a child with incontinence, however, does not necessarily pass urine more frequently than normal. Pollakiuria may be caused by nervousness or by hyperacidity of the urine.

Dysuria.—Parents very often suppose that screaming of infants is due to pain on micturition, when in reality it owes some other cause. Dysuria may be caused by cystitis, by bad phimosis, by vulvitis, by stone in the bladder. In infants whose urine is very acid, or contains a large quantity of oxalates or uric acid, micturition often appears painful. The hyperacidity may be corrected by giving potassium citrate. In little boys painful micturition is sometimes caused by a condition of the urinary meatus which is apparently of the same nature as urethral caruncle in the female. The lips of the urethra are swollen and slightly everted, and on close examination a small bead of granulations, which is very tender, is seen. The condition is probably associated with hyperacid urine. The best treatment is to snip off the granulation, and to close the wound with a stitch. I have never seen urethral caruncle in a little girl. After circumcision it is not uncommon to see a small painful crack or vesicle develop near the meatus; an antiseptic ointment should be applied. Vulvo-vaginitis is a common cause of painful micturition in little girls.

Retention of Urine.—Quite commonly one finds, on examining the abdomen of a child, that the bladder is distended nearly to the umbilicus without apparently

causing the slightest inconvenience. This is equally common in boys and girls, and it seems to be habitual in some children, for it has been noticed repeatedly in the same child. It does not call for treatment. Retention of urine to a degree which requires the catheter is very rarely met with, because of the infrequency of organic obstruction in childhood. Sometimes, however, a calculus becomes impacted in the urethra. Catheterization is seldom or never required on account of phimosis, but when a long, tight foreskin becomes oedematous, as may happen in general dropsy or from local septic infection, it may become necessary. Although a tight prepuce only retards the outflow of urine, the partial obstruction may ultimately lead to dilatation of the ureters and hydronephrosis. Retention of urine may be caused reflexly by painful conditions about the rectum and anus, by organic spinal mischief, and by hysteria.

ENURESIS.

Enuresis, or incontinence of urine, is a common disorder in childhood. In the great majority of cases it is a neurosis, although, as in other neuroses, some local organic affection may play a part in its production. The neurotic element is, however, the most important one, and it is this fact which explains the capricious way in which patients react to treatment; for it is notorious that in enuresis, more than in almost any other common disorder, to prophesy a cure from any drug is quite impossible (*cf.* Chapter XIII., 718).

In the young infant enuresis is physiological. The age at which control of the bladder is acquired depends in the first place on the way in which the child is trained. Under a careful nurse a child will seldom wet himself during the day after he is a year or eighteen months old, and by the end of the second year, if he is regularly lifted once or twice during the night, bed-wetting will be rare. If proper attention has not been given in this respect, habitual control of the bladder will be delayed; and one can scarcely speak of enuresis as a disease in children below the age of three years.

Enuresis in children over three years occurs as a persistence or as a recurrence of the infantile condition, the latter being the more common. We hear as a rule that control of the bladder had been acquired at about the usual time, and then, often after some illness, was again lost. The incontinence may be both nocturnal and diurnal; it may be diurnal alone, though this is rare. Most commonly the complaint is solely of nocturnal enuresis. There is, however, no essential difference between nocturnal and diurnal enuresis; in many patients whose chief complaint is bed-wetting it will be found that from time to time diurnal enuresis occurs. Still lays stress on the frequent association of pollakiuria (frequent micturition) by day with enuresis nocturna, and my own notes of cases entirely confirm this. In a considerable proportion of cases it is noted that during the day the patient is always needing to make water, and that if his wants are not at once attended to he wets himself. At the same time, incontinence may exist apart from pollakiuria, and pollakiuria apart from incontinence. Diurnal enuresis alone is rare; it occurred in only two out of forty-three consecutive cases, and in one of these was associated with incontinence of faeces. Enuresis is equally common in boys and in girls; it often begins during the early years of the second dentition, an age at which, as Still points out, the liability to such functional disorders as tics and stammering is also at its height.

Ætiology.—While, as has been said, most cases of enuresis are of functional origin, a few are due to organic causes. Thus, malformations of the urinary and genital organs, such as extroversion of the bladder, abnormal opening of the urethra into the vagina, or persistent urachus opening at the umbilicus, are accompanied by incontinence. Incontinence occurs in alacy, in some forms of myelitis, in injury to the cord. In other cases enuresis may be due to polyuria, as in diabetes, or to stone in the bladder. Nocturnal epilepsy is another rare cause.

Putting aside all these, in which the incontinence is a symptom, we are left with the large residuum of cases in which enuresis exists apart from any obvious cause. In infancy micturition is a purely reflex act, which is controlled by centres in the lumbar cord. When certain afferent impulses, especially from the bladder, reach these centres, the sphincter of the bladder relaxes, the detrusor of the bladder contracts, and the urine is expelled. As the infant develops, the control of this mechanism gradually passes more and more under the influence of the will. To explain the occurrence of enuresis, one need go no farther than assume that in some children the infantile reflex tends to persist. There is no evidence that the sphincter of the bladder is inherently defective, though in some cases the bladder seems to be hyper-sensitive. On this hypothesis the more frequent occurrence of enuresis during sleep is readily explained. A partial analogy is furnished by the plantar reflex, which normally retains its infantile character to a later age during sleep than during waking hours. It is also clear that various reflex stimuli, which might not produce incontinence in a normal child, may do so when the infantile state of matters persists. The causes of enuresis, looked at in this way, are threefold. In the first place, in not a few cases the early training is largely to blame. The reason for saying this is that many of the patients among the better classes are children of foolish, inconsequent mothers. It is not suggested that all, or even a majority of cases, are due to this cause, nor that cases of what may be called "bad habit" are specially easily curable on that account; but in some children the constitutional neurotic element is a relatively less important factor than the lack of training. Secondly, we have the cases in which a constitutionally unstable nervous system is apparent. The patients are nervous, excitable, and, if old enough, are seriously worried about their weakness. Other neurotic manifestations are not rare. I have notes of habit spasm, stammering and other speech defects, chorea-like movements, persistent biting of the nails, migraine, "faint turns," and spasmodic asthma, occurring along with enuresis. Fright is often said to have been the immediate cause of enuresis, and there is no doubt that punishment tends to aggravate the condition. Curiously enough, sleep disturbances and night terrors seem to be rather rare in these children. Thirdly come the patients in whom, as well as the neurotic tendency, there is a physical delicacy. Many children who suffer from enuresis are poorly nourished, anæmic, or otherwise ailing; not uncommonly some debilitating illness serves to bring on the incontinence of urine.

In many cases the most careful search fails to discover any local condition which may account for the incontinence, but sometimes one is found, and in all cases a thorough local examination should be made. In boys there may be a tight (or, more commonly, simply a redundant) prepuce, balanitis, a narrow urethral orifice, or preputial adhesions; in little girls, vulvitis, adhesions about the labia, etc. Threadworms are not uncommon, or a small fissure of the anus may be found. The urine should be carefully examined, and, though in most cases the result is

negative, we may find oxalates, hyperacidity, uric acid, or that the quantity is above normal. Sometimes there is a trace of pus, indicative of cystitis, or, as Thursfield especially has pointed out, bacilluria. Again, there may be stone in the bladder or glycosuria. Adenoids and enlarged tonsils are common, as they are among all children at this period of life. It is, however, a fact that in the majority of cases no local abnormality can be found; and, moreover, even when some remedial condition is present, it is unwise to conclude that it is causing the enuresis, which often enough continues after the supposed "cause" is removed. Masturbation is sometimes given as a cause of enuresis. The association is rare, and probably accidental.

SYMPTOMATOLOGY.—Enuresis generally begins after the third year; rarely for the first time after the tenth, although relapses may occur even later. It may be constant or temporary, and often varies with the child's state of health. Some children suffer from it only in the winter, and recover completely during the warm weather. In nocturnal enuresis the bladder generally empties itself within the first hour or two of sleep, or, again, in the morning shortly before the child awakes. Sometimes the bed is wetted more than once in the night. The enuresis may be of regular nightly occurrence, or may only be occasional. It does not seem as though full distension of the bladder necessarily precedes incontinence, because one often enough hears that the child is regularly lifted, and yet within half an hour the bed is wet. In enuresis the bladder generally empties itself completely in one act, dribbling is rare, and should suggest the possibility of an organic cause. In cases of diurnal enuresis there is not infrequently pollakiuria; we are told that the child is constantly wetting himself. Sometimes the bladder seems to become temporarily hyper-sensitive. Thus, a little girl, aged six and a half years, had for a month been constantly wetting her clothes during the day, and required to be lifted four or five times between the time she was put to bed and went to sleep. There was neither local irritation, pain, nor abnormality of the urine. She was cured by atropine in about a week.

The course of the disease is chronic and variable. In some cases it disappears as the health improves, only to recur if the patient again falls below par. As a rule enuresis ceases by the sixth or seventh year; in some cases the malady continues until puberty, when recovery nearly always takes place. In a few instances, usually in girls, nocturnal incontinence continues into early adult life.

The **PROGNOSIS**, therefore, is good in the long-run, but the prospect of being able to give early relief is rather uncertain.

The **TREATMENT** of a case of enuresis is generally rather a tax upon the patience of the doctor and of the patient's relatives, and the latter, in particular, are often disposed to let things take their course, in the knowledge that sooner or later the child will "grow out of" his trouble. The first thing, therefore, is to make it plain that treatment must be sedulously carried out for some little time. It is well at the outset to remove any possible source of irritation—to cure oxyuriasis, to have a long or tight perineum removed, to break down preputial or labial adhesions, to treat vulvitis or vaginitis, or any fissure or excoriation about the anus. If the urine is hyperacid, concentrated, or contains a deposit of uric acid, potassium citrate should be given. Bacilluria or cystitis should be treated with potassium citrate or urinary antiseptics (vide p. 648). Thursfield strongly recommends urotropin in cases of enuresis with bacterisuria. It is doubtful whether it is worth

while removing tonsils and adenoids in the hope of curing enuresis; it rarely has any good result in this direction. In the second place, rules should be laid down as to the regular lifting of the child, as to restricting the fluid drunk during the latter half of the day, and as to having the bladder emptied just before going to bed. As a rule, however, these steps have already been taken before the doctor is called in. In the third place the general health requires to be considered. I think that in many cases of enuresis restriction of the starches and sugars of the diet is certainly beneficial, because these children often suffer from chronic indigestion. Like other nervous, delicate children, they feel the cold; chills ought therefore to be ward off by warm underclothing and thick boots and stockings. If other nervous manifestations, such as tic, are present, a course of Swedish gymnastics may do good. Cold douches to the spine (avoiding the risk of chills) help to tone up the muscular and nervous systems.

Of the remedies which do good in enuresis, *belladonna* stands first. I have generally used it in the way suggested by Holt. A solution of $\frac{1}{4}$ grain of sulphate of atropine in 1 ounce of water is ordered, of which 1 minim contains approximately $\frac{1}{1600}$ grain. The dose is 1 drop for each year of the child's age, and this dose is given at first twice, then three, daily. This quantity of atropine ought to affect the sight and cause some dryness of the throat, but this is all. Only once have I seen toxic symptoms, in a girl aged five and half years, who, after five doses of 6 drops given twice a day, became bright-eyed, flushed, and wildly delirious. The quantity was reduced to one-half, and then gradually increased again without causing further symptoms. Given in this way atropine is fairly successful. In a series of forty-three consecutive hospital out-patients treated thus, seventeen were either very markedly benefited or cured—at least, so long as they were taking the drug. If tincture of *belladonna* is used, the dose should be from 10 to 15 minims three times a day, gradually increasing the amount taken. No benefit need be expected from doses too small to produce some of the physiological effects of the drug. *Belladonna* or atropine should have a thorough trial in all cases, and ought to be persevered with for three or four weeks, even if apparently unsuccessful at first. When once the incontinence is under control, the drug should be continued for several weeks before being gradually withdrawn. If prematurely or suddenly withheld, relapse is likely to occur.

If atropine alone fails, it is advisable, before giving it up altogether, to try the effect of adding $\frac{1}{16}$ grain of strychnine to each dose, or the strychnine (or tincture of *nux vomica* in doses of 3 to 4 minims) may be used alone. Next in efficacy to these is ergot (20 to 30 minims of the fluid extract three times a day), which may succeed in very intractable cases. Tincture of *lycopodium* and fluid extract of this aromatic are also remedies of repute; they are given in doses of 10 to 20 minims three daily. A number of other drugs, chiefly nerve sedatives, have been recommended, bromides and phenones in particular. They are much less useful. *Cantharides* has been advised when the sphincter is weak. Thyroid extract has also been employed; in one or two cases in which the writer used it no benefit was observed.

In addition to drugs, a variety of local measures have been tried. The passage of a suture, the application of silver nitrate to the neck of the bladder, massage of the sphincter of the bladder through the rectum, epidural saline injections through the sacral foramina, saline injections into the cellular tissue in front of the sacrum, blistering over the spine, all have their advocates. The writer has no personal

experience of any of them except the last, which is sometimes helpful, probably through its psychical effect. A faradic current from the pubic region to the perineum may be used. None of the others has ever received any wide recognition, or established for itself a place in the treatment of enuresis.

In treating a case of enuresis, moral and educational influences should not be neglected, because, like other neuroses, it may yield to, or be influenced by, a mental impression. Corporal punishment is cruel, and ought never to be allowed; but, on the other hand, a judicious system of rewards for success, and withholding them in case of failure, may help. In diurnal enuresis, especially with pollakiuria, the child should be encouraged to hold his water as long as possible. Children who suffer from enuresis ought not to be sent to a boarding-school; and when there is diurnal enuresis, arrangements should be made for allowing the child to leave the class-rooms when he requires to do so.

Incontinence of feces is usually due to bad habit or to hysteria; the treatment is on moral and psychical lines. *Belladonna* is of no use in the condition.

MALFORMATIONS AND MALPOSITIONS OF THE KIDNEYS.

These are neither common nor of great clinical importance. In the foetus the kidney is lobulated, and evidences of this lobulation are normally present in the full-term infant. Sometimes, however, well-marked fetal lobulation persists into childhood, or even adult life. It gives rise to no symptoms. Absence of both kidneys is incompatible with post-natal life; it is usually associated with absence or malformation of the bladder, genital organs, lower bowel, etc. It is not very rare in symphyliad monsters, acardiac foetuses, and such grave deformities.

Malformations of the kidney are more frequently left- than right-sided, in the proportion of 2 to 1. Three types of single kidney can be distinguished (Ballantyne): (1) Entire absence of one kidney; (2) one kidney rudimentary, the other functioning; (3) fusion of the two kidneys into one mass. In all these there may also be displacement of the organ from its normal position towards the sacrum. A single kidney may have two pelves and two ureters, and this generally means that the single kidney is really the two organs fused. In congenital atrophy of one kidney the organ is represented by a small fatty, cystic, or fibrous mass; the ureter may be a mere fibrous cord, unconnected with the bladder. The best-known form of fused kidney is the horseshoe kidney, in which the two organs are united at their lower poles by a band of kidney tissue which crosses the vertebral column. In other cases the union is more extensive, and the two organs form a disc-shaped mass, with a double or single pelvis and two ureters.

Various anomalies of the pelvis, ureter, and bloodvessels, may also occur in a kidney which is otherwise normal. Thus, there may be two pelves and one ureter, supernumerary arteries, or malpositions of the vessels and ureter at the hilum. In true floating kidney there is a mesonephron with abnormally long vessels.

As the kidneys are somewhat lower in position in the infant than in the adult, they are more accessible to palpation, and under favourable circumstances it is possible, if the abdominal walls are thoroughly relaxed, to detect the lower pole of the kidney in a young child by bimanual palpation, with one hand on the abdomen, and the fingers of the other pressed into the loin below the twelfth rib. The organ is felt as a rounded mass, which descends on inspiration. If a consider-

able part of the organ can be felt, it is usually a sign that it is enlarged, but may be due simply to an abnormally low position.

A fused or horseshoe kidney which is so displaced as to be accessible to palpation (as, for instance, when it lies on the sacral promontory) may readily be mistaken for a tumour.

Floating Kidney.—Floating kidney is very rare in childhood. It has been discovered in children under a year old, and in such cases is probably due to a congenital abnormality—a mesonephros. Jules Cornby suggests that the flatulent distension which is so common in infancy may assist in producing it. The right kidney is more commonly affected than the left, and the condition is more frequently met with in girls than in boys. Floating kidney may give rise to attacks of severe pain simulating appendicitis, to colic, or to peritonitic symptoms—abdominal tenderness, vomiting, and constipation (Cornby). In most cases the anomaly is discovered accidentally. Unless the symptoms are urgent, no treatment is required. If the kidney is giving rise to trouble, a suitable belt should be tried or an operation advised.

Cystic Degeneration of the Kidney.—Cystic degeneration of the kidney, in which the organ is converted into a mass of cysts, sometimes occurs as a congenital abnormality. The condition resembles cystic degeneration in the adult, but what relation exists between the two is doubtful. In congenital cystic degeneration both kidneys are usually affected, and in many cases foetal tumours so large as to render birth difficult. Cysts of the liver and pancreas, and other congenital malformations, may coexist.

PATHOLOGY.—The kidneys are converted into a mass of cysts varying in size from a pea to a cherry, and the intervening kidney tissue is sclerosed or atrophied. In a newly-born infant such a cystic kidney may weigh a pound or more. The cysts have fibrous walls, lined with flattened epithelium, and here and there between them recognizable glomeruli and urinary tubules with saccular dilatations can be found. The cyst contents are usually clear, but may be tinged brown or red with blood-pigment, and sometimes contain urea. There is no obstruction of the ureters or lower urinary passages. The current hypotheses as to the origin of cystic kidney fall under three heads: (1) Retention cysts due to foetal nephritis (Virchow); (2) new growths (Ranvier); (3) failure of the developing tubules to unite with the corresponding glomeruli, or an excess of either glomeruli or tubules, and cyst formation in the supernumerary structures.

Clinically, cystic kidney is of little importance. Few patients survive birth. In a few cases cystic kidney (sometimes on one side only) has been reported in children of two or three years of age.

REFERENCE.

HANAU: Ueber Congenitale Cysten-nieren. Gießen, 1880.

Hydronephrosis.—Dilatation of the pelvis of the kidney and of the ureter from chronic overdistension with urine is not uncommon in children. It is caused by some obstruction to the outflow of urine, although in a certain number of cases it is impossible on post-mortem examination to ascertain precisely where the

obstruction is situated. Hydronephrosis may be congenital or acquired, and unilateral or bilateral. Double hydronephrosis is, of course, much more serious than single hydronephrosis, and most cases are fatal in infancy. In this form death usually occurs before the kidneys have become sufficiently distended to give rise to palpable tumours, hence it is seldom suspected during life; in single hydronephrosis a large tumour may develop, and may be treated successfully. From a clinical standpoint, therefore, we have to distinguish between *double congenital hydronephrosis*, as we meet with in it post-mortem examinations on infants, and *single hydronephrosis*, giving rise to an abdominal tumour in older children. Pathologically, of course, the two sets of cases have much in common. Hydronephrosis may develop during intra-uterine life to such a degree as to render delivery difficult. Congenital hydronephrosis is frequently associated with other congenital malformations.

PATHOLOGY.—The appearance of a hydronephrotic kidney is too well known to require a lengthy description. The organ is enlarged, often to a marked degree. In slight cases there may be only some dilatation of the pelvis; if the process is more advanced, the calyces are pouched, and in extreme cases the whole or a great part of the kidney is converted into a loculated cyst, the parenchyma being compressed and absorbed. When both kidneys are involved, the process proves fatal before this state of matters is reached. The kidneys are usually enlarged to about double the normal size; on section the greater part of the organs is found to consist of dilated calyces, forming pockets from $\frac{1}{2}$ to 1 inch in diameter. The cortex may be reduced to $\frac{1}{2}$ inch in thickness, or even less. Microscopically the kidney tissue which remains shows signs of nephritis, or cirrhosis and dilatation of the tubules. The ureters are dilated, elongated, and sacculated; their walls are hypertrophied. If the obstruction is in the urethra, the bladder too is dilated and hypertrophied, and even in those cases of hydronephrosis in which no organic obstruction can be found the same condition is often observed.

Hydronephrosis frequently becomes pyonephrosis, the infecting organism commonly being the *B. coli*. Ascending infection is doubtless favoured by the urinary stasis.

ÆTIOLOGY.—Among the causes of acquired hydronephrosis (which is less common in children than the congenital form) are impacted renal calculi, tumours of the bladder or other organs which press on the ureters, old inflammatory adhesions, stricture of the urethra, or any other lesion by which the urinary passages happen to be obstructed. Calculus is probably the most common cause in childhood.

As regards congenital causes of the condition, it must be borne in mind that a hydronephrosis may originate in some congenital defect, and yet not reveal itself until after infancy. If the cause acts slowly and incompletely, several years may be required for the production of a palpable tumour. The chief congenital malformations which have been described are kinks and twists of the ureters, oblique insertion into the pelvis of the kidney, compression of the ureter by an abnormal renal artery, obstruction at the vesical orifice of the ureter; all these are much more common than stenosis of the urethra and valvular flaps of the urethral mucous membrane. In the last-mentioned the valvular flap may present so hindrance to the passage of a catheter, and yet seriously obstruct the outflow of urine. Lastly, a tight phimosis may unquestionably produce hydronephrosis.

It is possible that it does so indirectly; the difficulty of urination causes hypertrophy of the bladder wall in the first instance, the thickened bladder wall obstructing the flow through the vesical orifice of the ureter (Fig. 64).

The hydrosphrosis which is sometimes associated with chronic interstitial nephritis is probably secondary to that disease.

In a considerable proportion of post-mortem examinations on cases of double congenital hydrosphrosis, no satisfactory explanation of the condition can be found. There is hypertrophy and dilatation of the ureters, and also of the bladder, but no organic obstruction lower down. John Thomson has pointed out that hydrosphrosis of this type presents analogies to other congenital affections of hollow muscular organs—hypertrophic stenosis of the pylorus and idiopathic dilatation of the colon. The fact that in this form of hydrosphrosis the muscular parts of the urinary tract are hypertrophied as well as dilated proves that an obstruction to the outflow of urine must have existed, and that the condition is not simply a malformation in the ordinary sense of the term. He therefore suggests that the cause of the obstruction, since there is no organic stenosis, must be functional, and that it is a faulty co-ordination of the muscles which control the outflow of urine, due to developmental defect of the nervous mechanism. Both hydrosphrosis with dilatation of the ureters and idiopathic dilatation of the colon have been found along with hypertrophy of the pylorus, and the association is the more significant on

account of the extreme infrequency of other congenital malformations in cases of pyloric stenosis. Thomson's hypothesis is also supported by a case which recently came under the writer's notice. The patient was a girl, aged thirteen months, who was



FIG. 64.—HYPERTROPHY OF BLADDER AND DILATATION OF URETERS DUE TO A VESICAL STRICTURE (FROM A CHILD AGED NINE MONTHS).

The pelvis of the kidney were also dilated.

admitted to hospital on account of vomiting, constipation, and wasting. She was a healthy infant at birth, and had been hand-fed from the first. When one month old she began to vomit, and this continued more or less until the date of her admission. During the latter part of her illness she had wasted and become extremely constipated. On admission she was much emaciated, weighing only 8 pounds 10 ounces, and her abdomen was flaccid, with coils of bowel showing through the parietes. There was indistinct peristalsis, chiefly of the intestine, but no definite gastric peristalsis was made out. The bowels moved regularly, and vomiting was not urgent. There were no other noteworthy symptoms, but the patient lost weight, and died a week after admission. On post-mortem examination, in addition to the usual appearance of marasmus, the stomach was found to be markedly distended, and its wall to be thicker than that of an ordinary adult organ; there was no pyloric hypertrophy or stenosis. The duodenum and jejunum showed little naked-eye change, but the colon was distinctly hypertrophied. The kidneys

were small in size, their pelves not dilated; the left ureter was widely dilated and hypertrophied, being equal in size to the small intestine; the right ureter was slightly dilated and hypertrophied; the bladder, the vesical openings of the ureters, and the genital organs, were all normal. The association in this case of hypertrophy and dilatation of three of the hollow muscular viscera, without any sign of organic obstruction, is suggestive of a functional nervous origin.

SYMPTOMATOLOGY.—Bilateral congenital hydronephrosis is seldom recognized during life. If ascending infection occurs, the condition of the urine may attract attention, and in a case of this kind I have been able to palpate the enlarged kidneys. Owing to the fact that the disease is necessarily fatal and admits of no treatment, it is of little clinical importance, though of considerable pathological interest.

In cases of unilateral hydronephrosis, whether due to a congenital or an acquired cause, the only characteristic symptom is the development of a tumour in the kidney region. Most of the patients are over three years of age. The tumour is usually smooth and rounded, but may be irregular in outline. It may fluctuate or have the consistence of a cyst. It has the usual characteristics of a renal tumour—bulging back into the flank between the last rib and the iliac crest, and having the colon in front of it. A pathognomonic sign, not, however, present in every case, is diminution or disappearance of the tumour, associated with an increased flow of urine. Hydronephrotic tumours vary much in size; a large one may fill the greater part of the abdominal cavity. The fluid in the cyst does not consist of normal urine. It may be little more than a solution of sodium chloride, without either urea or uric acid, or traces of urinary constituents may be present. There may be epithelial cells, albumin, mucus, pus, blood, sugar, urates, oxalates, etc. Although in some cases hydronephrosis causes definite symptoms, they are not characteristic. Attacks of abdominal pain and vomiting, simulating "bilious attacks," have been described. When the condition is due to impaction of a stone, a history of renal colic may be obtained. Otherwise the symptoms are due to the mechanical effects of the tumour.

The **PROGNOSIS** in hydronephrosis is always grave. Double hydronephrosis is probably always fatal. The gravity of unilateral hydronephrosis depends on the cause, and on the condition of the opposite kidney; and on both of these points it may be very difficult to form an opinion. When hydronephrosis is due to an impacted calculus, it may undergo spontaneous cure if the stone be passed, as happened in a case recorded by Goodhart. In intermittent hydronephrosis from other causes the cyst always refills. For this reason aspiration is only useful as a diagnostic or temporary expedient. If hydronephrosis is untreated, the cyst may rupture into the peritoneum or become infected, or septicæmia may ensue.

The **TREATMENT** of hydronephrosis is surgical—removal of the obstruction if possible; failing that, nephrectomy is preferable to drainage. Stiles advises the transperitoneal route as the simpler and more satisfactory method.

REFERENCE.

TRACONIS: *Brit. Med. Journ.*, 1902, i, 578.

NEPHRITIS.

Infants and young children of all ages are liable to nephritis. Not only do all the principal forms of the disease which we meet with in adult life occur, but there are as well certain types which are peculiar to childhood. Several classifications have been suggested, and that which is adopted here is based solely on grounds of clinical convenience. We consider (1) Nephritis in Infancy; (2) Chronic Interstitial Nephritis; and (3) Nephritis in Older Children—Acute, Chronic, Pyelocystitis, Interstitial, and Uncertain Forms. The justification for dealing separately with the nephritis of infancy is that, though its manifestations are often analogous, and its pathology as yet obscure, what little is known about it points towards its playing an important part in the production of some of the chronic forms of the disease which occur in later childhood, the exact origin of which is at present uncertain.

1. Nephritis in Infancy.—Nephritis is probably more common in infants than we have hitherto supposed. There are several reasons why it may often escape recognition. Clinically the most conspicuous feature of nephritis—oedema—may be absent, and the presence of albuminuria with tube casts is, obviously, likely to pass unnoticed on account of the difficulty of obtaining a specimen of urine. Then, again, oedema is common in infants quite apart from nephritis, and the same applies to albuminuria. Oedema and albuminuria may even occur together, though the kidneys prove healthy on microscopic examination (Carpenter). Pathologically, the kidneys may appear to the naked eye perfectly normal, although marked microscopic changes exist. It is thus not surprising that nephritis in infancy may be unsuspected during life, and undetected after death.

Ætiology.—The principal causes of nephritis in infants are syphilis and gastro-intestinal disorders. A few cases have been recorded in the course of scorbutus (Still). It may also occur as a complication of many of the infectious diseases—broncho-pneumonia, erysipelas, meningitis, etc., and in cases of extensive skin affections. In some instances no ascertainable cause can be made out. In a case reported by Ashby, of a child who died at the age of four weeks, the nephritis appears to have developed at or before birth.

Latent Nephritis.—In most cases acute nephritis in infants is latent—i.e., its presence is revealed only by the detection of albumin, granular or hyaline casts, and sometimes blood, in the urine. Latent acute nephritis is apparently not uncommon in acute gastro-enteritis, and possibly the nervous symptoms which sometimes supervene in fatal cases of this disease are really due to æmia. In other cases, though the gastro-intestinal symptoms subside, the infant's general condition does not correspondingly improve, and on examination of the urine signs of nephritis are found. The nephritis of scorbutus is latent; albumin and casts may persist in the urine for several months after the scorbutus is cured.

Holt describes as primary acute nephritis in infants a form which is characterized by high irregular fever, anæmia, nervous symptoms, usually vomiting and diarrhoea, and sometimes dyspnoea. Dropsy is absent, and albumin may not at first be present, but always ultimately appears in the urine, which also contains casts. The disease is fatal in about two-thirds of the patients.

Syphilitic Nephritis.—In a considerable proportion of infants suffering from

hereditary syphilis, the kidneys, like many other organs, are affected. Casool found evidence of nephritis in six out of a series of thirty-one cases. Syphilitic nephritis is as a rule latent. It manifests itself only by albuminuria and casts. The quantity of the urine is not diminished, and it seldom contains blood. Both uremia and anasarca are rare. The occurrence of nephritis in syphilis does not depend on the severity of the other symptoms; it is as often met with in slight as in bad cases. It is not in itself fatal, nor does its presence increase the gravity of the case. Recovery takes place, so far at least as can be judged from analysis of the urine, if the other syphilitic manifestations improve.

Acute Nephritis with Œdema.—In some cases of acute nephritis in infants the clinical picture is more characteristic. There is general oedema, with scanty urine containing albumin, blood, and casts. The writer has seen fatal acute Bright's disease of this kind develop suddenly in a baby aged four weeks.

PATHOLOGY.—The pathology of nephritis in infancy still requires elucidation. The best-known form is that due to syphilis; our knowledge of the others is fragmentary.

In syphilitic nephritis, the kidneys to the naked eye as a rule show no abnormality; if, however, the cellular infiltration is extensive, the cut surface may have a yellowish-pink colour. Microscopically the most constant and characteristic change is an interstitial nephritis. Deposits of round cells are seen in the connective tissue of the kidney cortex, and with this there is associated proliferation of the adventitia of the arterioles. An actual formation of fibrous tissue does not take place. In some cases the cellular infiltration is more widely distributed throughout the cortex, separating the tubules and obscuring the structure of the organ. In addition to these lesions there is in some instances evidence that the normal development of the kidney has been hindered by the syphilitic poison. The glomeruli are unusually few in number, and some of them are rudimentary; the tubules of the kidney also show signs of imperfect development. Alterations of the parenchyma of the kidney occupy quite a secondary place. Here and there the epithelial cells of the convoluted tubules are degenerate, and there may even be changes in the glomeruli—thickening of the capsule, exudation into it, and degeneration of the epithelium covering the capillaries. Extensive catarrhal nephritis, in which the parenchymatous overshadowed the interstitial changes, was found by Carpenter in one case; but this is exceptional, and it is quite possible that the association between it and syphilis was fortuitous. The changes which are characteristic of syphilis may be summed up as—Acute or sub-acute interstitial nephritis; retarded development of the kidney; slight secondary affection of the parenchyma.

In nephritis due to gastro-intestinal disorders, the kidney appears normal to the naked eye. The principal microscopic change is a fatty degeneration of the epithelium of the convoluted tubules; the glomeruli escape. In the acute nephritis described by Holt, the main changes appear to be an acute interstitial nephritis, with secondary affection of the parenchyma. Very few post-mortems have been made on cases of acute nephritis with oedema in infants. In those which have been reported the lesion was a glomerular nephritis.

When the pathological findings are correlated with the clinical aspects of the disease, the following statements are justified: (1) The nephritis of young infants is usually interstitial (commonly syphilitic), acute, and recoverable. Parenchymatous changes are secondary, and are limited to the convoluted tubules. (2) In

certain toxic conditions, notably gastro-enteritis, the epithelium of the tubules is chiefly affected; in this form also recovery often occurs. (3) In both the preceding forms of nephritis the disease shows itself only by the presence of albumin and casts. (4) Frank acute nephritis is rare, and often fatal; the lesion is a glomerular nephritis. (5) Infantile interstitial nephritis may become chronic, and is possibly the precursor of granular contracted kidney.

TREATMENT.—Latent nephritis does not require any special treatment. In the nephritis of syphilis the possibility of mercury having a detrimental action should be borne in mind, and the drug should not be given in too large doses, lest uræmic convulsions take place (Guthrie). The treatment of acute nephritis with salinum is the same as in older children.

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II. Chronic Interstitial Nephritis.—Chronic interstitial nephritis is one of the rarer diseases of children, but typical cases are met with from time to time. In 1903 Sawyer was able to collect twenty-four examples in patients under twenty years of age, and since then this number has been considerably augmented.

ETIOLOGY.—Girls are twice as liable to chronic interstitial nephritis as boys—a marked contrast to the sex distribution of the disease in adult life. Hereditary influence is sometimes shown by the occurrence of the disease in more than one member of a family, or in collaterals. The ordinary causes of granular kidney—poor diet, alcohol, and lead-poisoning—are not operative in childhood, except in so far as a gouty ancestry may be held responsible. It is probable that congenital syphilis is the cause of some cases of granular kidney, and possibly others begin either as an infantile or an intra-uterine nephritis. Well-marked granular contracted kidney has been observed in a child aged two and a half years, but as a rule cases come under observation for the first time in somewhat older children.

SYMPTOMATOLOGY.—The onset of the disease is insidious, and in many cases seems to date from infancy, as the patients are often described as having been delicate since birth. They are usually wasted, stunted in growth, and often somewhat precocious for their years. There is often anæmia, which, however, is masked by a dusky flush, due to capillary congestion. A history of gastro-intestinal disturbance—vomiting, diarrhoea, or constipation—is common. Although there is generally polyuria, it is seldom complained of unless it is associated with enuresis. Guthrie lays great stress on the frequency of pigmentation of the skin in this disease. The discoloration resembles that of Addison's disease; it varies in intensity from mere sallowness to marked bronzing, and is most conspicuous over the abdomen and flanks. There is not, however, any pigmentation of the mucous membranes. In addition to being darker, the skin is dry, harsh, and inelastic. In most respects the manifestations of granular kidney in children

resemble those which are met with in adult life. The heart is hypertrophied, the blood-pressure high, and the arteries thick; the urine is abundant—up to 100 ounces per diem—of low specific gravity, with little or no albumin, and some hyaline or granular casts. There is little tendency to oedema so long as the cardiac muscle retains its vigour, but uræmic symptoms, of which headache is the most common, are frequently met with. Convulsions often occur towards the end of the illness. Albuminuric retinitis and retinal hemorrhages are rare (vide p. 626). Cerebral hemorrhage is not infrequent. Patients suffering from chronic interstitial nephritis are liable to serious bronchial catarrh and pulmonary oedema.

Infantilism associated with chronic interstitial nephritis has been described by Morley Fletcher and Parsons. The condition is apparently merely an exaggeration of the retarded development which is common in the disease (Fig. 65).

PATHOLOGY.—The kidneys are usually much atrophied; their combined weight may not exceed an ounce. They are distorted and puckered, and, when the thickened adherent capsule is torn off, show a granular surface. On section the cortex is found to be much shrunken, the medulla less so. The pelves and ureters are often somewhat dilated. The changes in the kidney, then, resemble those seen in the adult, but are somewhat less marked. Microscopically there is found throughout the kidney an increase of the connective tissue, which by its fibroid contraction distorts the organ and produces secondary changes in the secreting structures. The arterial coats are also thickened, and the lumen of the smaller vessels may be almost obliterated. The cirrhotic process tends to spread into the perinephric tissues, and it is possible that the pigmentation of the skin is due to the implication of the suprarenal glands in this process. Cardio-vascular lesions are even more constant than in the adult. Hypertrophy of the left and to a less extent of the right ventricle is almost invariable. Atheroma and thickening of various arteries is common. Cirrhosis of the liver is sometimes also present.

From the history of these patients and the fact that a slowly progressive fibrosis may attain such a height in a comparatively young child, there can be little doubt that in many cases chronic interstitial nephritis dates from early infancy. We have already seen that the type of infantile nephritis is an acute interstitial inflammation, and it is probable that in some cases this acute interstitial nephritis, instead of resolving, passes into the chronic stage. Gathie believes that in many cases congenital syphilis is the cause of chronic interstitial nephritis, and the evidence in favour of this is very convincing. But just as the interstitial nephritis of the syphilitic infant may become chronic, so may interstitial nephritis



FIG. 65.—ISAAC MANN, CASE OF CHRONIC NEPHRITIS.

Aged nine years. Height, 3 feet 2 inches (12 inches below average). Cephalic development that of three to four years. Mentally slightly backward. Polyuria, albuminuria, granular and hyaline casts. No evidence of congenital syphilis.

due to other causes. This would account for the occurrence of chronic interstitial nephritis apart from all evidence of hereditary syphilis.

DIAGNOSIS.—The diagnosis of this disease is seldom difficult, except in the early stages. Guthrie draws attention to the possibility of confusing with it cases of the uric acid diathesis, in which there may be albuminuria, headache, and high blood-pressure. The absence of thickening of the vessels and of polyuria are the chief distinguishing marks. Uremia may be mistaken for tuberculous meningitis; cases in which pigmentation is marked may resemble Addison's disease; the polyuria may suggest diabetes.

TREATMENT.—Owing to the more progressive character of the disease, less can be expected from treatment in chronic interstitial nephritis in the child than in the adult. It should, however, be conducted on the same lines. The diet ought to be such as not to tax the kidneys; meat therefore should be eaten sparingly. The patient should be warmly clad, chills should be avoided, and the skin kept active by warm baths. The quantity of water drunk need not be restricted in any way. Constipation is a common symptom, and should be relieved by salines. If there is anemia, iron is required. As the cardio-vascular changes are compensatory, drugs which lower the arterial pressure should not be given without a definite reason, and their effects should be carefully watched. If there are signs that the cardiac muscle is beginning to yield, such as diminution in the amount of urine, slight edema, or falling blood-pressure, digitalis and squills are indicated. The treatment of anemia is referred to on p. 636; in this form of Bright's disease, however, uremia is much less amenable to treatment than in acute nephritis.

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III. Nephritis in Older Children.—*Acute Nephritis.*—Acute nephritis is by no means uncommon in childhood; it may occur at any age. Acute nephritis with anasarca is, however, rare before the second year, although it may occur in infancy (p. 623). Both sexes are equally liable.

ETIOLOGY.—Scarlet fever is probably the most common cause of nephritis, but the kidneys may become affected in any infective disease. Putting aside scarlatinal cases, which rarely come under observation in general hospitals, there is no doubt that in the majority of patients acute nephritis begins without ascertainable cause. Among thirty consecutive cases of nephritis treated in hospital, twenty appeared to be primary. It is, however, probable that most of these so-called "primary" cases are the result of bacterial infection; and when we remember how easily slight attacks of tonsillitis are overlooked, there is no difficulty in supposing that infection may arise in this way. I have seen acute nephritis develop a week after the onset of tonsillitis and otitis. Measles rarely causes nephritis, but in one patient the measles rash appeared the day after the edema was first noticed. Pneumonia does not seem to be a very rare cause of nephritis; three of my cases were associated with pneumonia, and I have seen several others. Nephritis may

also complicate chicken-pox, measles, hooping-cough, influenza, and probably rheumatism; it very seldom follows diphtheria, though albuminuria is common in that disease. In the tropics malaria is a recognized cause of acute nephritis in children. Apart from the infective diseases, the only other recognized cause of nephritis is cold. It is, of course, impossible to prove absolutely that the disease is due to exposure, but there is a certain amount of evidence that this may sometimes be the case. Claude Ker quotes as an instance a newly-built fever hospital in which sixteen of the first twenty scarlet fever patients treated developed nephritis or albuminuria shortly after they were allowed to sit up in bed. It was found that the ventilating outlets placed above each bed were acting as inlets, so that a cold air douche fell on the backs of the patients. When the ventilators were closed, the outbreak of nephritis ceased.

SYMPTOMATOLOGY.—The symptoms of acute nephritis are usually characteristic. The onset is in most cases abrupt, and often the first sign that anything is amiss is the sudden swelling of the face. In other cases the appearance of the oedema is preceded by headache and vomiting or, somewhat frequently, by diarrhoea. At the onset there is usually some fever, but the temperature seldom rises above 101° F., and in most cases it is lower than this. Unless there is some complication, the fever disappears within a few days—long before the other symptoms of nephritis subside. While in most cases oedema of the face is an early symptom, this is not invariable; the face may never be affected at all, and the dropsy be limited to the loins and legs. Again, the urine may be scanty and contain blood and casts for a week or more before any oedema is seen; and in other cases of acute nephritis there may be, from first to last, no oedema at all. An extreme degree of dropsy is more frequently seen in chronic parenchymatous than in acute Bright's disease, but in the latter there may be general anasarca, with effusion into the serous cavities, particularly the peritoneum. Pericardial effusion is rare.

The urine is diminished in amount—from three to five ounces in the twenty-four hours in a moderately severe case—and very often contains a considerable quantity of blood. The altered appearance of the urine is not seldom the first indication that anything is wrong with the patient. As a rule the albumin is abundant; occasionally it is present in such quantity that the urine solidifies on boiling. Hyaline, granular, epithelial, and usually blood casts are also present, often in great numbers. The extent of the oedema does not correspond very closely with the amount of blood and albumin in the urine. Sometimes there is little or no puffiness, along with a considerable amount of albumin, and, again, fairly well-marked oedema with a mere trace. In acute nephritis there is probably always some blood, at least towards the beginning of an attack, although, if the child is not seen until he has been ill for two or three days, it may have disappeared, leaving only albuminuria. In some patients, owing to the scanty, concentrated urine, frequent painful micturition is met with, and occasionally there is abdominal pain. Henoch and others have reported cases of acute nephritis without albuminuria; this, however, is very rare.

In acute nephritis the blood-pressure is almost always raised, sometimes to a marked degree. Gordon, who investigated this point in a series of cases in the Edinburgh Sick Children's Hospital, found that at the beginning of an acute attack the systolic pressure might be as high as 160 to 180 millimetres Hg. When the initial pressure is very high, it generally falls to normal in a week or less; when it is

only moderate, it may remain above normal for a longer period. The normal systolic pressure in children, in round numbers, is 70 millimetres Hg under one year, 80 millimetres Hg under two years, 90 millimetres Hg under nine years, and 100 to 105 millimetres Hg from nine to twelve years. The blood-pressure is highest in those cases in which the oedema is slight, and accompanying the higher pressures there is usually considerable hæmaturia. On examining the pulse with the finger, the hypertension is often apparent, but no trustworthy estimate of the degree to which the blood-pressure is raised can be formed by the finger alone. In some cases the pulse remains persistently infrequent during the course of acute nephritis, returning to normal as the patient recovers. The heart sounds are usually feeble; the first is sometimes impure or reduplicated, and the aortic second accentuated.

Cases of acute nephritis vary greatly in severity. On the one hand we meet with mild forms of the disease in which there are no urgent symptoms—merely a little puffiness of the face and albuminuria, the former of which subsides in a day or two, while the albumin disappears in ten days or less. At the other extreme come the very grave cases, in which uræmia is present from the onset, the urine remains scanty or is totally suppressed, and the patient dies comatose in the course of a day or two. A little girl aged six, who had previously been perfectly healthy, suddenly became ill with feverishness and puffiness of the face. Next day her speech was thick and rambling, and she became drowsy; next morning she was unconscious. She was sent into hospital comatose, with partial suppression of urine, and died seventy-six hours later, despite all treatment, without recovering consciousness.

Acute uræmia is relatively uncommon in children, and on the whole there is less tendency to eclampsia than in adults. Uræmia usually sets in with vomiting and drowsiness, and the child becomes semi-comatose, with unequal pupils and twitching of an arm or leg. The breathing has sometimes a sort of hassing character. The symptoms of uræmia, as a whole, are often not unlike those of tuberculous meningitis (*vide p. 636*).

Scarlatinal Nephritis.—As the most important form of acute Bright's disease, scarlatinal nephritis demands special notice. In almost all cases of severe scarlet fever there is albuminuria, and sometimes a few tube casts, during the height of the febrile stage. This must be distinguished from true nephritis, which is a later complication. The nephritis is believed to be due, not to the direct action of the scarlet fever germ, but to toxins. Streptococci may also play a part in producing it in some cases.

Nephritis varies in frequency in different epidemics of scarlet fever. Ker, in a first series of 4,436 cases, found its incidence to be 11·02 per cent.; in a second series of 3,172 cases it was only 5·64 per cent. It is most common during the age-period five to ten years, children of from five to seven years being most of all susceptible. Nephritis usually develops towards the end of the third week or the beginning of the fourth. "In a series of 134 consecutive cases of acute nephritis, 88 occurred between the sixteenth and twenty-sixth days, and of these 68 had their first symptom between the twentieth and twenty-fourth days inclusive" (Ker). In some families there seems to be a special predisposition to scarlatinal nephritis, as is seen when several brothers and sisters develop this complication. The liability to nephritis does not in any way depend on the severity of the original disease; indeed, bad cases of Bright's disease are often met with where the ante-

recent scarlet fever has been so mild as to have escaped notice. No doubt the fact that such patients have not been protected in any way influences the occurrence of the kidney affection, because, whether or not a chill can originate nephritis in a healthy person, there is no doubt it can precipitate it in a scarlet fever patient.

Scarlatinal nephritis may develop suddenly or gradually. If the urine is being examined day by day, a little albumin and a few casts may be detected before there is any naked-eye change. Soon, however, the characteristic reddish-brown colour appears, and there are found albumin in quantity, blood, and casts of all kinds. There is more or less dropsy, and when the disease develops suddenly it may be the earliest sign. The other symptoms of scarlatinal nephritis do not differ in any way from those which have been described above.

Pneumococcal Nephritis.—I have several times met with cases in which both acute nephritis and acute pneumonia were present,* and had, so far as could be made out, developed almost simultaneously. I have also once seen pneumonia develop on the eleventh day of a nephritis, while the acute symptoms were still present. When both diseases begin about the same time, the pneumococcus is presumably responsible for the kidney affection as well as for the pneumonia; but in two cases in which a careful bacteriological examination was made the urine was sterile in one, and in the other contained no more organisms than might have been present in any pneumonia. Carnegie Dickson, Pathologist to the Sick Children's Hospital, regarded the condition as toxic rather than septic. In this respect, therefore, pneumococcus nephritis conforms to what is known about scarlatinal nephritis, which also is toxic.

In these cases the onset is sudden, the pneumonic symptoms being the most prominent, and as a rule oedema of the face is noticed within twenty-four hours. In one patient, however, there was no oedema, and in another the urine was noticed to be dark-coloured four days before the lung symptoms developed. The course of the pneumonia does not seem to be influenced by the co-existing nephritis, for in all the patients who recovered it ended by crisis within a week. Two patients subsequently developed empyema, and one of these was also uræmic. Patients attacked by this pneumococcal nephritis appear pallid, slightly cyanosed, and puffy about the face, and altogether look, and are, extremely ill; but the prognosis is probably more favourable than might at first sight be expected, for only one out of seven cases died. The blood and albumin may disappear from the urine within a week after the crisis; this happened thrice. Of the two patients with empyemata, one went out of hospital with, the other without, a trace of albumin. Of the two others who recovered, one began to desquamate three weeks after admission, and was transferred to the fever hospital. In the fatal case death took place on the sixth day of the pneumonia. The chief clinical interest of this group of cases is the relatively favourable prognosis, in spite of the apparently serious nature of the illness. In dealing with them, the pneumonia should be treated on ordinary lines, and hot-packing, etc., should be deferred until after the crisis.

Chronic Parenchymatous Nephritis.—Chronic parenchymatous nephritis is not common in children, nor, when it does occur, are its symptoms in any way peculiar. For these reasons, only a short description of it is necessary. An acute nephritis may gradually pass into the chronic stage, or there may be an interval

* Some of these cases were under Dr. John Thomson's care; he kindly allows me to refer to them.

of apparent health lasting for months, or even years. In some cases the disease appears to develop insidiously without any antecedent acute attack; the origin of such cases is obscure. Chronic parenchymatous nephritis is not common before the fourth or fifth year, and most frequently the patients are older.

SYMPTOMATOLOGY.—The leading symptom is dropsy, which may be extreme; it varies much from time to time, and the patient may be free from it for months together. The urine remains constantly albuminous, although when the patient is free from oedema there may be only a trace of albumin present. Casts, too, are always to be found. The specific gravity is normal or slightly raised. During an exacerbation the quantity of urine falls, and the daily amount passed may not exceed five or six ounces. A trace of blood is occasionally present. The blood-pressure is little raised in chronic parenchymatous nephritis, and there are no marked cardiovascular changes. In most cases there is some anaemia, but the reduction in the haemoglobin and red corpuscles is less than the pallor would suggest.

The course of an exacerbation of chronic parenchymatous nephritis is often erratic. It frequently happens that, when the patient is first put under treatment, the dropsy rapidly disappears; then, without any apparent reason, and without any change in the diet or treatment, fluid quickly accumulates again. Often, too, after treatment has been quite ineffectual for several weeks, a profuse diuresis sets in, and in a few days the patient is entirely free from oedema. A similar capricious response to dechlorination is sometimes met with; at one time a salt-free diet will rapidly cause the dropsy to vanish; at another time in the same patient it may be a fortnight or three weeks before the slightest effect is produced (Chart I., p. 638).

Chronic parenchymatous nephritis runs a course of several years. Death is usually caused by some intercurrent infection, such as pneumonia, pericarditis, or peritonitis, or by acute uraemia. Oedema of the lungs is the most dangerous complication, directly due to the accumulation of fluid; it often proves fatal.

Lardaceous Nephritis.—Lardaceous disease of the kidney is very rare in children. Prolonged suppuration, the most important cause of lardaceous disease, is now, thanks to the achievements of modern surgery, of uncommon occurrence. It is doubtful whether congenital syphilis ever gives rise to lardaceous disease of the kidney which is recognizable clinically. It is stated that in congenital syphilis an exceptional method of distribution of waxy degeneration may be found, in which "the disease is confined to the interstitial supporting connective tissue between the collecting tubules towards the apices of the Malpighian pyramids" (Beattie and Dickson).

The symptoms of lardaceous disease of the kidney are the same in the child as in the adult, and the diagnosis is not difficult. There is a history of long-continued suppuration, as from tuberculous bone disease or embedded cryptococci; and, along with this, albuminuria and polyuria, and usually oedema, are present. There may also be found an enlarged liver and spleen. In the only case of lardaceous nephritis in a child treated by the writer, the dropsy, which was the most prominent symptom, completely disappeared on a salt-free diet.

Chronic Latent Nephritis.—Heubner has drawn attention to a form of chronic nephritis which is almost peculiar to childhood. Its leading characteristics are the latency of the symptoms, and a tendency to complete recovery after a prolonged course of several years' duration. It apparently develops out of an acute nephritis, especially the scarlatinal variety, and possibly out of the nephritis which is associated with the gastro-intestinal disorders of infancy. In cases of

this kind the only indications of renal disease are the presence of casts and albuminuria. The general symptoms are indefinite, and much resemble those which accompany postural albuminuria. Dropsy, high blood-pressure, hypertrophy of the heart, retinal changes, and uræmia, are conspicuous by their absence. The albumin does not usually exceed 1 per 1,000; it may be absent from the morning urine, as in cases of functional albuminuria. The casts are mostly hyaline. Degenerated epithelial and blood cells may be present in the sediment.

The morbid changes in a case examined by Hentzer consisted in—(1) Scattered foci of renal-cell infiltration, chiefly in the boundary zone; and (2) fatty degeneration of the collecting tubules and arched tubules of the medullary rays. The glomeruli and convoluted tubules of the cortex were normal.

The prognosis of this form of nephritis is relatively favourable. Of sixteen cases followed by Hentzer for a period of over ten years, nine certainly, and two probably, made a complete recovery; in five the albuminuria still continued at the end of that time.

Patients suffering from this form of nephritis ought not to be placed under too many restrictions. Alcohol is absolutely forbidden, and also spices of all sorts and coffee; otherwise ordinary food should be taken. All the less violent forms of sport and exercise are allowable. The clothing ought to be warm, so that chill may be avoided. Regular warm baths, which keep the skin active, are beneficial. According to Hentzer, a "cure" at Carlsbad is sometimes followed by the disappearance from the urine of the abnormal constituents.

The diagnosis of this form of nephritis from functional albuminuria must, obviously, be very difficult in some cases. It will depend on the results of careful microscopical examination of the urinary sediment.

PATHOLOGY.—In *Acute Nephritis* the kidneys are enlarged, and often appear more rounded than normal. The capsule strips readily, and reveals a pale, mottled surface with prominent stellate veins. On section the cortex is enlarged, pale, and mottled, or in the more acute cases, particularly scarlatinal nephritis, shows greyish areas surrounded by deep red zones. Microscopically the glomeruli show hyperæmia; their capillaries are swollen, and there is an exudation of leucocytes, or even rupture and hæmorrhage. There may be slight proliferation of the cells lining Bowman's capsule. In the epithelium of the tubules there are signs of catarrh, cloudy swelling, and fatty degeneration. These changes are most marked in the convoluted tubules and the ascending parts of Henle's loops. Interstitial changes in the shape of leucocyte emigration around the vessels of the kidney substance are slight in comparison with what is found in septic nephritis.

Subacute and Chronic Nephritis.—In the early stages the kidney is enlarged, but in cases which have lasted for several weeks the swelling of the cortex will have had time to disappear, and the size of the organ may be but little above normal. If, however, a considerable amount of interstitial change has taken place, the so-called "large pale kidney" is found. The capsule is not thickened; it strips readily, and leaves a pale or pinkish surface. On section the superficial cortex especially is diminished in size; the inter-pyramidal cortex is often enlarged. The organ as a whole is pale, with patches of yellowish-white mottling due to fatty change. Microscopically, the arteries are found to be thickened; the glomeruli show all grades of change, up to complete fibrous atrophy; the epithelium of the tubules is catarrhal or atrophied, and the tubules themselves contain casts. There is also considerable interstitial change, especially round the glomeruli. If the

interstitial change reaches an advanced stage, one of the forms of granular kidney develops—the so-called “pale granular contracted kidney.”

DIAGNOSIS.—The diagnosis of Bright's disease is easy; it depends, of course, on the examination of the urine. Mistakes are possible in three circumstances only: (1) Nephritis may be mistaken for functional albuminuria, or *vice versa*. It must be remembered that the fact that albuminuria is present only when the patient is erect is not absolute proof of its functional nature, and also that in postural albuminuria the albumin may be comparatively abundant. Mistakes can only be avoided by careful repeated examinations of the urine. (2) Children occasionally suffer from oedema of the face which is quite independent of any local inflammation, and exactly simulates that seen in Bright's disease; it usually disappears in the course of a day or two. The cause of this apparently idiopathic oedema is not known; it may be allied to urticaria. (3) Cases of uræmia may simulate tuberculous meningitis; and if at the time of examination the urine is suppressed (as may be the case in either disease), the difficulty in diagnosis may for the moment be very great (vide p. 635). Lumbar puncture and examination of the cerebro-spinal fluid should prove conclusive; and if the case turn out to be uræmia, the operation will do no harm, but rather good. Uræmia should be thought of in any case of severe repeated convulsions in a child, although it is a somewhat rare cause, in comparison with others, at this age.

PROGNOSIS.—When one is confronted with a case of Bright's disease, several questions naturally arise. If it be an acute case, what will be the probable course of the attack? What the future of the patient—will recovery be permanent? If it be a chronic case, what are the prospects as to recovery, and what the probable duration of the illness? To give satisfactory answers to these questions is no easy matter.

1. *Acute Nephritis.*—We have first to consider the ordinary course of an attack of acute Bright's disease up to the time that the urine becomes free from albumin, and the patient is able to return to his ordinary life.

(a) In *substantial nephritis* recovery nearly always takes place. If the patient is treated early, anaemia either does not develop to any marked degree, or, if present at the onset, disappears within a few days. The initial fever also subsides within a week, and after the first ten days or so the amount of urine returns to normal. Blood and albumin may persist for from two to six weeks—rarely longer. Kerr states that the maximum detention in the Edinburgh City Hospital for this reason does not exceed fourteen weeks, and that during the last twelve years he has not discharged more than three or four patients with albuminous urine. Still quotes some statistics by Goodall which point in the same direction. Of 281 cases of scarlatinal nephritis (adults and children), 45 died. The albumin disappeared from the urine within four weeks in 54 per cent., and in only 19 per cent. did it persist beyond six weeks. So far as scarlatinal nephritis is concerned, therefore, the answer to the first question is that complete recovery may be anticipated within a month or six weeks.

With regard to the future of patients who have passed through an attack of scarlatinal nephritis, the probability is that the great majority remain perfectly well during the rest of childhood. The justification of this statement is that in children's hospitals cases of nephritis which can be traced to scarlet fever are comparatively rare. Yet scarlatinal nephritis is common, and if many of these

cases went on within a year or two to chronic nephritis, more of them would certainly be seen in the general hospitals for children. In a series of 30 cases of nephritis treated in my ward, only two could be ascribed to scarlet fever, one being a chronic, the other an acute case. In Still's series of 100 cases, only 22 were scarlatinal. Keer also believes that chronic nephritis rarely follows scarlet fever.

It is more difficult to say whether this immunity to ill consequences after an attack of scarlatinal nephritis lasts on into adult life. At present there is a tendency to trace the chronic nephritis of young adults to an attack of scarlatina in childhood, even although years of apparent health have intervened. So acute an observer as Holt, who formerly held that chronic nephritis was a rare consequence of acute scarlatinal or other nephritis, says, "Larger experience, however, has convinced me that this sequel is not very uncommon. The interval of apparent health may sometimes cover a period of several years, and the later nephritis may be attributed to other causes; but all cases of scarlatinal nephritis should be carefully watched for a long time, and after a severe attack a guarded prognosis should always be given as to the ultimate result." He reports a case of this apparent cure in a girl who died at nineteen years of age from uræmia. Twelve years earlier she had scarlatinal nephritis; she was supposed to have recovered completely, and enjoyed perfect health for nine years—until she was sixteen—when symptoms of chronic nephritis developed. Heubner, also, lays stress on the importance of scarlet fever in childhood as a cause of chronic Bright's disease in young adults.

[4] In acute nephritis, other than scarlatinal, the immediate result is as a rule recovery, provided that the patient is carefully treated. In a mild or moderately severe case the temperature falls within a few days, then the œdema vanishes, and last the blood and albumin disappear from the urine. In 18 consecutive cases of acute nephritis which left hospital with normal urine, the time which elapsed between admission and the cessation of the albuminuria was as follows:

5 days	1 case	3 weeks	1 case
6	1 "	4 "	2 cases
7	1 "	5 "	2 "
10	3 cases	8 "	5 "
11	4 "				

The numbers are very small, but they serve to show how rapidly recovery may take place. John Playfair found that, in 15 cases of acute nephritis treated in the Edinburgh Children's Hospital, the albumin disappeared from the urine in from three to forty days. Probably, however, the 33 Edinburgh cases happened to include a number of unusually mild attacks, because Still found that 37 out of 51 non-scarlatinal cases lasted more than three months.

In non-scarlatinal nephritis the mortality is not, in the writer's experience, high. Of 28 cases in hospital only 4 died; there were 5 deaths in Playfair's series of 20 cases. In all these death occurred within a week of the onset of the illness. In two of the writer's patients there was uræmia and suppression of urine; in the other two rapid heart failure. One patient was found at the post-mortem examination to be the subject of lymphæmia, which no doubt had contributed to the result. In Still's series of 51 cases the death-rate was 13, or 1 in 4, as against 1 in 7 of the writer's smaller number; his results also differ in that he had no deaths earlier than the seventeenth day, and most at a much later period of the illness.

Since there is great uncertainty as to the ultimate fate of patients with acute or chronic nephritis, the writer tried to follow these children after they had left hospital, and, although the attempt has been only partially successful, the figures may be given for what they are worth:

Of 30 cases of nephritis, 7 were chronic, or left hospital with albuminuria of at least three months' duration; 1 was acute, took scarlet fever, left hospital with albuminuria, and has been lost sight of; 4 died; 18 were acute, and left hospital with normal urine.

Of these last, 3 cannot be traced, but the present condition of 15 is known. None of these 15 has albuminuria; in all the urine has remained free from albumin and the health good for from one to three and a half years after the original illness.

A recent investigation by Ernberg also shows that the prospect of complete recovery from acute nephritis is good. He was able to trace 61 out of 106 patients discharged from hospital as cured or convalescent from acute nephritis during the period 1885 to 1892. These 61 patients he followed up after periods of from sixteen to twenty-three years, with the following results: 8 patients were dead—none, however, of nephritis; 43 were perfectly well; 1 had albuminuria; 9 had symptoms referable to the circulation—functional murmurs or slightly raised blood-pressure—but in none of these was there evidence of nephritis.

In acute non-scarlatinal nephritis, then, the immediate prospect of recovery is good; the mortality is probably not more than about one in five or six on an average. As regards a permanent cure, much would seem to hinge on the severity and duration of the original attack. When the albumin disappears within a month or less, the chances are that the kidneys will completely recover; if the albuminuria persists for three months, there is a very great risk that the kidney will pass into the chronic stage, if, indeed, it has not already by this time done so.

In forming a prognosis in any particular case of acute nephritis, one is guided by the age of the patient as well as by the symptoms. In patients under two years of age the disease is much more serious than in older children. In children over three, if there are no specially bad symptoms, recovery will almost certainly take place. Unfavourable symptoms are—(1) Rapid, feeble action of the heart with more or less cyanosis; (2) drowsiness, restlessness, coma, or convulsions; (3) suppression, or very scanty secretion, of urine; (4) an unusually high temperature, which is often due to some intercurrent infection. The supervention of pneumonia, however, though it adds to the gravity of the case, is not so serious a complication as might be expected. Marked dropsy, unless it persists in spite of treatment, or is associated with an erysipelatous rash, need not cause any great anxiety. The amount of urine passed is of more importance than the quantity of albumin and blood it contains.

2. *Chronic Parenchymatous Nephritis*.—Patients suffering from chronic parenchymatous nephritis may live for a number of years with comparatively few symptoms. I have watched patients in whom the disease has lasted for at least seven years, and, though all have had from time to time acute exacerbations with dropsy and scanty urine, they are not on the whole worse than when first seen at or near the beginning of their malady. In chronic parenchymatous nephritis there is little tendency to the occurrence of cardiac hypertrophy, so far at least as can be judged from a clinical examination of the heart. The blood-pressure is raised a little above the average, but the high pressures which are met with in acute nephritis do not occur in the chronic parenchymatous cases. Notwith-

standing the slow progress which the disease makes, there is no reason to think that it is ever checked, or that recovery comes, hence the lives of these children are in constant danger; a supervening attack of acute nephritis may prove suddenly fatal. A great deal depends on the extent to which the patient's circumstances allow of his being properly cared for, and in particular on the management of his diet. In patients in whom there is an ever-present tendency to dropy the long-continued use of a salt-free, or at least a salt-poor, diet may contribute greatly to the maintenance of health.

UREMIA.

Uremia is on the whole less common in children than in adults. It may occur in acute nephritis, especially in the post-scarlatinal variety of the disease, and is the most common cause of death in chronic interstitial nephritis. Uremia may also develop in the later stages of chronic parenchymatous nephritis, but the liability to it is less in this than in the two other types mentioned.

In acute nephritis uræmic symptoms may precede the development of anasarca and albuminuria, and furnish the earliest indication that the kidneys are affected. More commonly, however, they appear during the first few days of an attack, and are heralded by a diminished flow of urine, or even suppression. After the first week they are rarely met with.

The onset of uremia usually manifests itself by gastro-intestinal symptoms—coated tongue, vomiting, and sometimes diarrhoea. The pulse becomes less frequent, and the blood-pressure is raised. In acute nephritis it may rise to 160 or 180 millimetres Hg, and in a girl aged nine, suffering from chronic interstitial nephritis, in whom the pressure ordinarily was 140 to 150, it rose to 220 with the onset of uræmic symptoms. Headache is common, and the temperature may be raised. The patient becomes drowsy and restless, and twitching of various muscles, or squinting, may occur. The convulsions of uræmia may be general, or they may be localized to one side of the body or to a limb. A single convulsion is rare; recurrence at short intervals is the rule, and in bad cases the fits tend to increase in severity. Between the fits consciousness is more or less obscured, or there may be deep coma. In favourable cases the period of eclamptic seizures does not last more than twelve hours; at the end of that time the secretion of urine is re-established, and the patient gradually regains consciousness, although remaining dull and apathetic for a day or two. After a remission lasting twenty-four hours recurrence of the convulsions is improbable.

Other nervous symptoms may result from uræmia. The most common of these is temporary amaurosis, which may precede, but more often follows, the convulsions. There are no visible changes in the fundus in these cases, and the condition passes off in a few hours or days. In some cases visual troubles—blindness, irregularity of the pupils, squinting—occur apart from eclampsia. Temporary deafness, aphasia, and paralysis, are less common than amaurosis. I have seen facial paralysis develop the day before a uræmic convulsion, and persist for ten days after recovery. Maniacal delirium and psychoses are rare.

In cases of granular contracted kidney, chronic as well as acute symptoms may occur. Headache, with urgent dyspnoea which passes into coma, seems to be not uncommon.

The clinical picture of a case of uræmia is sometimes not unlike that of a case

of tuberculous meningitis, especially when the uræmia is the first indication of nephritis. The history of headache and vomiting, followed by convulsions and loss of consciousness, is suggestive of meningeal mischief; and if, as may happen, there is persistent local tonic spasm of a limb, the resemblance is great. In uræmia the urine may be suppressed, and this may also be the case in tuberculous meningitis. The breathing of uræmic coma has sometimes a characteristic hissing character, unlike that of the coma of meningitis. In doubtful cases lumbar puncture should be performed, and the fluid withdrawn examined microscopically.

RETINAL CHANGES.—Albuminuric retinitis and retinal hemorrhages are rare in childhood. They probably occur only in the chronic interstitial form of nephritis. On account of the insidious nature of the disease, visual defect is sometimes the first indication that the kidneys are affected. Most patients die within a few months of the development of retinitis, but one of Nettleship's patients, in whom the characteristic lesions of the fundus and other signs of granular kidney were present at the age of eight, is reported as being in good health at the age of twenty-five. Detachment of the retina, associated with retinitis, has been recorded in interstitial nephritis in children by Anderson and by Nettleship.

TREATMENT.—Acute Nephritis.—The patient must of course remain in bed, and, as it is important to postpone the action of the skin, he should be kept warm, and the temperature of the room ought not to be allowed to fall below 65° F. The diet should be so chosen as to throw the least possible work on the kidneys, and for that reason it is probably inadvisable to allow even milk in the initial stage of the disease. Instead, a malted food, such as Mellin's Food, made with water should be used. On the whole, cases treated thus improve more quickly than did those dieted, according to the older plan, on milk alone. After the acute stage is over, milk, farinaceous food, and fruit, may be given, and later fish and white meat.

The bowels should be kept freely open, and it is not always easy to attain this end, partly, no doubt, on account of the absence of solid residue left by the restricted diet. The most generally useful purge is Henry's solution of sulphate of magnesium in doses of from 2 drachms to $\frac{1}{2}$ ounce once or twice daily. Compound jalap powder, which is often used, is uncertain in its action. If the Henry's solution fails, a full dose of the compound liquorice powder may be given. Potassium bitartrate and sodium sulphate sometimes act well. The synthetic purgatives—purgin, etc.—are not of much use.

It is of the greatest importance that the skin should act freely, and for this nothing is better than a hot-pack. The child should be kept in this for one to two hours, or even longer, if the skin remains dry. After the pack the patient ought to lie between blankets, with plenty of hot bottles, so that sweating may continue. While the patient is in the pack he should have a drink of hot gruel or the like, with a little brandy. This will help to promote the action of the skin. Hot-packs seldom cause faintness, but on one occasion the pack apparently precipitated an attack of heart failure which proved fatal in about twelve hours. The patient, a girl aged eight, was found to have an enlarged thymus and other signs of lymphatism. Hot-packs are more convenient than, and as a rule quite as efficacious as, hot-air baths; but if the former fail, recourse may be had to the latter. In addition to these measures, a diaphoretic mixture of spiritus ætheris nitrosi ℞ x, liquor ammoniæ acetatis ad 5i, may be given every three or four hours.

When the amount of urine is scanty, a large hot linseed and mustard poultice over the loins is undoubtedly of use. One may be applied daily for an hour. Dry-cupping, and even leeching, over the kidney region may also be tried, but they are not so efficacious as the hot poultices.

There is some difference of opinion as to whether diuretics should be administered in the early stage of an acute nephritis. There is, however, no doubt that the patient ought to drink water and other diluents freely, and the writer generally orders 5- or 10-grain doses of potassium acetate combined with the above diaphoretic mixture. In the later stages, diuretin (5 grains three times a day), or theocin in the same doses, are useful if the amount of urine remains much below normal. For the anæmia which is often present during convalescence, a mixture of tinctura ferri perchloridi, spiritus ætheris nitrosi, glycerini, ââ ℥x , aquam ad Si , thrice daily, is sometimes useful.

When uræmic symptoms supervene, in addition to acting on the skin and bowels, blood should be withdrawn by venesection or by leeching. If there are convulsions, 5 to 10 grains of chloral should be given either by the mouth or by the bowel, and repeated at intervals of from two to three hours, according to the effect produced. Lumbar puncture and the withdrawal of a test-tubeful of cerebro-spinal fluid sometimes gives relief, and ought to be tried.

Chronic Nephritis.—Children with chronic nephritis ought to be protected from chills by warm clothing, and where possible by residence, during the winter months, in a warm climate. The skin should be kept active by warm baths. A mistake which is often committed is to restrict these children far too rigidly to milk and farinaceous foods. There is little doubt that a diet of this kind does more harm, by affecting the general nutrition, than good, in diminishing the albuminuria. It is better, so long as there are no acute symptoms, to allow an ordinary diet, omitting articles which are rich in purins, and all spices, which are apt to irritate the kidney on account of the volatile oils they contain.

When there is œdema, a salt-free diet should be tried; it is often very successful. A strict diet is constructed according to the following rules: (1) No salt is to be used in cooking, nor is any to be eaten at mealtimes. (2) Ordinary bread, including cakes and scones from the baker, fish (except freshwater fish), salt butter, cheese, all forms of dried, smoked, salted, and otherwise preserved food, such as tinned meat, bacon, dried fish, and the like, and sauces and pickles, must be avoided. (3) Not more than a pint of milk should be taken in the day. (4) Soups made from ordinary meat stock contain large quantities of salt; without this they are unpalatable. The soup for a patient undergoing a strict course of treatment should be made from vegetables, with a milk stock. (5) Apart from these restrictions, the patient may eat what he likes. He may have from $\frac{1}{4}$ to $\frac{1}{2}$ pound of meat, or its equivalent as poultry or freshwater fish, per day, and vegetables, cereals, bread made without salt, one or two eggs, etc., as his appetite demands. Tea and coffee are not prohibited. The liberal diet which is thus possible is much less insupportable than the "light diet" to which many nephritic patients are restricted, and children quickly become habituated to the absence of salt. After a time the diet may be relaxed somewhat, the addition of ordinary bread being the most grateful change. The good effect of desalination is illustrated by the following case:

The patient, Lewis C., a boy aged ten, at present has chronic nephritis of six and a half years' duration. During the first four years of his illness he was admitted seven times to hospital on account of general dropsy, with scanty urine containing

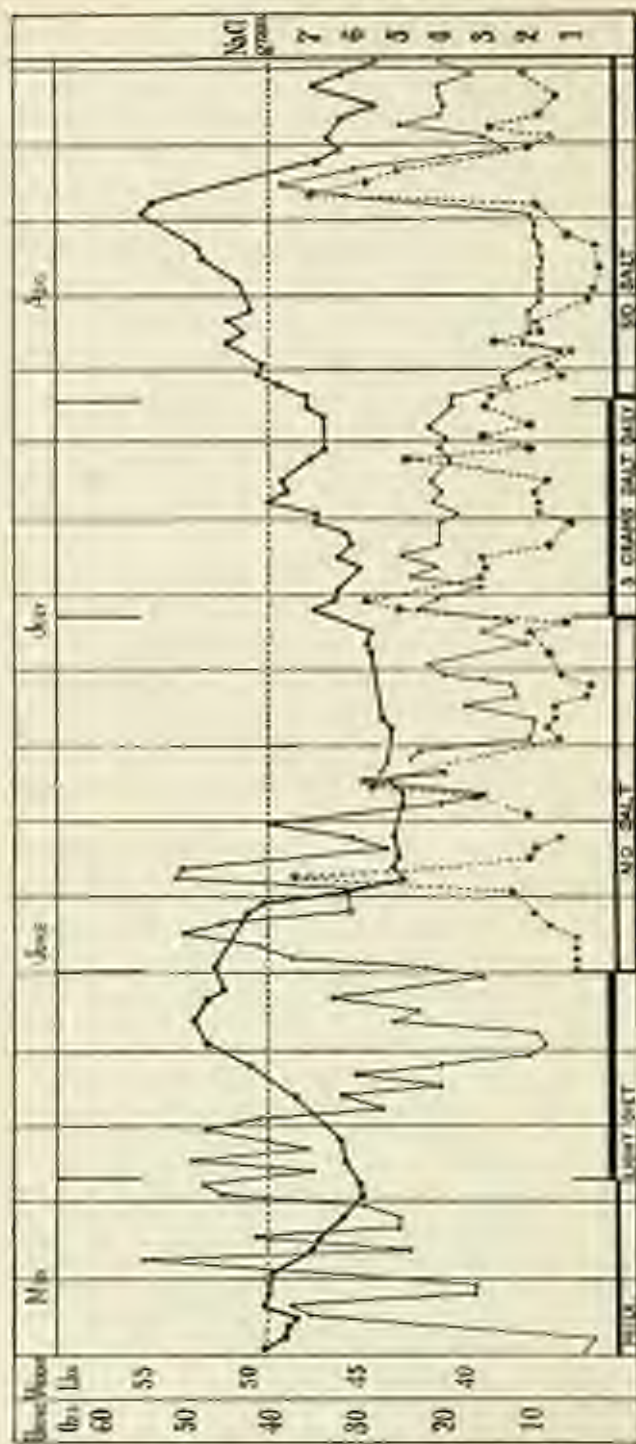


FIGURE 1.—STOMACH RECORD OF SALT-FREE DIET ON ARREST OF URINE (—), URINE (---), AND BODY WEIGHT (· · ·).

The horizontal dotted line shows the limit of weight without visible edema. The chart is divided into five periods: (1) Sixteen days—Milk diet; (2) Sixteen days—Light diet; (3) Sixteen days—Light diet; (4) Sixteen days—Light diet; (5) Sixteen days—Light diet. The chart shows the disappearance of edema, the appearance of edema, the disappearance of edema, the appearance of edema, and the disappearance of edema. Chloride output at first high, then low. (4) Twenty days—added salt; disappearance of edema, steady urine, chloride retention. (5) Thirty-one days—salt-free diet; edema, loss of edema, and increased output of chlorides after a delay of eighteen days.

much albumin and many granular casts. While outside he had a number of slighter attacks; in fact, he was scarcely ever free from some puffiness, and regularly attended the out-patient department for this reason. So little responsive was his malady to treatment that at one time the question of decapsulation was seriously considered. When he was seven and a half years old he was again admitted with general dropsy (see Chart I.). Under a milk diet this improved temporarily, but as soon as the diet was increased the output of urine fell and the dropsy returned. He was then put on a salt-free diet, with immediate benefit, the quantity of urine at once rising and the oedema disappearing, with a coincident fall in the body-weight. After his weight and the amount of chlorides excreted had become stationary, salt was added to his food, with the result that there was a recurrence of the oedema, which, however, disappeared, though after a somewhat longer interval, when he was again placed on a salt-free diet. Since leaving hospital in September, 1908, the salt in his food has been restricted, and from then until now (December, 1911) there has only been one slight attack of oedema. Apart from the constant presence of albumin and casts, the boy has been perfectly well and able to attend school.

Decapsulation (Edebohl's Operation).—Decapsulation of the kidneys has been advised in cases of chronic parenchymatous nephritis in children who do not respond to ordinary treatment, and several cases in which it has been performed have been reported. A cure is not expected from decapsulation, but sometimes the symptoms, especially the constant tendency to anasarca, are improved. Koplik has recently reported four successful cases of this kind. The operation ought not to be done when uræmic symptoms are present, and it is doubtful whether it is justifiable under any circumstances. It ought not to be considered until dechlorination has been thoroughly tried and failed.

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BACTERIAL INFECTIONS OF THE URINARY TRACT.

Bacterial infections of the urinary tract are not uncommon in childhood. They constitute an important group of diseases from the clinician's standpoint, because the symptoms are often so misleading that the cause escapes recognition and treatment, whereas, when the nature of the condition is realized, a dangerous disease can in many instances be speedily checked by appropriate treatment. Bacterial infections are usually primary; sometimes they are secondary to some local condition (e.g., stone) or to general septicæmia. The most common, and by far the most important, infecting organism is *B. coli*, which is found in about three-fourths of all cases; next in importance come staphylococcal and streptococcal infections. Other organisms, such as *B. aerogenes capsulatus*, Friedländer's

bacillus, *B. enteritidis*, *B. typhosus*, gonococcus, *B. pyocyaneus*, *B. proteus vulgaris*, and *B. tuberculosis*, are much less frequently met with.

Bacterial infections give rise—(1) To simple bacilluria, (2) to cystitis, (3) to pyelitis, and (4) to suppurative pyelonephritis; and though these conditions merge into one another to some extent, it is usually possible to distinguish between them clinically. The best known and most typical are the *B. coli* infections, which therefore will be first considered. Much of our knowledge of them is due to the writings of Helt in America, Escherich in Germany, and John Thomson in this country.

A. Infections with *B. Coli* and Allied Organisms.—In most of these cases *B. coli* can be recovered from the urine in pure culture, but in some instances there is a mixed infection, usually with staphylococci. We must, however, distinguish between a mixed infection from the bacteriologist's point of view and a mixed infection from the clinical standpoint. Many urines from cases which are clinically *B. coli* cystitis or *B. coli* pyelitis contain, besides the predominating organism, a few extraneous germs, without the clinical picture or course of the disease being apparently influenced thereby. Only in a minority of cases is there a true mixed infection in which both the colon bacillus and, for instance, pyogenic cocci take part; but when they occur, such cases are much less amenable to treatment than those due to *B. coli* alone. It is with the latter that we are now concerned.

An idea of the relative frequency with which different bacteria occur in the urine of children is afforded by the following table. The figures are for consecutive cases in which bacteriological examination was positive.

	Total	<i>B. coli</i> alone.	<i>B. coli</i> with staphylococci or streptococci	<i>B. coli</i> with other organisms	Friedländer's bacillus.	<i>B. aerogenes capsulatus</i>	<i>B. typhosus</i>	<i>Escherichia</i>	<i>B. proteus vulgaris</i>	Staphylococci.	Streptococci
Great Ormond Street	58	50	10	—	—	—	—	4	5	12	
Edinburgh Children's Hospital	45	26	10	2	1	1	1	—	—	5	
	103	82	20	2	1	1	1	2	5	17	0
Colon typhoid group. 109 = 80 per cent.											
24 = 20 per cent.											

The large proportion of organisms belonging to the colon-typhoid group is noteworthy; in addition to *B. coli*, we find Friedländer's bacillus, *B. enteritidis*, *B. typhosus*, and *B. aerogenes capsulatus*. *B. proteus vulgaris* is not a member of the group, and I have seen no cases of cystitis due to it, but Jeffreys states that they closely resemble the *B. coli* cases clinically. The acute infections are most frequently pure, and tend to occur in infants; chronic infections are more common in older children, and are often mixed. In nearly all cases of *B. coli* infection the urine is markedly acid when passed.

Ætiology.—Sex is the most important factor. Girls are much more liable to the disease than boys. This is the experience of all who have written on the subject. Thus, Gippert had only 10 to 11 per cent. of boys in his series of 108

cases; Lethbrg, 8 per cent.; Thomson, 4 cases out of 35. There is, however, some reason to think that in one clinical group of cases—viz., suppurative pyelo-nephritis—boys are more frequently affected; but, as these cases are relatively uncommon, the fact does not invalidate the general statement that infections with *B. coli* are about ten times as common in females as in males. Notwithstanding certain objections, the obvious explanation of this special proclivity of the female—viz., a greater liability to ascending urethral infection—is probably correct (*cf. infra*).

Age.—Children under two years of age form a majority of the sufferers (93 out of 108—Göppert). In Jefferys' series the average of sixty patients was five years. While the disease may develop during the first month, it is most common between the third and ninth months, and thereafter diminishes in frequency. In older children its manifestations are less striking and typical than in infants.

Season.—The disease is somewhat more prevalent in summer than in winter.

Predisposing Causes.—The chief set of predisposing causes are connected with the intestinal function. While some patients appear to have been in good health up to the onset of their illness, in most there is a history of diarrhoea, indigestion, or constipation. The last was present in twelve of Thomson's series, and he notes that treatment by suppositories or injections had often been adopted. Box suggests that threadworms may cause a urethral infection. Any debilitating illness seems to render a child more liable to the infection, which has been recorded in the course of scurvy (Ashby) and anaemia. Several cases are reported as having followed circumcision. *B. coli* infections are more common in hand-fed than in breast-fed infants; they are as frequent among the better as among the poorer class of the community.

Symptomatology.—Clinically several types of *B. coli* infection can be recognized.

1. *Simple Bacilluria*.—This is not uncommon; it gives rise to few or no symptoms, and is usually discovered only on routine examination. There may, however, be enuresis or frequent micturition. The urine is acid, and contains the organisms, but no pus. Some cases of bacilluria probably go on to cystitis, but in many no recognizable inflammatory reaction ensues, even although the condition persists over a long period. Why some infections should remain thus limited is not known.

While bacilluria of this kind is met with apart from other diseases, it seems to be relatively common in cases of follicular enteritis. Truapp found it in thirteen out of sixteen patients; in some bacilli were present alone, in others there was a mild degree of cystitis. In this connection Wroden's experiments may be cited. In rabbits he found that any abrasion of the rectum above the level of the prostate caused cystitis. In tuberculous meningitis it is often possible to discover colon bacilli in the urine, and this fact is of practical consequence, because it has led to the disease being mistaken for pyelitis. Bacilluria sometimes occurs in the course of measles, diphtheria, scarlet fever, and other acute disorders.

2. *Cystitis*.—In cystitis the urine contains pus cells in addition to bacilli. The urine is acid, pale yellow, and turbid, but in pure *B. coli* infections does not deposit a heavy layer of pus. It has sometimes a disagreeable odour, and contains a trace of albumin. The ordinary symptoms of cystitis—frequent and more or less painful micturition, etc.—are generally present, especially in older children, whereas in young infants they are often difficult to recognize. There is little or no fever

and no marked constitutional disturbance. The leading symptom of cystitis in young infants is extreme irritability and fretfulness; pallor is often marked. In any doubtful case of this kind, especially if the temperature is raised to 99° or 100° F. without apparent cause, the urine should be examined.

3. *Pyelitis (Pyelo-cystitis).*—The acute pyelitis of infants constitutes the most important group in this infection. Its salient feature is the predominance of severe constitutional disturbances, local symptoms being obscure. The disease sets in acutely with high temperature and usually vomiting; restlessness, drowsiness, and even delirium, are common; squinting and stiffness of the neck may occur. The child is fretful, looks wretched, and resents handling as if in pain. A history of previous cystitis can sometimes be elicited. On account of the irritability, it is often impossible to be sure whether there is any abdominal tenderness, but in some cases there is resistance over one or both loins, and occasionally an enlarged kidney can be detected on palpation. As Thomson has pointed out, rigors occur in many cases, and are of great assistance in diagnosis, because at this age they are



GRAPH II.—ACUTE COLIC-PELITIS, SHOWING RELATIVE WITH ABSENCE OF BACILLI FROM URINE, DUE TO TEMPORARY BLOCKING OF URETER.

practically unknown in this country in any disease except acute pyelitis. Certainly, in a large proportion of cases a history of "shivering fits" or "shuddering" is obtained. These attacks are sometimes associated with unconsciousness, or pass into convulsions. Frequent painful micturition occurs in some, but not all, cases. There is generally a moderate leucocytosis. The urine is strongly acid when passed, but becomes alkaline on standing; it is therefore important to test the reaction while fresh. To the naked eye it has the characters described above under Cystitis. When a drop is placed under the microscope, numerous bacilli, single or in clumps, and pus cells, with occasionally one or two hyaline or granular casts, are seen. The bacilli are the important feature, because they are present from the onset of the illness, whereas pus is often absent for the first day or two.

Course of the Disease.—If the disease be untreated or inefficiently treated, the above symptoms continue. The temperature remains high—from 101° to 103° F. in an average case—and is generally of an irregularly remittent type. I cannot speak from personal knowledge of the course of the disease apart from its treatment by alkalies (which abort it within a few days); but if one may judge from

reported cases, it would seem that, while in mild infections the temperature may fall spontaneously in the course of ten to fifteen days, the more severe cases tend to run a protracted course of weeks or months, with alternating periods of fever and apyrexia, eventually either becoming chronic or progressing to abscess formation in the kidney and death. It is important to note that in the later stages of the disease both pus and bacilli may temporarily disappear from the urine. Gipsper's explanation—that this is due to transient blocking of the affected ureter—is probably the right one. The writer has known the disappearance of the organism coincide with a relapse, which supports the view that blocking had occurred (see Chart II.).

Pyelitis in Older Children.—In acute pyelitis or pyelo-cystitis in older children the constitutional symptoms are less severe, and the local symptoms more manifest. There is generally, however, marked irritability and loss of appetite. The course of the temperature resembles that in infancy, but the range is less. In chronic cases the patients are usually pale and ill-nourished, and suffer from enuresis or some degree of dysuria. They are liable to periodical exacerbations of their symptoms, accompanied by more or less fever. In some cases these attacks simulate periodic vomiting (Thursfield). Joint pains, filigreeing, and headaches, may occur. Box has observed uræmic coma supervene.

4. *Suppurative Pyelo-nephritis.*—In comparison with acute pyelitis, suppurative pyelo-nephritis is rare—fortunately, because, when multiple abscesses form in the kidney substance, recovery, if not impossible, is at least unlikely. Five fatal cases of suppurative pyelo-nephritis have been examined post mortem in the Edinburgh Sick Children's Hospital during the past three years; two have been already reported by Thomson, and three came under the writer's care. In some instances the clinical picture is sufficiently clear to allow of this variety being distinguished from acute pyelitis, and from the point of view of prognosis this is of some moment. The following description is founded on the symptoms of these five cases: The ages of the patients ranged from three to thirteen months, and four of the five were boys. The symptoms were not unlike those described above, but were less acute, and in only two was the onset sudden. No patient had a rigor. In the antecedent history, vomiting and diarrhoea (three), abdominal pain (one), painful micturition (one), wasting and fretfulness (four), figured. In one case the illness came on two days after an ischio-rectal abscess was opened. All the patients were extremely prostrate, most were pale and feeble, and two were emaciated and cachectic. In all cases the urine contained pus and bacilli; a moderate degree of abdominal distension, but no tenderness, was noted. In two cases the kidney was enlarged and palpable. The temperature was high (104° F.) in one case, temporarily raised to 101° – 102° F. in two cases, practically normal in two. The duration of the illness was apparently only two or three weeks in the cases where the onset was definite; in one there had evidently been a chronic infection of some considerable duration. Alkalies did no good in any case. The prominent clinical features of this form of pyuria are—(1) Extreme prostration, even with a normal temperature; (2) absence of rigors; (3) failure of response to alkalies. These, especially in the case of a boy, and if one or both kidneys are found to be enlarged, are highly suggestive of suppurative pyelo-nephritis.

PATHEOLOGY.—The anatomical changes in mild cases of *B. coli* infection are not well known, because recovery is the rule. In bacilluria without symptoms no lesions are found post mortem, and the writer has known the urine to contain pus and

bacilli for several months before death without there being any macroscopic changes whatever in the bladder. Frequent painful micturition may exist without recognizable cystoscopic changes, but in some of Jeffreys' cases congestion of the mucosa, redness of the ureteral orifices with plugs of mucus, and ulceration of the bladder wall were seen. No fatal case of acute pyelitis uncomplicated by abscesses in the kidneys has occurred in the Edinburgh Sick Children's Hospital, but in such cases as have been reported the pelvis of the kidney and the ureters showed swelling of the mucous membrane, and contained creamy pus. In more chronic cases the renal pelvis and ureter are dilated, and there is evidence of commencing inspiration of the kidneys, which are slightly enlarged, and when the capsule is removed show surface mottling of pale areas surrounded by congestion. These pale areas are due to foci of commencing suppuration, which on section of the organ appear as yellowish-white radiating streaks, some reaching, some stopping short of, the periphery of the organ. Both kidneys may be affected, but if the lesions are unilateral they are generally right-sided. In suppurative pyelo-nephritis these appearances are exaggerated. The kidneys are usually considerably enlarged, and on section show many small abscesses, often grouped in wedge-shaped clusters, some of which project slightly on the surface of the organ. The abscesses vary in size from a pin's head to a pea, and on section are yellowish-white, surrounded by a congested zone. In addition to groups of abscesses, radiating pale lines, without actual suppuration, may be present. Microscopically the changes ordinarily associated with suppurative nephritis are found, and there is also pyelitis, the calyces and apices of the pyramids being here and there denuded of their epithelial covering, while the subjacent tissues are acutely inflamed and infiltrated with lymphocytes. In the two cases carefully examined by McDonald no embolic foci of the infecting organism could be detected, and the appearances were suggestive of an ascending infection, either from the erosions in the pelvis or by the collecting tubules. In old-standing cases widespread changes take place in the urinary organs—hypertrophy and dilatation with chronic inflammation of the bladder and ureters, hydronephrosis and atrophy of the kidney substance, pyonephrosis, or fibroid changes round old inflammatory foci. It is possible, however, that in some such cases the hydronephrosis is secondary, and that it has favoured the occurrence of the pyelitis.

The pathogenesis of these affections is not yet settled. How does the organism invade the urinary tract? Hypothetically it may obtain access (1) by the urethra, (2) directly from the bowel by way of the lymphatics, (3) from the blood-stream, the bacilli being excreted by the kidney. In the first two modes the infection is ascending, in the third descending. The group of cases we are discussing do not occur as part of a general septicæmia, and the not infrequent limitation of the lesion to *one* side favours the idea of an ascending, and almost negatives a blood, infection. Discarding the third mode of infection as unproven, what of the other two? Infection by the urethra seems the more probable. The great preponderance of females is simply accounted for on this hypothesis, and is not explained by direct spread from the bowel. That it is mechanically possible for bacilli to pass up the ureters is shown by Bard's experiments, which proved that particles of pigment could pass along them against the course of the urinary stream. Another fact which is in favour of urethral infection is the frequency with which cystitis accompanies or precedes pyelitis, or occurs independently of it. There is no direct pathological evidence of a lymphatic spread. The colon bacillus cannot pass through

the intact mucous membrane of the healthy bowel, but in these cases there is so frequently a history of intestinal trouble that it is reasonable to suppose that some breach of continuity may exist. Trunpp's observations on bacilluria in follicular enteritis are suggestive in this connection. Jeffreys explains the more frequent involvement of the right kidney, on the hypothesis that there is a direct spread from the bowel, by "the close anatomical relation of the right kidney and the ascending colon, caecum, and appendix." No one, however, has actually demonstrated the spread of a *B. coli* infection from the bowel by way of the lymphatics, and it is a matter merely of pathological interest whether the organisms travel along the lumen of the urinary canal, the surface of the mucous membrane, or the lymphatics of the submucous coat, or by a combination of these routes.

Infection with an organism which has its normal habitat in the intestine would naturally be favoured by a weakening of the defences of the host, increased virulence of the organism, or increased opportunities for infection. Except that most of the patients are debilitated, nothing is known as to the first of these possibilities. Although under certain circumstances *B. coli* may be responsible for some forms of diarrhoea, it is not clear that there is any concomitant increase of ability to invade the tissues. From a bacteriological point of view there is not much evidence that this organism tends to acquire increased pathogenic properties. It is easy to see, however, that diarrhoea, with its frequent soiling of the perineum, must multiply the chances of the organism entering the urethra, and Trunpp's results can be readily accounted for in this way. While, therefore, the possibility of a direct spread from the bowel cannot be denied, it is probable that in most cases infection takes place by the urethra.

Pfandler has shown that in cases of pyelo-cystitis due to *B. proteus* and *B. coli* the patient's serum agglutinates the organism obtained from the urine. The same observation was made by Ritchie in two fatal cases reported by Thomson, and the serum of one of them also agglutinated the organism derived from the other, but not an ordinary laboratory culture. These facts show that there is a general as well as a local reaction, but they yield no further information, and cannot be employed in diagnosis.

The coli-typhoid group of organisms is large and important. Jordan classifies the chief forms thus: (1) *B. coli communis*, *B. lactis aerogenes*, *B. pneumonise*; (2) *B. enteritidis*, *B. paratyphosus* A and B; (3) *B. typhosus*, *B. dysenteriae*. It is noteworthy how largely the members of this group preponderate among the organisms infecting the urinary tract in children.

DIAGNOSIS.—The diagnosis turns on the examination of the urine. To avoid contamination with secretion of the vulva, etc., a catheter specimen should be obtained. As only bacilli, but no pus cells, may be present at the onset, microscopic as well as naked-eye examination is essential. The organisms can be seen in a drop of freshly-passed urine; staining and culture are necessary only for exact bacteriological diagnosis. The presence of pus in an acid urine may arouse the suspicion of tubercle. Tuberculosis of the urinary tract is, however, rare, especially in infancy, in comparison with *B. coli* infection. At the same time, in older children, it must be borne in mind that the latter organism may be grafted on to a primary tuberculous infection. The severity of the symptoms serves to distinguish pyelitis from cystitis. Pyelitis is apt to be mistaken for pneumonia, meningitis, and otitis. The extreme distress of the patient, the remittent, irregular temperature, and the

history of shivering, together with the absence of localizing signs in the chest or nervous system, ought to give a clue. As has been said, a few *B. coli* are often found in the urine in tuberculous meningitis. Chronic *B. coli* infections are liable to be mistaken for latent tuberculosis, or may simulate periodic vomiting. To differentiate between pyelitis and suppurative pyelo-nephritis is doubtless impossible in many cases. The distinctive features have been alluded to under the symptomatology; those on which most reliance should be placed are the failure of alkaline treatment to improve matters, the presence of palpable enlargement of the kidneys and the extreme prostration.

PROGNOSIS.—The prognosis of these bacterial infections is somewhat uncertain. Excluding cases of simple bacilluria, and including suppurative pyelo-nephritis, the mortality is about 10 per cent. The immediate prognosis as regards life is

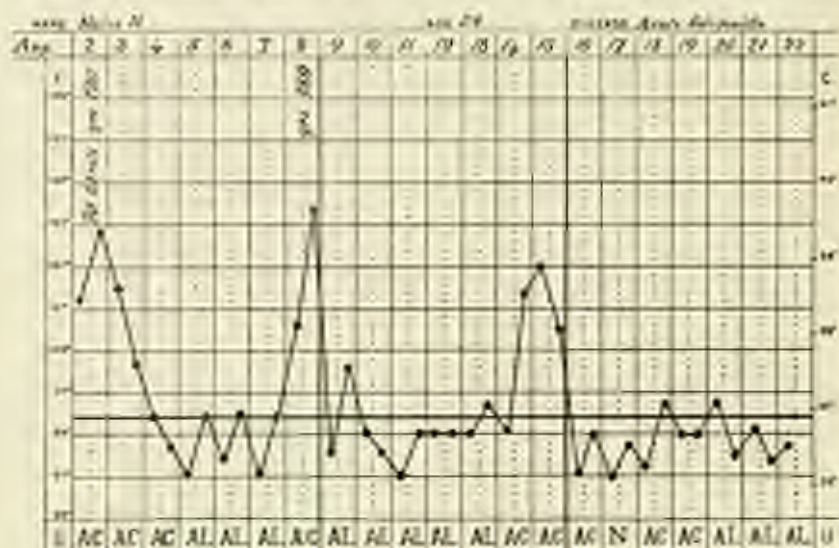


CHART III.—ACUTE COLI-PELITIS, SHOWING (a) EFFECT OF ALKALIES, AND (b) SECONDARY RISES OF TEMPERATURE.

therefore not unfavourable. Much depends, however, on the promptitude with which the disease is recognized, and the effectiveness of the treatment. The prognosis is extremely grave in suppurative pyelo-nephritis, and is less favourable when there is a mixed infection than when the colon bacillus is the sole cause of the disease. So long as bacteria are present in the urine relapse is possible, and when the disease has become chronic it is difficult to render the urine sterile. This is apparently the reason why the disease is less amenable to treatment in children of five or six years than in infants. Relapses may occur at intervals of a year or more, but most cases ultimately recover. From the point of view of prognosis it is practically important to impress parents with the necessity of keeping children suffering from urinary infections under medical observation until the urine is free from bacilli. A symptomatic cure is not enough.

The best means of treating *B. coli* infections is to render the urine alkaline, and to keep it alkaline, as advised by Thomson. This is done by giving potassium

citrate in adequate doses. For infants 48 to 60 grains should be given daily, and if the urine still remains alkaline the dose should be increased to 2 to 3 drachms. The urine generally becomes alkaline in four or five days, but a week may elapse before it does so (Thomson). The effect of the alkaline treatment is to bring down the temperature, relieve the symptoms, and remove the pus from the urine, though bacilluria may persist for some time. The urine often tends to become acid again a few days after the temperature has fallen, and this is associated with a secondary rise of temperature. When this happens, the citrate should be continued (see Charts III. and IV.). In large doses the drug may cause diarrhea; and if for this or any other reason there is difficulty in administering it by the mouth, it may be given by the rectum (McLaw). A child suffering from acute pyelitis should be kept in bed, and must take plenty of diluents to promote diuresis. The bowels should be kept open with sodium phosphate or calomel.

The alkaline treatment is so successful in acute pyelo-cystitis that other remedies

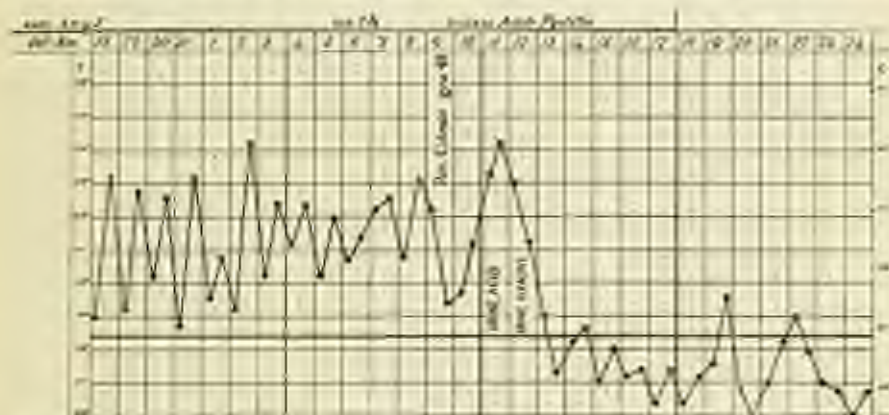


CHART IV.—ACUTE CHOLECYSTITIS, SHOWING TEMPERATURE CURVE BEFORE AND AFTER ALKALINE TREATMENT, AND SLIGHT SECONDARY RISE. (AFTER THOMSON.)

are seldom required, and ought not to be used until the citrate has had a thorough trial. There remain, however, urinary antiseptics and vaccines; these will be referred to in connection with mixed infections. Hexamethylamine (urotropin) and its allies are of little or no use in infections with *B. coli*, as the organism is resistant to this series of antiseptics.

In chronic *B. coli* cystitis and pyelitis of older children the results of alkaline treatment are less satisfactory, and if it fails the remedies described under Mixed Infections should be tried. By way of prophylaxis, threadworms ought to be treated, and the cleanliness of the perianal region attended to.

B. Staphylococcal, Streptococcal, Mixed, and Other Infections.—As a rule staphylococci or streptococci are not found in pure culture, but are associated with organisms of the coli-typhoid group (see table, p. 640). Most patients suffering from urinary disorders due to these organisms are over two years of age. The symptoms produced are much the same as those described in connection with chronic *B. coli* infections. There is usually frequent and painful micturition; the urine may be acid or alkaline and offensive, and often contains a little blood. Pus is more abundant than in the pure *B. coli* cases.

Infection with other organisms is comparatively rare, and usually occurs as part of a general septicæmia—e.g. *B. pyocyaneus* cystitis. Differential diagnosis is possible only by bacteriological examination, which should always be performed in order that the appropriate vaccine may be employed if need be. In chronic mixed staphylococcal or streptococcal infections with an acid urine, the suspicion of tubercle may arise. Tubercle bacilli are seldom demonstrable in the urine by ordinary staining methods, and animal inoculation may be necessary to settle the point. Tubercle of the urinary tract is, however, rare in children. These forms of chronic pyelitis and cystitis may be secondary to stone in the bladder or kidney. In obstinate cases the bladder should be sounded, and the kidneys examined by X-rays.

TREATMENT.—The patient ought to drink freely of water and bland fluids, and to remain in bed while the symptoms are acute. Alkaline treatment generally does harm, because staphylococci, the organisms most often present, flourish in alkaline media. The principal lines of treatment are—Urinary antiseptics, vaccines, irrigation of the bladder. *Urinary Antiseptics*: The formaldehyde series are the most useful; of these, hexamethylenamine (protosol) and hexmitol are the chief. The dose is 15 grains three times a day. Salol, in 5-grain doses, is sometimes effective. Sodium salicylate or ammonium benzoate may also be given. In the more acute infections sedatives are needed, and sandalwood-oil is one of the best of these. Diuretics, such as spiritus ætheris nitrosi, are also indicated. Vaccines are probably of no use in acute cases, but should be tried in chronic infections. For staphylococci a stock vaccine suffices; in other cases an autogenous vaccine is essential. In *B. coli* infections the initial dose of an autogenous vaccine is 2,500,000 to 5,000,000, repeated at intervals of two or three days, and subsequently larger doses up to 50,000,000 are given, generally at intervals of a week. In *B. proteus* infections, Jeffreys reports success with a vaccine. *Irrigation of the bladder* ought to be reserved for chronic cystitis; in acute inflammation all unnecessary instrumentation is better avoided. Boric acid lotion is generally used, or weak solutions of the astringent metallic salts. Jeffreys advises that after washing out the bladder an injection of iodoform emulsion should be given.

In addition to these measures, any discoverable cause should be dealt with. Threadworms or vulvo-vaginitis may require treatment. If ulceration of the colon is suspected, the bowel may be irrigated. A few cases in which unilateral pyonephrosis has developed have been operated on successfully. Cases of stone also require surgical treatment.

In chronic cases it is generally well to change the urinary antiseptic from time to time, and to be guided by the bacteriological finding as far as possible.

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TUMOURS OF THE KIDNEY

Neoplasms of the kidney are relatively frequent during early life. They are in many respects of great interest. A number of them are of developmental origin, and hence are commonly met with in infants or quite young children. From recent work on the subject, it seems that we must distinguish more definitely than hitherto between renal tumour, particularly the so-called "renal hypernephroma," and tumour of the adrenal glands. The latter may give rise, according to circumstances, to remarkable nutritive alterations, such as precocious puberty and excessive bodily development, or to metastases in special localities, such as the skull and orbits, producing a characteristic clinical syndrome. Hypernephroma proper, therefore, falls to be discussed in connection with diseases of the ductless glands (vide Chapter X., p. 573). A remarkable and quite unexplained feature in some cases of renal tumour is the co-existence with them of cerebral lesions, such as glioma, psammoma, and psammo-lipoma, and in particular of the curious condition tuberous sclerosis (q.v., p. 882).

Tumours of the kidney vary from the absolutely benign to the extremely malignant. Among benign tumours, lipomata, fibromata, and fibro-lipomata, may be noted. They seldom exceed the size of a walnut, and give rise to no clinical symptoms. To the highly malignant group belong sarcoma and carcinoma, while intermediate between the simple and malignant tumours come so-called "hypernephromata," which may remain benign, or exhibit more or less malignancy and give rise to secondary deposits in other organs. In some cases of leukaemia the kidneys become so much enlarged by lymphocytic deposits as to give rise to well-masked renal tumours.

Most simple tumours are of no clinical importance, and require no further remark. Benign tumours of the kidney are met with in at least 60 per cent. of cases of tuberous sclerosis (Fischer). They are mixed neoplasms, consisting of non-striated muscle, fat, arteries, and sometimes primitive kidney cells, in variable proportions; lipo-myomata, angio-lipomata, fibro-lipo-myomata, lipo-myo-sarcomata, are some of the names which have been used to describe them. They very seldom give rise to symptoms during life.

Hypernephromata are supposed, following Grawitz's hypothesis, to arise from aberrant masses of adrenal tissue which have become embedded in the kidney during development—adrenal "rests," as they are called. These tumours are more or less distinctly demarcated from the kidney tissue by a fibrous capsule; they are irregularly rounded in shape, and may attain the size of the closed fist. Occasionally they are found to contain cartilage, or even bone. In some cases metastases occur to the bones, liver, opposite kidney, etc. Doubt has recently been cast on the correctness of this view of the origin of renal hypernephromata on the following grounds (Glynn): (1) Adrenal "rests" are apparently rarely met with in the kidney. (2) Microscopically a renal hypernephroma differs markedly from a true hypernephroma arising from the adrenal glands. According to Glynn, it consists of polygonal or cylindrical epithelial-like cells with round or oval nuclei and a clear cytoplasm; smaller dark cells with granules may also be present. The cells may be arranged either in solid alveoli, or in tubular acini con-

taining degenerated epithelium and blood, in a vascular stroma. (3) It is improbable that true renal tumours, whether so-called "hypernephromata" or sarcomata, ever give rise to the affections of nutrition associated with tumours of the adrenal glands, such as are described elsewhere.

Round- and spindle-celled sarcoma of the kidney is met with, and also mixed types, such as lympho-sarcoma. Rhabdo-myomata, or myo-sarcomata, consisting of striped muscle fibres together with sarcoma elements, sometimes occur in early infancy. They are embryomata (teratomata), and arise from fetal intermingling of tissues. Sarcomata are as a rule encapsulated, and grow rapidly, destroying the kidney substance; they may attain an enormous size—15 to 20 pounds, or even more. Metastases to the other kidney and elsewhere occur early.

Primary carcinoma of the kidney is much less common than sarcoma, but occasionally occurs.

CLINICAL FEATURES.—A large proportion of cases occur in children during the first five years of life. In Steffen's collected series of 219 cases in patients up to sixteen years of age, 34 occurred in the first year, 53 in the second year, and 165 during the first five years. Boys are somewhat more liable than girls, though the difference between the sexes in this respect is less than in adult life.

The leading symptom is the presence of an abdominal tumour, which may be very large. The tumour is situated in the loin, is usually painless, and has a more or less irregularly rounded contour. The colon lies in front of it; it moves very slightly with respiration. There are often no urinary changes whatever; in particular, the presence in the urine of recognizable tumour elements is very rare. Hematuria is the most common of the urinary symptoms; it may be painless and occur early in the disease. It is more often met with in carcinoma than in sarcoma. As the tumour grows it becomes adherent to other structures in the neighbourhood, and various pressure symptoms may arise. Patients suffering from malignant disease of the kidney emaciate rapidly; gastro-intestinal symptoms are common, and in the later stages of the disease there may be much abdominal pain and discomfort.

The **DIAGNOSIS** of a renal new growth is not always easy. It has to be distinguished from (1) other forms of abdominal tumour and (2) other enlargements of the kidney. The points in favour of kidney tumour are—(1) The relation of the colon, which may be rendered more evident by inflating the bowel; and (2) the position of the swelling, lying near the costal margin, and in many cases projecting into the loin. Suprarenal tumours are palpable nearer the middle line, and tumours of the liver and spleen move more distinctly with respiration and lie in closer relation to the costal arch. Pelvic tumours are rare in children, though dermoid cysts of the ovary are occasionally met with. The most common error is to mistake a large tuberculous deposit in the loin for a kidney tumour, and in some cases it is impossible to make a correct diagnosis even by abdominal palpation under chloroform, except by observing the progress of the case or exploring the abdomen. A clear history that the swelling dates back several months is against sarcoma. A negative examination of the urine does not exclude tumour.

TREATMENT.—Owing to the latency of the symptoms, a diagnosis is seldom made until the tumour has reached a considerable size—hence the results of operation are not good. As, however, surgery holds out the only possible prospect of

cure, an operation should be advised unless the child's general condition precludes any hope of success. Even after successful removal of the kidney restitutions is very likely to take place.

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PERINEPHRITIS AND PERINEPHRIC ABSCESS.

Inflammation of the cellular tissue surrounding the kidney, which may resolve, or (more commonly) go on to abscess formation, is occasionally met with in children. It may be secondary to some focus of inflammation in the neighbourhood, either in the kidney itself—e.g., calculus or abscess—or in the spine. I have known it arise from acute localized periostitis of the twelfth rib. More commonly, however, it is primary. In some cases it is due to trauma, but very often no cause can be discovered. In Glynn's series of 28 cases the majority of the patients were between three and ten years. There is no special predilection for either kidney, and both sexes are equally liable.

SYMPTOMATOLOGY.—The onset is generally acute, with fever. The symptoms are suggestive of hip or spinal disease. The leg on the affected side is held semi-flexed, and attempts to straighten it are painful, but it can be flexed on the abdomen without discomfort, and there is no fixation of the hip-joint. In the early stage there is no local swelling, but there is usually some tenderness in the loin. The patient is lame, and walks like a case of early hip disease—with the thigh flexed and the leg everted. As the disease progresses the symptoms increase, and some fulness develops in the lumbar region; there is often some deviation of the spine, with the concavity towards the affected side. If pus forms, the tissues become oedematous, the veins prominent, and the skin reddened. On bimanual palpation of the loin, an indistinct tumour, it may be with deep-seated fluctuation, can be made out. There may be frequent and painful micturition; pyelitis is a rare complication.

DIAGNOSIS.—The chief interest of perinephritis lies in the fact that it is liable to be mistaken for hip-joint disease. The resemblance is greatest in cases where there is no localized abscess. The main points of distinction are—(1) The only movement which is restricted is extension; the limb can be rotated, or fully flexed on the abdomen, without causing pain. In hip disease, on the other hand, there is limitation of the movements of the joint, and pain over it. (2) The onset of perinephritis is sudden, and marked lameness develops within a few days. In hip disease the onset is insidious. (3) The course of the disease is different. In perinephritis which resolves without suppuration the symptoms abate in a week or two; if an abscess appears, it will do so within the same time. Hip disease is much more chronic. Primary perinephritis is distinguished from spinal caries by absence of fixation of the spine. In cases secondary to vertebral disease rigidity of the spine will of course be present.

TREATMENT.—The patient should be kept in bed, and in the early stages an icebag or hot fomentations may be applied. If an abscess forms, it should be opened at once, lest it rupture into the peritoneum.

URINARY CALCULUS.

Urinary calculus, whether in the kidney or the bladder, is rare in infants. It is remarkable that this should be so, for the allied condition lithiasis is common at this age (see p. 600). In infancy there appears to be a considerable tendency to the deposit of uric acid crystals in the urinary passages, and the excretion of urine containing uric acid sand; the development of a stone of any size is, however, rare. After four years of age the tendency to calculus formation increases.

The frequency with which stone occurs in different countries varies; thus, it is common in Hungary, rare in the United States; in some parts of England it is not uncommon, while in Scotland generally it is rare. Very few cases have occurred during the last twelve years in the Edinburgh Sick Children's Hospital.



FIG. 66.—CORALLINE CALCULI IN KIDNEY OF A BOY AGED TWO YEARS AND SIX MONTHS.

Pathology.—In infants and young children calculi are generally composed of ammonium urate; in older children, approaching puberty, uric acid stones are most common. Calculi consisting of oxalates, phosphates, cystin, etc., are sometimes met with. Boys are more frequently affected than girls.

Renal Calculus may be single or multiple; calculi may be scattered throughout the kidney or lie in the pelvis. They may be round, or faceted from friction with one another. Branching calculi, forming a cast of the pelvis and calyces of the kidney, are rare. An example of this so-called "coralline calculus" is shown in Fig. 66. It was found on post-mortem examination in a case of mental defect, and, so far as could be ascertained, had given rise to no symptoms during life.

When a large calculus forms in the pelvis of the kidney, it may obstruct the outflow of urine, and give rise to hydro- or pyo-nephrosis or suppuration in the kidney substance.

SYMPTOMATOLOGY.—Renal calculus may exist for a considerable time without giving rise to symptoms. So soon, however, as it passes into the ureter it causes renal colic, which is generally attended by severe pain; rigors, vomiting, collapse, and even convulsions, may occur. The pain subsides speedily when the stone leaves the ureter, either by escaping into the bladder or returning to the pelvis of the kidney. In the latter case relapse is likely to occur. Renal colic is attended by pain in, and retraction of, the testicle.

Apart from these acute attacks of colic, there are often urinary changes. Attacks of hæmaturia with or without the passage of gravel may occur. The urine may contain traces of albumin or pus. Pain in the loins, increased by exercise, and alleviated by rest in bed, is common, and there is often tenderness on palpating

one or other kidney region. If hydronephrosis or pyonephrosis develops, the enlarged kidney can sometimes be felt.

DIAGNOSIS.—The diagnosis of renal colic in a child who is too young to describe the locality of the pain is extremely difficult. Definite tenderness in one loin, or the discovery of blood or gravel in the urine, together with absence of signs of intestinal disorder, would suggest it. In older children, the site and nature of the colic, and the testicular pain, ought to raise the suspicion of calculus. It must be remembered that, although the pain of renal colic is usually severe, it may be comparatively slight, and in some cases is quite overshadowed by the bilious vomiting. The diagnosis of stone in the kidney has been greatly simplified by the introduction of radiography, and this examination should be made in all cases of chronic pyuria in which there are any symptoms suggestive of the kidney being affected—pyrexia, tumour, tenderness in the loin, etc. The diagnosis between renal calculus and tubercle of the kidney may be very difficult; here radiographs are of the greatest value.

Vesical Calculus.—Stone in the bladder is a surgical affection, and requires only brief mention. In all cases of frequent painful micturition, for which no obvious cause can be found, and in which the symptoms are not promptly relieved by treatment, the bladder should be examined for a stone. This can be done by bimanual palpation under an anæsthetic, with one finger in the rectum, by sounding the bladder, or by radiography of the pelvis.

TREATMENT.—The treatment of urinary calculus in children is the same as in the adult. For the colic, warmth, alkaline diluents, and narcotics are required. During the intervals the patient should take alkaline drinks, such as Vichy water, freely. In all cases of stone the question of operation has to be considered.

TUBERCULOSIS OF THE KIDNEY.

Tuberculous disease of the kidneys in children differs in several respects from that of the adult. Broadly, it is more common as a pathological finding, and less common as a clinical disease.

In general tuberculosis the kidneys are often affected, while in adults they as a rule escape. They are found on post-mortem examination to be studded with grey or yellow milary tubercles, or yellowish deposits of somewhat larger size. This form of tuberculosis of the kidneys never gives rise to any symptoms during life, and is of no clinical importance. Large tuberculous caseating masses sometimes also occur, but as a rule the patient dies of general tuberculosis before the disease of the kidneys has reached an advanced stage.

Except as a part of general tuberculosis, tubercle of the kidneys is rare in children. When, however, it does occur, it has more tendency to remain localized in the kidney, and is seldom part of a widely-spread genito-urinary tuberculosis (*cf.* Tuberculous Testicle, p. 489). The infection is primarily of the kidney, and is a descending, not an ascending one. The disease, at its commencement at least, is nearly always unilateral, although when it reaches an advanced stage the opposite kidney usually becomes affected. In some cases there is evidence of a descending infection from the primary focus, and an ascending infection of the opposite kidney. The ureter is usually thickened and the seat of disease, but tuberculous

cystitis is uncommon. In most cases the tuberculous mischief begins in the cortex, and spreads to the papille and pelvis of the kidney; in advanced cases nearly the whole organ may consist of caseous material. Perinephric abscess is not uncommon, and in some cases renal calculus is also found. The ureter may become obstructed in the course of the disease, and pyonephrosis develop.

SYMPTOMATOLOGY.—The symptoms of tubercle of the kidney are often difficult to interpret. Pyuria is an early symptom, and there is often, also, more or less hæmaturia. Blood, however, may be absent for a considerable period, but pus is usually constant. It is not as a rule very abundant; small clots or fragments of caseous matter may be found in the urine, which is usually acid in reaction. Frequent and painful micturition and incontinence have been noted as early symptoms in several cases. In many cases these symptoms alone persist for several months, and, unless there are definite symptoms of tubercle elsewhere, diagnosis will be difficult or impossible. Sooner or later, however, pain or tenderness in one loin, and possibly a palpable tumour, will develop. If the swelling occurs rapidly and is accompanied by fever, it is suggestive of a secondary perinephric abscess or hydronephrosis. Owing to the fact that the disease may for long remain localized in one kidney, the general health may suffer but little, and in some cases recovery, or at any rate great improvement, is said to have occurred.

DIAGNOSIS.—Chronic pyuria is so commonly due to other organisms that caution is required in making a diagnosis of tubercle of the kidney. When pyuria continues for a considerable time, especially if the reaction of the urine is acid, the possibility of tuberculosis should be considered. While the detection of the bacillus in the urine by ordinary methods is positive proof, it is seldom obtained; merely negative findings, or the presence of pyogenic cocci alone, do not exclude tuberculosis. The only decisive test is to inoculate a guinea-pig.

In cases of chronic pyuria with a palpable swelling in the loin, pyelitis due to other causes has to be taken into account. A condition which simulates tubercle closely is renal calculus with the formation of abscess in the kidney. Such cases may run a chronic course, with an irregular temperature and general symptoms resembling tuberculosis. An X-ray examination of the kidneys ought therefore to be made.

TREATMENT.—So long as pyuria alone is present it is advisable to rely only on general measures, and on the free administration of alkalies, Vichy water, and other remedies for ordinary cystitis. At this stage of the disease it will usually be extremely difficult, if not impossible, to decide which kidney is affected, and considering that there is always the possibility of a natural cure, the question of operation may be delayed. If, however, there is a definite enlargement in one loin, or if it can be settled which kidney is affected, surgical intervention should be considered. Whether or not an operation should be undertaken will depend in the first place on the general condition of the patient; if there is evidence of generalization of the disease, operation is not likely to do good. If the tumour is large and tender, either incision and drainage, or nephrectomy, should be advised. The former operation seems to have given better results. If there is no, or only slight, enlargement of the kidney, it is advisable to delay operation until general measures have been fairly tried. If the enlargement has developed in spite of medical treatment, it is of course a different matter. When the disease does not respond

to medical treatment, operation is advisable even although it cannot be determined which kidney is affected. In such a case the abdomen should be opened in the middle line, and the diseased organ removed.

MALFORMATIONS OF THE BLADDER AND GENITAL ORGANS.

Extroversion of the Bladder.—This is the most common congenital malformation of the bladder. It is due to arrested development of the ventral wall of the urogenital canal, resulting in failure of fusion in the middle line. There is thus absence of the anterior wall of the bladder, of the corresponding part of the abdominal parietes, and defect of the pubic symphysis, which gapes one to four inches. The cleft is usually complete, but sometimes only the upper part, very rarely only the lower, is defective. In the male the external genitals also are deformed. The cleft continues along the dorsal surface of the penis, giving rise to epispadias; the penis is short and bent upwards, so that its dorsal surface is applied to the pubic region. The prostate is also rudimentary, and the dorsal part of the prepuce defective. The scrotum also may be absent, in which case the testes lie in the inguinal canal. In the female the labia are separated anteriorly, and the cleft urethra forms the lower part of the extrophy. In this malformation there is no sphincter at the neck of the bladder, and the ureters, which open at two nipple-like projections on the exposed surface, have often an abnormally twisted course, which may obstruct the outflow of urine and lead to hydronephrosis.

Clinically the condition is at once recognizable. The extroverted bladder forms a red protrusion in the pubic region, over the surface of which urine trickles from the ureteral openings. Owing to exposure, the bladder mucosa undergoes catarrhal changes, and these increase the tendency to ammoniacal fermentation of the urine. On account of the constant dribbling the neighbouring skin becomes excoriated, and the patient is exposed to constant distress from this cause, and from the odour of ammoniacal urine with which he is always surrounded. There is perpetual risk of ascending infection and pyelitis or pyelo-nephritis, and the mortality among subjects of the malformation is great; few reach adult life.

The treatment of extroversion of the bladder is, of course, surgical, and the chief forms of operation performed are two: (1) Plastic operations directed to repairing the cleft and forming a new bladder and urethra. Owing to the absence of a sphincter the artificial bladder is an inefficient reservoir, and dribbling is likely to continue. (2) Transplantation of the ureters into the rectum. This has been performed with success in a number of cases, and holds out the best prospect of relief. The risk of ascending infection is less than might be expected, and the rectum forms a fairly efficient reservoir for urine. It can evidently to some extent take on the function of the bladder, because after the usual morning evacuation of the bowels the patient may void urine, unmixed with feces, at intervals during the day.

Epispadias.—Apart from extroversion of the bladder, epispadias is a rare malformation. The cleft in the urethra may be glandular or penile. The penis is atrophied and curved back on itself. The amount of control of the bladder is variable. Epispadias is rare in the female.

Hypospadias.—Hypospadias, or cleft of the ventral wall of the urethra, is practically limited to the male sex. According to their position and dimensions, clefts of the ventral wall of the urethra are divided into (1) glandular, (2) penile and peno-scrotal, and (3) perineo-scrotal and perineal. In glandular hypospadias the urethra terminates in a pin-point opening at the base of the glands. The frenum and prepuce are sometimes absent. From this opening the urethra may extend forwards as a groove, or there may be a normal meatus leading into a blind canal. In peno-scrotal hypospadias the urethra opens at the base of the scrotum, and in perineo-scrotal or perineal hypospadias at the junction of the scrotum with the perineum. In the last variety the two halves of the scrotum are separated, and the penis is atrophied, giving rise to an appearance resembling the vulva.

The only treatment for epispadias and hypospadias is a plastic operation.

Ectopia of the Testis.—Ectopia of the testicle is often associated with hernia. It must not be confused with a movable testicle, in which the organ is sometimes at the opening of the inguinal canal, at others in the canal itself. An ectopic testicle cannot be drawn down into the scrotum, whereas it is easy to replace a movable testicle. The testicle normally reaches the scrotum by the eighth fetal month, but its descent may be delayed until infancy, childhood, or even puberty. Malposition is due either to its being checked in its descent (abdominal, inguinal, or inguino-scrotal), or to its taking up an altogether abnormal position (anal, perineal). An ectopic testicle is often functionless, and is said to be peculiarly liable to malignant tumour formation. It often produces hernia. In order to give the testicle a chance of descending, it is advisable to delay operation until about the twelfth year.

Atresia of the Vulva.—Atresia of the vulva is not uncommon in female infants. On separating the labia majora, instead of the hymen and vaginal orifice being exposed, a level area of mucous membrane, extending from the perineum to the urethra and clitoris, is seen; this is formed by union of the two labia minora. The adhesions between their edges are soft, and can easily be broken down with a probe, after which a pledget of wool smeared with vaseline should be inserted.

Prolapse of the Bladder.—Prolapse of the bladder sometimes occurs in little girls. It is said that it may be caused by repeated straining—e.g., from constipation or dyscolitis. The prolapsed bladder protrudes from between the labia as a dark red swelling, over which urine trickles. In some cases the protrusion is due to occlusion of the vesical end of the ureter, which becomes distended with urine, projects into the bladder, and then works its way out by the urethra. In this case the prolapsed mass will consist of a cystic tumour, and a palpable hydronephrotic kidney may be detected.

Prolapse of the Uterus.—This sometimes occurs as a congenital abnormality. The prolapse may be present at birth, or develop during the first few days of life. In most of the reported cases spina bifida has also been present.

Hydrocele.—Three forms of hydrocele occur in infants and young children—congenital hydrocele, infantile hydrocele, and hydrocele of the cord. In the first two forms there is a collection of fluid in the tunica vaginalis testis, giving rise to an elastic translucent swelling surrounding the testicle. In the congenital form there is persistence of the fetal communication between the tunica vaginalis and the peritoneum, while in the infantile form there is no such communication. A congenital hydrocele therefore disappears when the patient lies on his back, or can

be reduced by pressure. Reduction is effected without the characteristic gurgle of returning hernia, unless, as is not infrequent, the hydrocele is complicated by a hernia; if the communication is very small, reduction may be difficult. Congenital hydrocele tends to undergo spontaneous cure.

In hydrocele of the cord the upper part of the canal remains patent, and gives rise to a swelling in the inguinal canal, having the characters of a congenital hydrocele, but distinctly separated from the testicle. An encysted hydrocele of the cord consists of a small cystic tumour distinct from the tunica vaginalis, at or near the inguinal canal. It is movable to a limited extent, and is distinguished from hernia by the absence of gurgling, by its cystic character, and by the impossibility of reducing it. Both forms of hydrocele of the cord are rare.

TREATMENT.—Infantile hydrocele usually disappears spontaneously; if it persists, it may be aspirated and 10 to 20 minims of alcohol injected. In congenital hydrocele a trust may be worn in order to assist in obliterating the patent canal. If this fails, a radical cure should be performed. Like the infantile form, congenital hydrocele tends to undergo spontaneous cure. In hydrocele of the cord a radical cure may be performed, or the cyst aspirated and injected with alcohol.

Phimosis.—Phimosis, or undue narrowing of the orifice of the prepuce, is a common ailment. It must be distinguished from mere redundancy without narrowing of the orifice. A tight prepuce, however, may also be redundant. Normally the orifice should be wide enough to allow of the prepuce being retracted sufficiently to expose the entire glans; if this is impossible, a greater or less degree of phimosis is present. If the orifice of the prepuce is wide enough to allow of the urethral meatus being exposed, any phimosis which may exist is insufficient to cause difficulty in urination. In many cases, however, the opening of the prepuce is so small that the outflow of urine is definitely obstructed. Adhesions between the foreskin and glans are almost physiological, but usually disappear spontaneously. In many cases, however, whether the prepuce is normal or unduly tight, they persist and prevent complete retraction. These adhesions are soft, and can easily be broken down. A long prepuce, whether or not it is also tight, is liable to become sodden with urine, and redness, or even excoriation, around around the tip is common. If the prepuce cannot be retracted, either on account of phimosis or adhesions, accumulation of smegma is likely to occur, and hard gritty masses of whitish material gather in the sulcus at the base of the glans. When phimosis is so great as to obstruct the outflow of urine, the prepuce may balloon out during micturition, and phosphatic deposits form in the preputial sac.

It must not, however, be supposed that these results always follow phimosis. In young adults one sometimes finds a preputial orifice so small as scarcely to admit a probe, and hears that the patient has never been able to pass a full stream of urine; yet, when circumcision is performed, the glans appears perfectly healthy.

In infants and young children phimosis may give rise to pain on micturition, and simulate the symptoms of stone, or the straining which it necessitates may lead to the production of hernia or prolapse of the rectum. In some cases the obstruction to the outflow of urine may cause dilatation of the ureters or hydro-nephrosis (vide p. 638). The local irritation of a tight prepuce is apt to excite emotions, and may induce the habit of masturbation. Various reflex disturbances, such as limping, so as to simulate hip disease, paralysis, and contractures in the lower extremities, are said to have been occasionally caused by phimosis, but

this is doubtful. Other nervous disturbances, such as insomnia or enuresis, may occur.

TREATMENT.—The object of treatment is to bring about such a state of matters that the whole glans penis can be freely exposed. Unless this can be done cleanliness is impossible. If complete retraction is prevented by adhesions, these should be broken down with a probe or director, and any accumulated smegma removed. If the prepuce is only moderately narrowed, the opening may be dilated with a pair of forceps; this is painful, and usually leads to a good deal of abrasion of the part, which requires to be dressed with an antiseptic ointment in order to prevent irritation during micturition. When there is much narrowing, circumcision is required. The operation is also desirable if the prepuce is very long, although not tight.

Balanitis; Balano-Posthitis.—Inflammation of the glans or of the inner surface of the foreskin is not uncommon in young children. It is usually due to lack of cleanliness, and is most liable to occur when the foreskin cannot be completely retracted. The parts are congested and slightly swollen, and the normal secretion is increased, flakes of soft, curd-like smegma, with sometimes an offensive odour, being present. The condition yields rapidly to simple measures—bathing with boracic lotion, and applying a dusting powder of *ac. bor.* 1 part, *pulv. amyli* 9 parts. Circumcision is often advisable to prevent recurrence.

Paraphimosis.—Paraphimosis is a not uncommon accident in little boys. When a tight foreskin is retracted, the orifice may constrict the penis just behind the corona glandis. The swelling of the glans, which prevents the foreskin being pulled forwards again, gives a characteristic appearance—redness and oedema of the end of the penis, bounded by two sulci, the distal being at the reflexion of the prepuce and corona, and the proximal at the orifice of the prepuce. If the condition is neglected, spontaneous cure nearly always takes place by ulceration and yielding of the constriction; strangulation of the glans is not common.

TREATMENT.—Manual reduction is usually possible, and in performing it the essential point is to diminish the size of the oedematous glans by steady pressure before attempting to pull the constricting prepuce forward. Pressure may be applied by the hand or by a few turns of elastic band. If the paraphimosis cannot be relieved thus, the constriction should be notched in several places. Circumcision should be performed to prevent recurrence.

ORCHITIS—EPIDIDYMITIS.

Acute inflammation of the testicle is rare in children. It may be due to trauma, to specific urethritis, or to mumps. The last-named of these is the most important; the body of the testicle is involved, and atrophy of the gland may result. Orchitis secondary to mumps is, however, practically unknown in boys below fourteen or fifteen years of age, but as adolescence approaches the liability increases. Mumps should therefore be looked on as a grave serious matter in boys at or after the age of puberty than in young children, and the patient ought to be kept in bed for a week or a fortnight. The treatment of acute orchitis in children is the same as in the adult.

Chronic or subacute orchitis or epididymitis may be due to tubercle or to syphilis.

Though rare, tubercle of the testicle is more common in infants under two years than in older children. It often develops suddenly, and gives rise to a large subacute swelling, involving the body and epididymis; in other cases the process is more chronic, resembling that in the adult. Both testicles may be affected. Tuberculosis of the testis most frequently occurs as a primary affection, or it may be secondary to tuberculosis of the urinary tract. In addition to attention to the general health, surgical treatment may be called for, but removal of the organ is inadvisable in the early stages of the disease, and should only be practised if the whole gland has become disorganized.

In congenital syphilis interstitial orchitis and epididymitis are not uncommon, and sometimes give rise to definite, usually bilateral, enlargement of the organs.

VULVO-VAGINITIS.

A catarrhal inflammation of the mucous membrane of the vulva and vagina is not at all uncommon in little girls. Probably the cervix uteri and urethra are also occasionally affected. In most cases the patients are over two years of age, but infants also suffer.

Clinically we have to distinguish between four forms of this disease: (1) Simple catarrhal vulvo-vaginitis; (2) gonorrhoeal vulvo-vaginitis; (3) diptheritic vulvo-vaginitis; and (4) herpetic or aphthous vulvo-vaginitis. The two latter are relatively rare, and from a practical point of view most cases of purulent vulvo-vaginitis are due to the gonococcus.

Ætiology.—Lack of cleanliness and bad hygienic surroundings are among the most important causes of vulvo-vaginitis, which is therefore much more common among the poorer section of the community than among the well-to-do classes. It is more liable to occur in debilitated children than in the robust, and is said to be especially common after measles. In cases of gonococcal vulvo-vaginitis the source of infection cannot always be traced. It may, however, be accepted as a fact that attempted sexual intercourse is rare indeed in comparison with infection in other ways. It is probable that in most cases the mother, or some other person in the house, suffering from a gonorrhoeal discharge, conveys the infection accidentally to the child. An infant may be infected with gonococcal vulvo-vaginitis at birth, but this is rare. Gonococcal vulvo-vaginitis is easily communicable from one child to another by direct contact or through the agency of sponges, towels, etc. Many epidemics of this kind have been reported in institutions, especially in New York. Holt believes that the spread of the disease in institutions, though often caused by nuptials, is not due to this alone, but that a regular house infection may develop. In Hamilton's series of 344 cases, 87 patients contracted the disease while in an institution, and 93 were infected by leucorrhoeal discharges of mothers or sisters.

Among other causes of vulvo-vaginitis are trauma, attempted rape apart from gonorrhoeal infection, and auto-infection by scratching in cases of local irritation of the skin or of threadworms. Masturbation may be a cause, but, when the two are associated, it is probably more often a result, of the vulvitis.

SYMPTOMATOLOGY.—As a rule the first symptom noticed, and that for which advice is sought, is vaginal discharge. There may be some discomfort in walking, evinced by a straddling gait, or pain on micturition, but these are comparatively

infrequent. On local examination, in a mild case, there is merely redness of the vulva, with a few crusts where the edges of the labia come in contact, and a slight mucopurulent discharge. In more serious cases pus is abundant, and small excoriations round the orifice of the vagina, or even larger superficial ulcers of the vulva, may be present. The discharge is usually greenish-yellow, and may be tinged with blood. Urethritis, and still more cystitis, are rare. The Bartholinian glands are rudimentary in children, and very seldom become inflamed. The great majority of cases of vulvo-vaginitis, accompanied by a definitely purulent discharge, are due to the gonococcus. This infection tends to run a chronic course, and in its later stages there may be nothing more than a discharge of mucus, so slight as not to soil the linen. An exact diagnosis, therefore, can be made only by examining stained smears. The gonococcus, if present, is usually found easily, and as a rule exists alone. In non-gonococcal cases there is generally a great variety of organisms in the smears.

Complications.—Gonorrhoeal vulvo-vaginitis may be complicated by conjunctivitis (from direct infection), by arthritis, or by gonococcal septicæmia, endocarditis, etc., but none of these is common. Practically, the arthritis is the most important of these, because it may be mistaken for rheumatism. The occurrence of joint swelling in a child under three years should always suggest examination of the vulva; in older children the failure of salicylates to relieve the pain and swelling may give the first clue to the nature of the case. In the 273 epidemic cases reported by Holt, arthritis was not uncommon in boys, apart from any urethral affection; he believes that in these cases infection had entered by the mouth. Extension to the uterus, tubes, and peritonæum, is extremely rare. In Hamilton's series of 344 cases, arthritis was met with thrice, conjunctivitis four times, and enlargement of the inguinal glands twice.

Prognosis.—Cases of simple vulvo-vaginitis usually yield rapidly to treatment. Gonococcal vulvo-vaginitis is intractable, and may persist for many months. A cure is not to be looked for in less than six or eight weeks.

Treatment.—In simple vaginitis all that is necessary is to use irrigations of boric lotion or weak permanganate of potash three or four times a day, and, if there is much excretion, to leave a pledget of wool smeared with a mild antiseptic ointment (e.g., ammoniated mercury, 5 grains to 1 ounce) between the labia. In gonococcal cases, irrigations of boric acid lotion, or Condy's Fluid, or zinc sulphate ($\frac{1}{2}$ grain to 1 ounce), may be used. A pledget of wool should be kept between the labia, and a napkin worn. In view of the great difficulty of curing gonococcal vulvo-vaginitis by local applications, vaccines ought to be tried. The writer has little experience of them in this disease, but in Hamilton's hands they appear to have been remarkably successful. Out of 84 cases, 74 were cured by the use of a stock vaccine of gonococcus, whereas out of 290 patients treated by irrigation only 158 were cured. No less striking was the shortening of the period of treatment. To obtain a cure by local means alone, ten months on an average was needed; in the cases treated with vaccines, only one month. Hamilton advises five or six injections at intervals of about five days, beginning with 50,000,000, and increasing by 10,000,000 at each dose (vide also *Proteogenes* III., p. 26).

Before a patient can be said to be cured, gonococci must be absent from three successive smears from the vagina, taken at intervals of a week.

Diphtheritic Pseudo-Vaginitis.—Five or six cases of vulvo-vaginitis due to the

B. diphtheriæ have come under the writer's notice. The following were the appearances in a severe case in a child aged fourteen months: "Both the labia are swollen and bluish-red in colour, forming together a mass about the size of a plum. Their inner surfaces are lined with a greyish-yellow membrane, which also covers the labia minora and the vaginal orifice. There is also a small patch, about the size of a pea, on the outside of the vulva. The vulva has been noticed to be red for a week, but the membrane has been present only one day." In all the cases there was a distinct membrane, the appearance of which at once suggested diphtheria. The patients were infants, of about a year old or less. In none of them was there diphtheria of the throat, and none looked seriously ill. No source of infection was discovered in any case, nor did any case transmit diphtheria.

INFLAMMATION.—Membranous exudation on the vulva is always due to the *B. diphtheriæ*, which should be demonstrated and identified by culture.

TREATMENT.—In addition to local measures, antitoxin should be given.

Herpetic (Aphthous) Vulvo-Vaginitis.—One occasionally meets with cases of vulvitis in which there are one or two yellowish-white erosions on the labia minora or around the urethral orifice, surrounded by an angry zone of redness, and a little swelling of the parts. The erosions are very like what are seen on the lips and tongue in aphthous stomatitis, hence the name. In this form of vulvitis, which is most common in infants, there is usually pain and screaming during micturition. It should be treated by cleanliness, and the local application of a mild antiseptic detergent to protect the excoriations.

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HELT: *New York Med. Journ.*, 1905, lxxxv, 524, 569.

Noma.—Noma of the vulva closely resembles noma of the cheek (*cancrem oris*), but is much more rarely met with. It is due to the same causes as *cancrem oris*, and, like that disease, may arise spontaneously or be preceded by an aphthous or ulcerative condition of the parts. It consists in a gangrenous inflammation of the labia, which spreads rapidly and is accompanied by great prostration. The disease is quite as fatal as *cancrem oris*, and requires the same treatment (*cf.* Chapter IV, p. 169).

CHAPTER XIII

FUNCTIONAL DISEASES OF THE NERVOUS SYSTEM

LEONARD GUTHRIE

INTRODUCTION.

CLINICAL DIVISION OF FUNCTIONAL NEUROSES:

A. SOMATIC NEUROSES.

B. PSYCHO-SOMATIC OR SOMATO-PSYCHIC NEUROSES.

THE NEUROtic TEMPERAMENT.

EVOLUTION OF MENTAL FACULTIES AND SPECIAL SERIES.

EVOLUTION OF EMOTIONS.

RELATIONSHIP OF MENTAL FACULTIES TO EMOTIONS.

THE RÔLE OF THE SYMPATHETIC IN FUNCTIONAL NEUROSES.

THE CORTICO-THALAMIC SITE IN FUNCTIONAL NEUROSES.

TYPES OF NEUROtic TEMPERAMENT.

A. Neuroses of somatic origin:

1. Somatic functional neuroses in early infancy:

Tetany.

Laryngismus.

Eclampsia.

2. Functional co-ordination neuroses:

Nystagmus.

Head-rolling.

Congenital stidor.

3. Neuroses due to faulty development of the cerebral centres:

Aphasia.

Congenital word-blindness and word-deafness.

B. Neuroses of psycho-somatic or somato-psychic origin:

1. Disorders of sleep.

2. Dreamy mental states.

3. Tics and habit-spasms.

4. Convulsions and epilepsy.

5. Headache and migraine.

6. Koroisis.

7. Hysteria.

8. Neurotic halits.

9. Delayed walking.

10. Stammering and other speech defects.

INTRODUCTION.

Nervous disorders which are not due to destructive lesions of any part of the nervous system are called "functional." The existence of any permanent structural damage is disproved by the complete, rapid, and even sudden, recovery which may occur. Functional nervous disorders, however, may have a pathological basis. Nearly all organic diseases of the nervous system are attended by symptoms which cannot be produced by the actual lesion itself. In a severe case of poliomyelitis, for instance, a limb or limbs may at first be completely paralyzed, but in time all that may remain is weakness and wasting of a single muscle group, innervated by a minute collection of anterior cornual cells. We cannot assume that the initial widely-spread paralysis in such cases is functional, in the sense of having no morbid anatomical changes as its cause, merely because it rapidly subsides. More probably a generalized toxæmia has affected vast numbers of cornual cells at first, but the damage has only been sufficient to destroy a few. Recovery from pathological conditions is doubtless possible, provided that the lesions do not exceed a certain degree of extent and severity. The initial fleeting paralyses of disseminated sclerosis are examples of such recoveries.

If complete and rapid recovery be the criterion of functional nervous disorders, we must include amongst them paralyses due to lead, alcohol, and diphtheria. Yet these complaints are rightly regarded as forms of toxic neuritis; whereas tetany, although equally attributable to toxic influences, is included amongst functional neuroses. Chorea until within recent years was regarded as a functional neurosis, but now is considered a manifestation of rheumatic toxæmia. Laryngospasms and convulsions are no doubt, like tetany, dependent on the presence of toxins of undetermined nature. Profound neurasthenia may result from sepsis. Auto-intoxication of various kinds accounts for phenomena, such as delirium, spasms, tremors, eclampsia, besides many disorders of metabolism, nutrition, secretion, and absorption, which are, strictly speaking, functional neuroses.

We cannot, therefore, assert that a functional neurosis is unattended by physical and anatomical changes in the nervous system, because the symptoms come and go quickly, and because recovery may be complete and lasting.

Even the fact that a "functional neurosis" may be caused by suggestion and cured by persuasion does not exclude the possibility that material though temporary, changes in the nervous system may attend the presence and the disappearance of symptoms.

We can only speculate on the nature of such changes. They may be chemical or cytological, or both. Definite cytological changes have been found in the nerve cells of pigeons and honey-bees as the result of fatigue, and nervous exhaustion in human beings may be attended by similar changes.

As all nervous and vital functions are dependent on blood-supply of the organs concerned, and as the circulation is governed by the vasomotor system, the part played by the sympathetic in the ætiology of functional neuroses must be important.

Characteristics of Functional Nervous Disorders.—Functional nervous disorders are more easily recognized than described. They are generally characterized by infinite variety of symptoms, by Proteus-like changes, by a certain histrionic exaggeration which conveys a misconception of their nature to the physician, and by

the dramatic or tragic manner of their appearance and disappearance. A clue to their origin is often afforded by peculiar traits of disposition and temperament in the individual who manifests them.

A functional disorder may simulate the symptoms of organic disease affecting any or every part of the nervous system, but the portrait is always "Good! very like! not altogether so!" The picture is overdrawn, over-elaborate, and is at once detected as a copy by the connoisseur.

A *functional motor paraplegia* is too complete to be compatible with the degree of muscular tone, nutrition, and condition of tendon responses found on examination of the limbs. Anesthesia or other sensory disturbance does not correspond to the distribution of cutaneous nerves or to that produced by disease of spinal segments. Tremors and myoclonus, "tics" and convulsions as "functional" disorders, all show to the initiated minor points of differentiation from similar symptoms resulting from organic disease.

Hysterical contractures and *hysterical flaccid paralysis* are usually more extreme and complete than those produced by destructive lesions of the motor tracts. Relaxation of the spasm under an anesthetic in the one case, and the detection of muscular tone and of slight voluntary contractions of the muscles when the patient is off his guard in the other, reveal the functional nature of the affection.

Inconsistency is a marked feature in the symptoms of hysteria. The patient, for example, who cannot adduct his vocal cords for speaking purposes can yet give vent to an explosive cough.

Hysterical amblyopia, even when the patient professes inability to distinguish light from dark, can be distinguished from true blindness by the presence of the pupillary reflex and of healthy condition of the fundus.

Hysterical contraction of the visual fields as revealed by the perimeter is equal in all directions, roughly circular, and tends to increase under examination until vision is at last confined almost to the fixation point. It may be thus distinguished from the toxic amblyopia in which vision at the fixation point itself is chiefly at fault.

The presence of *true hemianopia* as shown on a chart is only roughly imitated by *hysterical crossed amblyopia*, in which there is extreme contraction on the hemianesthetic side, with slighter limitation on the other, combined in many cases with a complete or partial colour blindness.

Weakness of convergence is common, but *paralysis of ocular muscles never occurs in hysteria*. *Hysterical ptosis* is really spasm of the orbicularis muscles, and not due to paralysis of the levator palpebre, such as is seen in myasthenia gravis or disease of the third nerve or of its nuclei.

True nystagmus is not a sign of hysteria, though nystagmoid jerking, especially on lateral deviation of the eyes, is common.

A few only of the more common objective signs of functional or hysterical nervous affections have been mentioned, merely to show that their prevailing characteristics are exaggeration, inconsistency with each other, incompatibility with the classical signs of organic disease, which they merely parody. Similar characteristics mark the enormous variety of subjective symptoms, the disturbances of organic functions, and the mental conditions which are associated with functional neuroses.

CLINICAL DIVISION OF FUNCTIONAL NEUROSES.

Functional neuroses may be classified as (A) Somatic, (B) Somato-Psychic or Psycho-Somatic.

A. Somatic Functional Neuroses are those in which a psychic element may be excluded. A purely somatic origin may be assumed in infants under one year, and less certainly under two years of age.

1. Examples of somatic functional neuroses in early infancy are tetany, laryngismus, carpopedal contractions, eclampsia, salivary spasm, epilepsy. In all these affections some definite somatic disease is present, usually in the shape of auto-intoxication, causing local or generalized instability and explosiveness of nervous elements.

2. Temporary or permanent fault in exercise or development of co-ordination accounts for such affections as nystagmus, head-nodding (spasmus nutans), some cases of stertor, congenital stridor, dysphagia. Some would include under this heading such diseases as spasm of the pylorus (hypertrophic pyloric stenosis) and of the cardiac sphincter; and even hypertrophy of the bladder associated with hydronephrosis and dilatation of the ureters, and idiopathic dilatation of the colon, have been attributed to inco-ordination in the action of the sphincters and detrusors of hollow viscera.

3. In yet another class of cases there seems to be faulty development of cerebral centres, especially of the visual and auditory cortical areas. Congenital word blindness and deafness, colour blindness, idioglossia, may thus be accounted for.

B. Psycho-Somatic and Somato-Psychic Neuroses are those in which a psychic or mental element is present in addition to a morbid physical or bodily condition. It is often difficult to decide whether the psychic or the somatic cause is predominant. Any local or general cause of physical ill-health may give rise to functional neuroses, such as tic or habit spasm, stammering, neuritis, migraine, night terrors, swooning, eclampsia, or even epilepsy, which may therefore be regarded as somato-psychical.

The difficulty in accepting all views implying a purely somatic origin of functional nervous disorders is that the supposed causes may exist without producing the nervous disorders, and that the disorders may occur in the absence of the supposed cause. Chorea, for instance, is a manifestation of rheumatism, but the most severe cases of chorea are usually those in which rheumatic manifestations are slightest, and many children suffer severely from rheumatism, yet never develop chorea. An indigestible meal will give rise to nightmare in any child, but the most aggravated form of night terrors may occur without evidence of indigestion or any other physical cause. Spasmodic asthma, again, in some cases is attributable directly to certain articles of food, or to certain atmospheric conditions, or to local causes of irritation in the air-passages, yet in many, no such causes can be ascertained.

Functional nervous disorders include, besides various spasmodic affections, disturbances of circulation, heat regulation, secretion, absorption, metabolism, and nutrition, all of which may be due to definite organic disease, yet evidence of such is often slight or absent; whilst, except in very early infancy, all functional disorders, motor or otherwise, may seem to be the consequence of psychic, mental, or emotional conditions alone.

These considerations lead to the conclusion that purely somatic or physical conditions are not alone sufficient to account for all functional nervous disorders. Other factors must be present, and these consist in some peculiar and anomalous conditions of the cerebro-spinal and sympathetic nervous systems, which render the individual unduly sensitive and responsive in reaction to stimuli derived from his environment and from his internal self.

This peculiarity in reception, storage of impressions, and reaction to stimuli, constitutes the neurotic or emotional temperament.

The writer is not disposed to agree with the modern doctrine that functional nervous disorders, such as neurasthenia, "psychasthenia" (so called), and hysteria, are entirely psychic in origin. He prefers to regard them as psychosomatic or somato-psychic, believing that every symptom has its basis in a physical or bodily change, however slight such changes may be, and that sensations due to such changes may be misinterpreted or distorted by the imagination and mental condition of the individual who experiences them.

It is difficult to accept the views of certain modern psychologists who maintain that functional nervous disorders in general are affections of the mind, and not of the brain or body; that the mind and its faculties, such as "will," perception, attention, emotions, desires, and "conative" processes in general, are entities distinct and separate from all material things, and that the mind is not a function of the brain nor the brain the organ of the mind.

If such views be accepted, we may cast aside all the knowledge which for years and years has been slowly and patiently amassed by neurologists and physiologists as to the nervous functions in general, and their localization in different areas of the nervous system.

THE NEUROTIC TEMPERAMENT.

All nervous tissues are sensitive, or respond in some way to various kinds of stimulation. Vitality, including reflex action, consciousness, mental and emotional capacities, depends on the degree of nervous irritability or responsiveness to stimulation which prevails in the individual. The neurons receive and store impressions derived from stimulation, and give out energy, but no man knows the nature of these processes nor of nervous energy itself.

From the moment of birth irritant stimuli pour incessantly upon our brains through the medium of the organs of special and common sensations. Temperament results from the mode in which stimuli are perceived, responded to in the form of motor reactions, emotional and otherwise, and from the capacity of storing experiences of past stimuli, recalling them to consciousness, comparing them, and formulating ideas, judgments, and conduct based thereon.

The neurotic temperament is characterized by hyperesthesia and unduly active response to all forms of stimuli, both physical and mental. Hyperesthesia on the physical side is shown in exaggerated reflexes and tendon jerks; in the readiness with which spasmodic affections—tremors, ties, spasms, and convulsions—are induced by trivial causes; in abnormal severity of the symptoms attending any ordinary ailment, and the ease with which cardio-respiratory and digestive functions—secretory, excretory, absorptive, and metabolic—are impaired.

Hyperesthesia is shown in cutaneous irritability, itching, discomfort, and frequent appearance of eruptions of erythematous, urticarial, and eczematous type.

It affects also the special senses. The neurotic child often shows marked dislike or intolerance of certain colours, sounds, tastes, and odours, and anything which he dislikes usually violently disagrees with him. He is also sensitive as a barometer to changes of temperature and climate.

Emotional Hyperæsthesia.—Emotions, like instincts, are involuntary motor responses to sensations, percepts, and ideas. Without the involuntary motor response or expression, the intensity, and even the existence, of an emotion cannot be gauged or surmised by an observer. Some maintain that it is the expression that gives rise to an emotion, and not the emotion which causes the expression.

Darwin said: "Most of our emotions are so closely connected with their expression that they hardly exist if the body remains passive."

Professor James and Lange trace emotions to sensations derived from the activities of certain muscles (voluntary and involuntary) and glands (sudorific, lachrymal, intestinal, etc.). Under a stimulus, pleasurable or otherwise, voluntary muscles are thrown into activity, and the characteristic attitudes expressive of emotion are involuntarily assumed. The stimulus also excites activity in involuntary muscles, giving rise to certain circulatory changes, such as increased or diminished frequency of the pulse, local flushings, and pallor, and also to increased or diminished activity of sudorific, lachrymal, intestinal, and other glands. The various sensations produced by attitudes, gestures, circulatory, secretory, and other changes, give rise to the different kinds of emotions.

The neurotic and emotional individual is the one in whom such physical changes are most readily induced by insignificant internal or external stimuli. Both sensitiveness and reaction to stimuli are exaggerated.

Control of Emotions.—Although neurotic persons are said to be lacking in self-control, the emotions are not under direct control by the will. We can work up an emotion by assuming the attitude which characterizes it, as every actor knows, and we can partly counteract an emotion by assuming a contrary attitude. For instance, we can combat a desire to laugh by putting on an air of preternatural solemnity. But although the outward expression of an emotion may be voluntarily suppressed, and the emotion itself dulled by such suppression, it is impossible to stifle the effects of a stimulus or shock to the nervous mechanism of circulation, respiration, and secretion. We cannot prevent our eyes from filling, our face from flushing or becoming pale, our heart from throbbing, our respiratory rhythm from altering, our mouth from watering, or our flesh from creeping at a sight or sound or thought. All these conditions are essential, whereas the characteristic attitudes are only adjuncts to the expression of our emotion. Hence, persons whose emotions are most readily excited by internal disturbances may show them least by outward manifestations.

EVOLUTION OF MENTAL FACULTIES AND SPECIAL SENSES.

It is perhaps necessary to support the bald statement that mental processes are reactions to stimuli by a brief reference to the evolution of psychology and of the conscious personality of the individual.

The primitive instincts of the new-born babe are reactions to stimuli of the simplest nature. It sucks if anything is placed in its mouth, cries and struggles if hungry, hurt, or uncomfortable. At first it reacts only to unpleasant or painful

sensory stimuli. For the first few weeks of life, response to pleasurable sensations is negative.

Taste.—In regard to appreciation and reaction to stimuli of special sense organs, according to Kussmaul, the sense of taste is congenital. Sweet substances excite movements of sucking; sour or bitter produce grimaces.

Hearing.—Newly-born infants are deaf for at least twenty-four hours, and may remain so for several days. The deafness is attributed to swelling of the tympanic mucous membrane and absence of air in the tympanic cavity. The swelling subsides, and air enters the middle ear as a rule within a few days of birth.

The fibres of the auditory tract leading from the nervous cochlearia to the first tetrapetal gyrus can be traced at the end of the second month.

Sight, so far as perception of light is concerned, is congenital. Bright sunlight causes evident discomfort during the first few weeks. It is said that as early as the sixth day an infant may follow a light in the room with its eyes, and even turn its head for that purpose. But as a rule it is not until the third month that co-ordination of ocular movements is established, and often not until the sixth month that infants seem to recognize objects and persons seen.

The infant, therefore, at birth and for the first few weeks of its life reacts only to the crude stimuli. Yet on its reaction to these depends its future mental existence.

The origin of consciousness is in the simplest sensations, and by them alone all the higher faculties of the mind may be developed. Helen Keller, rendered blind and deaf in infancy, would have been an idiot throughout her life, but talents and intellect of the highest order were cultivated in her, solely through the medium of her sense of touch. Her case is as important to psychology as that of Alexis St. Martin is to the physiology of gastric digestion.

An infant permanently incapable of perceiving, registering, and reacting to, stimulation of its special sense nerves, and of nerves of common sensation, both superficial and deep, must necessarily be mindless. Fortunately, when one portal is closed, others are open to stimuli; otherwise the deaf-mute or congenitally blind person would also be an idiot or an imbecile. Defective appreciation of stimuli through one afferent or ingoing channel may be compensated by increased sensitiveness to stimuli passing through another.

THE EVOLUTION OF EMOTIONS.

The infant possesses instincts from birth, but emotions, strictly speaking, are not developed until after the first few months of existence. The new-born infant screams instinctively if uncomfortable from any cause, but does not weep. Although lachrymal secretion is present, tears are not shed as a rule before the age of two or three months. As mentioned already, the sense of taste is congenital; but at first it is crude, only applying to substances too hot or too cold, sour or bitter, which may be ejected from the mouth with grimaces expressive of disgust. As a rule the infant will swallow anything—castor-oil, for instance—however nauseating it may be. At the age of four or five months its taste becomes more discriminating. Anger and resentment are shown by struggling, wriggling, arching the back, and by a peculiar cry, whenever food is disliked or any personal discomfort is experienced. In fact, the emotions of anger and resentment are developed earlier

than others. The baby sleeps when fed and comfortable, but shows no signs of pleasure. It is not, as a rule, until four or five months of age that it delights its parents with the tribute of a smile expressive of contentment.

By degrees more complex emotions than those of mere pleasure and its reverse make their appearance. But all are primarily reactions or motor responses to stimuli which are pleasant or unpleasant to the individual. In time, when the ingoing paths of sensation and the outgoing motor paths become developed by use, and the centres in the brain become stored with myriads of impressions or perceptions derived through the organs of special and common sensation, the complex and varied emotions which make up conscious personality are elaborated.

Estimation of the Physical Concomitants of Emotions—"Feeling Tone."—Instruments of precision have been used to estimate the physical concomitants of the simplest emotions, pleasurable and otherwise, which, as already mentioned, are the foundation of all others. The *plethysmograph* shows that a positive or pleasant tone of feeling is accompanied by an increase of bodily volume (dilatation of arterioles), and that a negative or unpleasant tone is attended by a decrease of bodily volume. The *sphygmograph* reveals acceleration or retardation of the pulse in association with positive or negative tones of feeling. The *pneumograph* shows increase or decrease in depth of respiration in pleasant or unpleasant tones of feeling. The *dynamometer* shows that increase of muscular power accompanies a positive, and decrease a negative, tone of feeling.

On the other hand, it seems certain that the physical concomitants of emotion may exist in consequence of irritative stimuli, without giving rise to the emotions themselves. Darwin said long ago that he could not refrain from starting back as if in fear when a caged snake struck at him, although he knew that thick plate-glass protected him from the reptile. Many of us experience shuddering, tingling all over, and "goose-skin," on reading a thrilling ghost story, although we have not the slightest belief that it is true. Similar sensations to those of fear are induced in some by the sound of a saw being sharpened, or of a pencil squeaking on a slate, of a cork being cut, or even by certain forms of music. Perhaps the most typical example of the occurrence of physical concomitants of emotion without the emotion itself is seen in *exophthalmic goitre*. Here, as Hector Mackenzie pointed out many years ago, the aspect of the patient is that of extreme fear or horror, which is quite out of accord with the emotional condition present.

In some cases, however, the physical concomitants of emotion seem to give rise to a spurious emotion, which the patient regards as genuine, although perfectly aware that the stimulus is inadequate to produce it. For instance, a person experiences the abdominal sensations associated with melancholy, and complains of feeling sad and depressed without cause, or some petty stimulus excites in him the attitude and all the physical signs of anger, and he regrets his unreasonable irritability and loss of temper. Similarly may be explained the various "phobias" and obsessions of the neuroathenic, or the grandiose delusions of the general paralytic. The former has sensations usually associated with fear, and is therefore frightened; the latter is misled by the sensation of general *bien-être*, and becomes *megalomaniac*. In each case the physical concomitants of emotions are mistaken for the emotions themselves.

The ever-changing mental moods, phases, and fancies, of neurotic persons depend on exaggeration, and often misinterpretation, of a pleasant or unpleasant "feeling

tone." It will be observed that the "feeling tone," or physical concomitant, which is inseparable from the experience of an emotion, is intimately related to variations in the actions of the sympathetic nervous system.

RELATIONSHIP OF MENTAL FACULTIES TO EMOTIONS.

Although some psychologists maintain that will is an "entity," and exists apart from any somatic or physical change, it seems more correct to classify it with emotions.

Will is a desire, more or less imperious, to perform or not to perform a given action. It may be logical or illogical, and if the latter we call it "obstinacy." If will be an entity, obstinacy must be so too.

The will, like other emotions, cannot be exercised without pre-existing bodily change in the shape of attitude and expression. A person lies in bed in the morning limp, flaccid, and loath to get up, although he knows he must. He cannot do so until he has roused his will by stretching himself, contracting his muscles, and "pulling himself together." Or he meets with sudden calamity, and is found cowed, dejected, huddled up in his chair, unable to face his lot. A hearty friend enters, claps him on the back, and adjures him to cheer up. At once he starts to his feet, squares his shoulders, and becomes the man of will and energy that he was. It is the "air of determination" which excites the will. We do not expect an exercise of will from the man with wandering, vacant eyes, who shifts nervously from foot to foot in face of an emergency. The man who cannot make up his mind is either physically flabby, molasse-like, so as to be practically emotionless, or he is conscious of so many conflicting stimuli raining into him at once from present circumstances, recollection of past experiences inspiring caution, and consideration of consequences, that none is sufficient to excite the physical condition without which definite action is impossible.

Will, then, like all other emotions, has a physical basis, and its exercise depends on the intensity of the stimulus. If the stimulus, whether derived from a thought, a circumstance, or a sensation, is sufficiently strong to induce a physical attitude or expression, and also certain circulatory changes, an action or exercise of will is the consequence. Weakness of will may be due to multiplicity and inefficiency of stimuli.

Weakness of will-power and impulsiveness are not synonymous. The impulsive person acts, as we say, on the spur of the moment. A stimulus which excites but a passing whim in one person excites inordinate desire clamouring for immediate gratification in another, whom we call "impulsive." Stimuli derived from storage of past experience or memory, which should inspire in him the attitude of caution and reflection, are unheeded. He cannot balance "pros" and "cons"; the "pros" decide immediate action. This inattention to counteracting stimuli, or "restriction of personal consciousness," as Janet calls it, is responsible for most of the phenomena of hysteria, whereas hyper-sensitiveness to stimuli and undue reaction thereto explains many of the symptoms of neurasthenia. In both complaints, but especially in neurasthenia, there is a tendency to exhaustion, local or general, of nerve elements as the result of over-stimulation.

Attention and Concentration.—The importance of the faculty of attention needs emphasis. Attention is no more an "entity" than is "will," in the writer's

opinion. It is an attitude voluntarily assumed in order to favour the reception of stimuli, as every drill-sergeant and every teacher is aware. Unless the attitude of attention becomes a habit, perception is blunted, reception and storage of impressions is impossible.

Attention, however, which favours reception of particular stimuli cannot be maintained unless other distracting stimuli are ignored, and the power of ignoring distracting stimuli is called "concentration." A child cannot maintain its attitude of attention during a geography lesson if the stimulus of a fly buzzing on the window-pane distracts it. A student cannot "take a lecture in" unless he "takes it down." Note-taking prevents mind-wandering. An extreme degree of attention or concentration is the boon of the philosophical and the bane of the hysterical mind. Disregard of all but one particular set of stimuli enables the philosopher to solve the problem, and gives rise to *Fidèle* *foir* in the hysterical.

It is through attention that facts, events, and knowledge of all kinds, becomes "photographically lined on the tablets of our mind." Under-exposure blurs the image and causes loss of memory.

Memory is the faculty by which past experiences are recalled. Its exercise implies repetition of stimuli which act as keys to locked-up storerooms. The master-key may be a visual, auditory, olfactory, gustatory, or cutaneous stimulus. Sometimes it can only be exercised through the process called "association of ideas," by which the stimulus which arouses a sensation and reaction in the shape of an idea evokes others, arbitrarily or logically as the case may be. Attitudes, or the sensations induced thereby, are aids to memory just as they are aids to the experience of an emotion.

Imagination is the faculty by which ideas derived from a stimulus to one set of sense organs excite other ideas. The sight of a cabbage, for instance, excites in the unimaginative person nothing more than a passing idea that the dinner-hour is approaching, whereas in the imaginative it may call up a picture of "She-beans, apple-pies, barbers' shops, a death, a wedding, and the great Panjandrum himself."

An inconsequent and incoherent train of ideas follows a single stimulus, and it is this odd and unexpected association of the ideas which constitutes "originality," just as a shake of the kaleidoscope produces a new combination of heterogeneous materials and a new picture.

Synesthesia.—Imagination is akin to the process called "synesthesia," or involuntary association of a subjective sensory impression affecting one sense with an actual sensation belonging to another sense. "Colour hearing" and "sound seeing" are examples in point. Sounds, whether of the human voice or of musical instruments, produce definite colour sensations and peculiar hallucinations of vision. Synesthesia is not confined to visual and auditory senses. According to Parish, particular tastes or smells, sensations of temperature, and even the exercise of muscular sense, arouse sensations of colour in some people.

Another form of synesthesia is shown in galvanic stimulation of the skin, which produces cutaneous sensations, and also hallucinations of taste, smell, and vision.

Synesthesia, in the form of "colour hearing" or excitation of one special sense by stimulation of another, is a physiological and not necessarily morbid peculiarity, but in one form or another it probably accounts for many of the fears, obsessions, fixed ideas, and hallucinations, of hysteria and insanity. It is probably a factor in the indelible association of ideas which, formed in early life in response to

some stimulus or other, creates in us prejudice, likes and dislikes dominating the course of our existence and career.

The writer has expressed frankly materialistic views as to the nature and origin of instincts, emotions, and, indeed, of all mental faculties. Our personal consciousness, or mental existence, is based upon, and elaborated from, experience of stimuli perceived, received, impressed upon, and recalled by, our brain, through the medium of common and special sense. Such views do not commit us to any vitalistic theory. The mode of action of the stimuli, and the nature of the changes which they produce in living nervous structures, are left undetermined. Some changes must take place, but we do not know how far they are chemical, molecular, vibratory, or whether they consist in association or dissociation of neurones in response to mechanical or other forms of irritation. We are bound to assume the existence of nerve force or "nervous energy," but we do not know what it is. Synonyms for nervous energy have abounded in medical philosophy throughout all ages. Thus, we have the "entemon" or "impetum facies" of Hippocrates; the "animal spirits" of the Galenic School; the "sucus nervosus" of Borelli; the "archei" or "ferments" of Van Helmont; the "anima" of Stahl; the "vis nervosa" of Haller; the "materia vitæ diffusa" of John Hunter; and quite recently the school of Ferri, Oscar Vogt, and McDougall, have attributed all mental and nervous processes to the action of a mysterious force called "neurin" or "neurokyme," for which, finally, Hale White has suggested the name "leptor-rheuma."

All attempts to define and describe nervous energy have hitherto failed. No advantage is gained by inventing new names for it. The inevitable result has always been that a pure hypothetical abstraction becomes regarded as a physical entity endowed with various material attributes and subject to mechanical laws. Aloud systems of treatment have always followed conjectures as to the nature and modes of exertion of nervous energy, by whatever name it may be called. At present we can only, like Locke, "sit down in quiet ignorance of those things which, upon examination, are found to be beyond the reach of our capacities"—and one of these things is nervous energy.

At the same time it is not right to ignore such knowledge as we possess with regard to nervous action in general. We are at least justified in saying that nerve tissues receive, store, and react to, stimuli, mechanical or otherwise, and that on these properties all our nervous functions and mental processes depend. The views of modern psychologists who regard mental processes, such as "will," "attention," etc., as "entities," or primitive faculties having no physical basis, seem to be untenable when one considers that bodily and circulatory changes are essential for their exercise. On the assumption that all physiological, mental, and nervous processes normally depend on reception, perception of stimuli of various kinds, on motor reactions thereto, and on storage of perceptions and the power of subsequently recalling the same under stimulation, various anomalies and abnormalities of nervous and mental processes may be explained. Perception of stimuli may be abnormally acute, and motor response excessive. Such hyperæsthesia may be on the part of the nerves; of the afferent sensory tracts; or of the reception centres; or of the efferent or motor apparatus. Direct evidence of excessive irritability of accessible nerves is forthcoming in tetany. Cutaneous hyperæsthesia and hyperalgesia point to irritability of the sensory nerves and tracts, and perhaps of higher receptive centres in the thalamus, cortex or elsewhere. Excessive motor reactions

to stimuli are seen in various spasmodic affections, such as the tics, chorea, myoclonus, laryngismus, convulsions, and epilepsy. The tics, chorea, and perhaps myoclonus, may be regarded as manifestations of psychomotor irritability. Convulsions and epilepsy are usually ascribed to "explosiveness" on the part of storage centres in the cortex, either due to direct irritation or to some inherent instability. Abnormal irritability of the sympathetic system accounts for excess of emotional display, and for many functional neuroses attributable to sensation produced by its action on the circulation, glandular function, and metabolism in general.

Another wide class of functional neuroses is attributable to hypo-anesthesia and deficient reaction to stimuli, and to imperfect power of recalling past stimuli. Stimuli may be unfelt, and motor reaction may be absent owing to anaesthesia of cutaneous and deep sensory nerves, or to absence of conduction through the afferent sensory tracts, or to inappreciation of stimuli by the cerebral receptive centres. Hysterical anaesthesia and paralysis are explicable on the assumption that peripheral stimuli do not reach the brain, and do not cause reactions, and that the power of recalling past stimuli is temporarily lost. In hysteria the capacity of feeling and expressing emotion in general is in abeyance, but one particular emotion or idea or desire holds sway. Disturbance of the sympathetic is a prominent feature in hysteria. Ischaemia and anaesthesia go together. Local ischaemia of various areas will account for practically all hysterical phenomena.

The explanation of all "miraculous" cures is that some form of stimulus or sudden shock has opened the floodgates of the sympathetic, which hitherto has remained uninfluenced by ordinary impressions.

THE ROLE OF THE SYMPATHETIC IN FUNCTIONAL NEUROSES.

Irritability, hyperaesthesia, and abnormal excess of reaction to stimuli on the part of the sympathetic nervous system, explain most of the phenomena of functional nervous disorders. There is hardly a symptom or physical sign of hysteria or anaesthesia which is not associated with disturbance of the sympathetic nerves and centres governing circulation, glandular secretion, and activity of unstriated muscles.

Just as the emotions are inseparable from vasomotor changes—either affecting the vaso-dilator or vaso-constrictor fibres of the sympathetic—so also the various functional neuroses may be dependent on ischaemia or the reverse of that condition. Blood is life, and any portion or structure in the body may be rendered anaesthetic and functionless by local ischaemia. The paralyzed and anaesthetic hysterical limb does not bleed when pricked, but blood flows freely the instant that power and sensation are restored in consequence of some sudden shock which opens up the sluice-gates of the circulation. It is difficult to dissociate anaesthesia and paralysis in such cases from the ischaemia which prevails, and it seems legitimate to infer that bloodlessness similar to that which is demonstrable in parts seen may exist in those unseen. Local cerebral anaemia or hyperaemia consequent on vasomotor disturbance affords a ready explanation of symptoms such as migraine, headache, giddiness, tinnitus, deafness, restriction of visual fields, vertigo, dream states, somnambulism, trance, syncope, and convulsions, besides other disorders of many of the higher faculties and special senses, including those of taste and smell.

Irritability of the sympathetic accounts for tachycardia, palpitation, asthma,

and dyspnoea of other kinds not due to organic disease. The abdominal and gastrointestinal affections which may be attributable to abnormal states of the sympathetic splanchnic areas include, not only a host of subjective sensations of discomfort connected with emotions, but such complaints as gastric atony, enterospasm, nervous colic, lenteric diarrhoea, enteropneosis, orthostatic albuminuria, polyuria, and exuresis, gastro-stasis andreno-stasis. Abnormal pulsation of the abdominal aorta commonly accompanies these symptoms and conditions.

Of local manifestations of sympathetic disturbance, mention may be made of abnormal blushing, flushing, and pallor, various forms of erythema exudativum, urticaria, dermatographia, pigmentation of the skin, "dead fingers," and also paræsthesia.

The objection usually raised to the vasomotor theory in the case of cerebral and pulmonary functional disorders is that the cerebral and pulmonary vessels are ill supplied with sympathetic fibres. But the cerebral and pulmonary arterioles contain unstriped muscular tissue which is contractile. Loss of tone or heightened tone is demonstrable in accessible cerebral arteries, and must affect their terminals. Moreover, it has been shown that the secretion of the posterior part of the pituitary gland causes contraction of the arterioles in general, but particularly of those in the brain. As regards pulmonary neuroses, although the vessels are ill-endowed with vasomotor nerves, the unstriped muscles of the bronchioles are richly innervated, and varying degrees of contraction in the latter may account for asthma as well as other kinds of functional dyspnoea.

In fine, not only the emotional and mental characteristics of the neurotic temperament itself, but the physical signs and symptoms of disorders commonly associated with it, afford evidence that the sympathetic is at fault.

Influence of Glandular Secretions on the Production of Functional Neuroses.—As already mentioned, the activity of the lachrymal, salivary, odorific, and intestinal glands is inseparable from the experience of emotions, and such glandular activity or inactivity is dependent on the circulation, which, again, is governed by stimuli acting on the sympathetic.

It is possible, also, that the secretions of ductless glands may have considerable influence in generating emotions, seeing that they act by stimulating the sympathetic. This is the case with the pituitary and suprarenal glands, which have a vaso-constrictor action, whilst the thyroid exerts a vaso-dilator influence. In hyper-thyroidism (exophthalmic goitre) we see all the physical signs of intense emotional shock or fright. In hypo-thyroidism (myxoedema) we find the slow pulse, abnormal temperature, apathy and lethargy, associated with a purely unemotional existence, which again implies sympathetic inactivity.

It is possible, therefore, that the occasional success of treatment of functional neuroses by organic extracts may bear a physical interpretation.

THE CORTICO-THALAMIC SITE OF FUNCTIONAL NEUROSES.

Considerable light is thrown on the phenomena of hysteria, neurasthenia, and other functional neuroses, by recent investigations of the symptoms produced by lesions of the optic thalamus and its cortical connections.

The cortex cerebri is the highest and latest development of the nervous system. Its functions are receptive, reminiscent, critical, initiative, but above all inhibitive

of the reactions of lower centres to stimuli both physical and mental. It regulates and controls reflex responses to stimuli of any kind, and particularly the more primitive reactions of the optic thalamus to sensations which reach it.

The "Syndrome Thalamique."—Déjerine, Ronssy, and others, have shown that a lesion of the optic thalamus may produce the following characteristic changes:

1. A persistent loss of superficial sensation of one half of the body and face. The loss to touch, pain, and temperature, is more or less definite, but the loss to deep sensibility is always more pronounced.

2. Slight hemi-ataxia and more or less complete astereognosis.

3. Acute pain on the same side, persistent, paroxysmal, often intolerable, and yielding to no analgesic treatment.

4. Slight hemiplegia, which produces no contractures and rapidly passes off.

5. Chorea and athetotic movements in the limbs of the affected side.

Ronssy pointed out that the sensory loss and the pain are alone due to the lesion of the optic thalamus, whilst the other symptoms are produced by destruction of adjacent parts.

Henry Head and Gordon Holmes have confirmed these observations as to the results of lesions of the thalamus or surrounding parts, but they have also shown that certain characteristic conditions are associated with lesions of the lateral nucleus of the thalamus, which interrupt the cortico-thalamic paths, and thus leave the intrinsic organ of the thalamus itself uncontrolled by cortical influence. In such conditions of uncontrolled thalamic activity they have found a tendency to react *excessively to stimuli both unpleasant and pleasant*, especially the former. The prick of a pin, painful pressure, excessive heat and cold, all produce more distress on the affected than on the normal side of the body. Moreover, stimuli (thermal, for instance) which produce only mildly pleasurable sensations on the sound side induce extravagant expressions of delight when applied to the affected side. This "excess of feeling tone" accompanies perception of stimuli affecting superficial and deep sensibility, visceral sensibility, and also is evoked by reactions to purely mental and emotional states. Head and Gordon Holmes record interesting illustrations of the heightened feeling tone which prevails on the abnormal half of the body in such cases. In some, certain sounds, such as pathetic and solemn music, induced intolerable discomfort, or even pain, together with contraction of the limbs and increased athetosis on the affected side of the body. One patient spoke of "amorous impulses," a "craving for sympathy and consolation confined to the right side," and added, "My right hand seems to be more artistic."

The manifestations of this increased susceptibility to states of pleasure and pain are strictly unilateral in lesions of the lateral nucleus of the thalamus. In all cases the thalamic response to stimuli, although excessive in degree, is delayed in time of onset; it is also prolonged in duration. The importance of the discovery that an excess of "feeling tone" in respect to affective stimuli is pathognomonic of lesions which leave the optic thalamus uncontrolled by cortical influence cannot be gainsaid. It enables us to understand the emotional and physical hyperaesthesia which prevails in hysteria and neurasthenia. We can no longer dismiss the excruciating pains and discomfort of which neurasthenic and hysterical patients complain as purely imaginary or psychic; they are evidence of excess of feeling tone induced by thalamic disorder. Excess of thalamic activity explains some of the motor manifestations of hysteria—the tremors, cramps, and convulsions, and also the corybantic orgies of "dancing dervishes," "revivalists," and victims of

epidemic chorea major. But functional neuroses may imply, not only excess, but also defect, of functional activity. In some cases of hysterical paralysis it would seem that the thalamus, always slow to rouse, becomes abnormally sluggish in reaction. A cure depends on our ability to supply stimuli sufficient to excite a "feeling tone" pleasurable or otherwise.

The thalamus may be regarded as the seat of emotions, for the expression of emotions depends on its integrity. Evidence of this is seen in certain cases of thalamic disease. Mimetic capacity is lost; the patient cannot smile or weep on the affected side of the face, although volitional movements are unimpaired. Absence of thalamic reaction explains the extraordinary apathy and resignation of martyrs to hysteria, and the torpor which attends profound emotional shock.

There still remain other symptoms and conditions in hysteria which are attributable to uncontrolled action of heat regulation and vasomotor centres in the thalamus. With such an explanation at hand, we cannot pass over, as mere instances of pathomimia or malingering, the hyperpyrexia, erythematæ and various skin eruptions, the hæmorrhages from lungs, stomach, kidneys, and elsewhere, the phantom tumours, and enterospasms, which are of frequent occurrence in hysteria. Without doubt, pathomimia and malingering are prominent features of hysteria, yet the curiously significant remark of Head's patient who suffered from thalamic disease—"It is my right side which seems to crave for sympathy and consolation"—suggests that the similar craving of hysterical subjects may depend on thalamic disorder.

As previously maintained, hæmorrhages and all forms of erythema exudativum are attributable to disturbance of the sympathetic nervous system, and such disturbance may be the result of irregularity of action of the thalamic vasomotor centres when freed from cortical control.

We may conclude that hysterical and functional neuroses are traceable to disorder of cortico-thalamic activities. The site of disturbance may be in the afferent sensory cortical tracts, thus depriving the cortex of stimuli which should guide it in perceiving, appreciating, and reacting to, peripheral sensations—hence loss of posture sense, astereognosis, atopognosis, which suggest to the patient paralysis of limbs. It may involve various cortical areas, thus causing amnesia of past mental and physical impressions, or "restriction of personal consciousness" in regard to present and past experience. If, again, the cortico-thalamic paths are blocked, cortical restraint is abolished and thalamic reactions run riot; "excess of feeling tone" is attached to every stimulus which reaches the organ. Finally, we may assume in some cases hyper-function, and in others hypo-function of primitive thalamic activities in order to explain excess of "feeling tone" and its absence in different hysterical phases.

TYPES OF NEUROTIC TEMPERAMENT.

Although subdivisions are endless, there are two main types of neurotic temperament:

1. In Persons of the **Unrestrained Type** there is super-sensitiveness and excessive reaction to all forms of stimuli. Their emotions are readily excited, and they have little control over their outward display. Their feelings are strong for the moment, but shallow, changing like the chameleon's colour. Some are extrav-

gantly affectionate at one moment, brutally callous, indifferent, the next; selfish beyond measure, craving for sympathy, regarding themselves aggrieved if every whim of the moment is not satisfied; goaded to fury by interference, and resentful of all discipline. They are high-spirited, impulsive, but easily discouraged; now enthusiastic and "castle-building," and now crushed and abased, worried by trivial causes, anticipating difficulties and making no effort to meet them. They are often timid, fickle, untrustworthy, imaginative, and superstitious. Their intelligence is often above the average; they are quick at learning, but forgetful of facts; seldom capable of prolonged industry, hating drudgery, but working with feverish energy in fits and starts, they are speedily exhausted bodily and mentally. They are continually seeking new forms of emotional and mental excitement, and unless provided with them become bored, morose, and hypochondriacal. They never seem to learn prudence, common-sense, and caution, from experience, and their actions are capricious, eccentric, and unreasonable. With all their faults, they are often personally attractive, ingratiating, witty, and "good company." Chances of success in after-life which make sober men's mouths water, are constantly offered them, but they throw them away out of pique and vanity, accuse their would-be benefactors of tyranny and unappreciation of their merits, because a little perseverance and attention to duty is expected of them. This class provides the "brilliant wasters" of the community, alcoholics, and narcotics. But a spark of ambition, and even brief periods of "taking pains," exalts them to the rank of genius of the artistic, poetic, and imaginative order.

Evidence of their unstable neurotic temperament is forthcoming in early infancy. As a rule they are brought up by hand, and the greatest difficulty is experienced in finding a diet which does not disagree with them. If breast-fed, similar troubles occur at weaning-time. Diarrhoea and vomiting, and even convulsions, follow every innovation in their food. The temperature rises on the slightest provocation, and a common cold makes them seriously ill. They sleep badly, wake screaming at any sound, and cannot be soothed to sleep again. Even before the age when babies begin to "take notice," they start, show signs of fear at a strange face or any novel experience, such as the sight of a professional nurse, with her voluminous cap and flowing ribbons.

In later childhood one is usually asked to see them because they are thin. They are pale and sallow, with dark circles round the eyes, dilated active pupils, and puffy lower eyelids, which are tremulous when closed. Their expression is animated, their colour changes rapidly; they talk fast and volubly, and ask strings of questions without waiting for an answer. Their attitude is restless and constantly changing; their fingers twitch and tremble. For days together they will be full of life and energy, and never seem to tire; then they collapse utterly in an attack of headache, vomiting, pyrexia, and so-called "gastric catarrh." Their appetite is capricious, and they have usually been subjected to a rigid diet excluding all things which ordinary children like. Yet it is true that they often show idiosyncrasies in the matter of food, such as fat, carbohydrates, and other common articles.

The chief complaints from which they suffer are attributable to an excitable, unstable, and easily exhausted nervous system. They are liable to repeated night terrors, somnambulism, headaches, migraine, various forms of tic and "bad habits," "phobias" and obsessions, asthma, cardiac palpitation, flushings, pallor, syncope, sweating, dead fingers, cold extremities and pooriness of circulation, eczema,

urticaria, and erythema exudativum in its varied forms—cyclic vomiting and cyclic or postural albuminuria, stercoris, and biliary diarrhoea, alternating with constipation, and mucous colitis. If rheumatic, they invariably develop chorea, severe out of all proportion to any cardiac or arthritic symptoms which may be present. At an early period of their existence they are usually branded as having "weak hearts," "weak lungs," or "weak digestions," or tendencies to consumption, and are treated with the utmost deference and consideration in view of such vague proclivities, until they become introspective, hypochondriacal, neurasthenic, psychasthenic or hysterical.

True idiopathic epilepsy is uncommon in this class, but under great emotional excitement or physical or mental exhaustion they may be subject to epileptiform seizures.

2. The Restrained Emotional and Receptive Type.—In this type, as in the first, there is super-sensitiveness to all forms of stimuli, but the reactions are suppressed. Emotions are very strongly felt, but the control of their outward display is equally strong.

The features are expressionless and apathetic, the attitude staccato or stooping; the gait is slouching, slow, and clumsy. Children of this type are often regarded as wanting in natural affection, but really yearn to be loved, brood over slights—imaginary or otherwise—and become gloomy, morose, solitary in habits, introspective and superstitious, gloating over, yet secretly frightened by, all that is uneasy and horrible. They harbour various kinds of "phobias," and sometimes develop abnormally conscientious scruples of a moral and religious nature—ponder over the reason of their existence, and entertain thoughts of suicide. Their apparently stolid indifference to their surroundings is varied by sudden fits of ungovernable rage or weeping. Some are observant, intelligent, but so reticent that they often pass for being stupid, sullen, and obstinate. They take all things seriously, and have little or no sense of humour. This disposition, with its characteristic suppression of outward expression of emotions, is as exhausting as that of the other type in which emotional excess is obvious, and is associated with many similar complaints.

The character of the suppressed emotion varies. It may be fear in one shape or another, or desire for love and sympathy, or ambition coupled with a sense of inferiority, and envy, hatred, and malice, towards others who succeed. In any case the suppressed emotion becomes an obsession, governing or excluding all stimuli-derived from experience and environment which normally should counteract it. The result is mental and physical exhaustion—dreamy mental states, somnambulism and automatism, psychasthenia, neurasthenia, and hysteria.

ÆTIOLOGY OF THE NEUROTIC TEMPERAMENT.—The neurotic temperament is partly inherent and inherited, and partly the result of environment. The neurotic child is most frequently the only one, or, what is practically the same thing, a long interval of years separates it from brothers and sisters. He may be the pet or the ugly duckling in the family, or on account of his delicacy he is treated differently from the rest of the family. One or both parents are often themselves neurotic or neuropathic, and the child, having no comparison of his own age, learns to imitate his parents, to share their worries and anxiety about himself, his ailments and pains, and the household cares in general.

Environment has more to do with the development of the neurotic tempera-

nent than heredity. Parents are not always to blame; the influence of nurses, governesses, teachers, and overbearing brothers and sisters, has to be taken into account.

As to exciting causes, any severe illness may yield crops of neuroses, though frequently they arise in children who are never very ill, but never very well. Rheumatism is generally held to be the chief cause of neuroses in mid-childhood, but the prominence of the neuroses does not coincide with the severity of the rheumatism. All neurotic children are liable to aches and pains, and, although caution is necessary in the matter, it is not advisable to regard and treat them indiscriminately as likely victims to heart disease. The suggestion of weak heart in a neurotic child who suffers from growing pains, flushing and pallor, palpitation and occasional fainting attacks, is readily assimilated by anxious parents, with results disastrous to the child's welfare.

Of other factors concerned in the production of functional neuroses, a sudden emotional shock may be the exciting cause, but more frequently they are the outcome of long periods of fear, dread, worry, anxiety, suppressed or not as the case may be, or of overpressure, a life of unhealthy excitement, an environment uncongential for any reason.

Errors of refraction, diseases of the naso-pharynx, and other local causes of peripheral irritation and ill-health, are rarely the sole causes of neuroses. In all neurotic children "great events from little causes spring." The super-sensitiveness and ready response to irritative stimuli of any description, which make up the neurotic temperament, have in all cases to be considered.

A. NEUROSES OF SOMATIC ORIGIN.

1. SOMATIC FUNCTIONAL NEUROSES IN EARLY INFANCY.

Tetany, Laryngospasm, Eclampsia, Irritability of Accessible Nerves (Chvostek's and Trousseau's Phenomena).—In infants under two years of age these affections are so frequently associated that they must be regarded as different manifestations of one and the same disease. They do not always occur simultaneously; either tetany or laryngospasm or eclampsia may be the first symptom to appear, but severe cases of tetany are usually followed sooner or later by one or both of the remaining affections. After completion of the second year, tetany commonly occurs alone. In one case of tetany, however, in a girl aged five years and eight months, laryngospasm caused such urgent dyspnoea that the need for tracheotomy was only averted by timely relaxation of the spasm. Primary tetany after two years of age is rare; the attacks are usually relapses.

AGE AND ONSET OF TETANY.—Of 15 patients sufficiently ill to be admitted to hospital, the youngest was four months, and another was under six months of age; 3 were in the first year; 5 in the second year; 3 between the second and third year; 1 was five and 1 was eight years old.

SEASONAL INCIDENCE.—Tetany is more prevalent in the winter and spring than in summer months. Eleven of fifteen severe cases occurred between November and April, and the remaining four in July, August, and September; three

of the latter were relapsing cases. In one only, an infant aged four months, the attack was in July, and was primary, associated with severe diarrhoea and vomiting.

Epidemics of tetany have been recorded in Vienna and Heidelberg, and in certain goitrous districts (Kashmir) it appears to be endemic.

SYMPTOMS OF TETANY.—Tetany is characterized by tonic, recurrent, or persistent spasms which affect chiefly the extremities, but may also involve the trunk and other parts, without loss of consciousness. The duration of the spasms is variable. They occur suddenly, and may last for several hours or even days together, with remissions. They are bilateral and symmetrical. Both upper and lower extremities are usually affected, but the latter may escape.

Carpo-pedal contractions are peculiar and characteristic of the affection, though varying slightly in detail. In the common form the fingers and thumb are



FIG. 67.—TETANY OF THE FEET, SHOWING CONTRACTION OF THE FLEXORS OF THE TOES.

approximated in the form of a cone, the fingers flexed at the metacarpo-phalangeal joints and extended at the distal joints; the thumb is inverted, its tip approximated in opposition to that of one of the fingers. Less commonly the tip of the thumb is inserted between the first and second fingers, or it may be flexed on the palm of the hand. The palmar arch is contracted transversely, the wrist is flexed, deviating usually to the ulnar side; the elbow is flexed, and the humerus adducted to the side.

The feet adopt a similar position; the toes are flexed at the proximal and extended at the distal joints, usually in a line with each other, but sometimes the great toe is hyper-extended; the concavity of the plantar arch is increased, the foot inverted. The heel is drawn up; the knee and hip may be flexed.

The dorsal surfaces of both hands and feet are often oedematous, bluish-red, and cold, and so exquisitely tender that the child screams with pain if handled or if it anticipates being handled. The contractions themselves may cause pain.

In mild cases, although the characteristic attitude may be preserved, pain and tenderness are absent, and the patient can overcome the contractions by an effort of will. In rare cases the tonic spasms spread to the trunk and neck muscles, causing opisthotonos and retraction of the head.

When the face is affected the expression becomes rigid, the brows wrinkled, the pupils may be fixed, and squint may occur. The lips are sometimes pursed, the upper protruding over the lower (carp mouth). Very rarely there is spasm of the vesical sphincter, causing retention of urine.

Carpopedal tonic spasms are the manifest symptoms of tetany, but there are also latent symptoms elicited only on investigation. These latent symptoms are known as *Chvostek's*, *Trousseau's*, and *Erb's* phenomena. They indicate excessive irritability of the nerve trunks. *Chvostek's* or the "facial phenomenon" illustrates this mechanical irritability of the facial nerve.

A light tap over the nerve trunk about midway between the zygomatic process and the angle of the mouth produces quick contractions of all the facial muscles supplied by the nerve. Similar momentary spasms of the orbicularis oculi or of the levator anguli oris may be produced by tapping the region of the external angular process or the corner of the mouth. The latter phenomenon should be distinguished from the normal sucking reflex produced in a similar manner in the lips of sleeping infants. Other peripheral nerves besides the facial show increased mechanical irritability. *Chvostek's* sign varies greatly in activity and constancy. It may be absent throughout a severe attack of tetany, and present persistently, or occasionally, long after all carpo-pedal contractions have ceased.

Trousseau's phenomenon is another manifestation of irritability of nerves. During the intervals between attacks of tetany, spasms of the hand may be produced by squeezing the arm above the elbow, and pressing on the nerves in the neighbourhood of the bicipital groove. The pressure should be sufficient to stop venous circulation, and one to several minutes usually elapse before the phenomenon appears. Its absence is of no significance, but its presence is a proof of tetany. The test sometimes causes pain.

Erb's phenomenon demonstrates an increase of faradic and galvanic irritability in the peripheral nervous system. The median nerve at the bend of the elbow has usually been selected for testing, and contractions of the muscles which the nerve supplies are induced by far weaker currents than are required in the case of normal children. Moreover, according to Mann and Thiernich, the anodal opening contractions are in excess of the closing contractions of a galvanic circuit. Erb found A.C.C. greater than K.C.C. The observations are interesting in showing the presence of electrical as well as mechanical irritability on the part of the



FIG. 68.—TETANY OF THE FACE.

Note the contraction of the zygomatic and also nasolabial ("carp") mouth.

peripheral nerves. Erb's test is, however, unnecessary for diagnostic purposes, and its application may be painful.

It is necessary to distinguish between the irritability of nerves in tetany and that of muscles which is shown in many wasting diseases. In the former, contraction of muscle groups is produced by percussing the nerve which supplies them; in the latter, percussion of individual muscles produces a momentary fibrillary twitching in them alone.

GENERAL SYMPTOMS.—Fretfulness, irritability, and sleeplessness on account of pain, are commonly observed in the height of an attack of tetany, but in some cases the carpo-pedal contractions appear to cause no discomfort. The temperature is commonly raised a little, but seldom exceeds 100° F. In fatal cases there is sometimes hyperpyrexia shortly before death. One cannot lay too great stress



FIG. 69.—FACIAL IRRITABILITY IN TETANY.

Chvostek's sign—viz., tapping the face with the index bone gives rise to an active contraction of the orbicularis palpebrarum, and frequently to other muscles of the face.



FIG. 70.—TETANY OF THE HAND, SHOWING FLEXION OF THE WRIST, FLEXION OF THE FINGERS AT THE METACARPAL-PHALANXIAL JOINT, AND EXTENSION AT THE METACARPAL JOINTS.

The thumb is adducted to the mid-line.

on the gastro-intestinal affections associated with tetany. Vomiting and diarrhea with loose, green, foul, and copious motions are practically always present.

Laryngospasm occurred in eight out of fifteen cases with more or less frequency and severity. The spasmodic attacks are characterized by sudden apnea, duskeness, and anxious expression, with sometimes slight twitching followed by a loud crowing inspiration. They are most frequent at night, but may happen by day, and are readily induced by crying or emotional excitement or sudden movements. Their duration is seldom more than a few seconds. When more lasting, they may terminate in a fit of general convulsions, probably asphyxial. In severe cases the spasm may spread to the diaphragm and remaining muscles of inspiration. A condition of expiratory apnea is produced, and may be fatal. As a rule, however, the attacks, though sufficiently alarming, end, like milder seizures, in a crowing inspiration, after which the breathing becomes normal.

In the case already mentioned of laryngospasm in a girl aged five years, an opportunity for laryngoscopic examination was afforded. The cords during the spasm were seen to lie in the mid-line, very slight abduction occurring occasionally. The glottis suddenly opened and closed again, the opening being due, apparently, to abduction of the left vocal cord, the right remaining in mid-position.

Eclampsia.—It is unnecessary to describe here the fits in detail. They resemble infantile convulsions in general (p. 705). They may, as already mentioned, be asphyxial when following laryngospasm, but they also occur independently of spasm of the glottis, and are then, like the other phenomena of tetany, toxic in nature. In 15 cases of severe tetany, laryngospasm and convulsions occurred in 7; convulsions alone in 2; laryngospasm alone in 2. Both laryngospasm and convulsions were absent in 4.

ÆTIOLOGY OF TETANY.—Soltman endeavored to explain the convulsive disorders of infants, including those under consideration, by assuming that they were due to immaturity of the infant's nervous system. He thought that the development of irritability of peripheral nerves proceeded more rapidly than that of their inhibitory centres. The result of such deficient central control and increased excitability of peripheral nerves was, in his opinion, a nervous instability and tendency to spasmodic affections, which he termed "physiological spasmophilia." Were convulsive disorders due simply to physiological spasmophilia, few infants would escape them. Even the most trivial injury or irritation would cause convulsions in every child. Yet, clinically, we know that even severe injuries, such as burns and scalds, are not necessarily, or even usually, followed by any convulsive disorder. Moreover, Soltman's statements as to the difference in development of inhibitory centres and peripheral irritability are not in accordance with the experimental findings of other observers (Torchanoff, Lemonis, Panath). It is impossible to believe that spasmophilia is a normal physiological condition, as it is obviously inadequate to explain convulsive disorders which affect comparatively few, and not all infants alike. Hence, as the existence of spasmophilia or irritability of nerves in individuals cannot be denied, it has been attributed to a pathological predisposition to partial or general clonic and tonic convulsions. Finkelstein speaks of a "spasmophilic diathesis," Heubner of a "spasmophilic condition," in order to account for the phenomena. To describe spasmophilia, or irritability of the peripheral nerves, as a diathesis savours of bygone days. Were spasmophilia a diathesis, it would be extremely difficult to cure. Moreover, if due to a diathesis, this irritability of accessible nerves which constitutes spasmophilia should be a permanent condition, and not variable from time to time, generally ending in complete recovery. The fact that tetany, Chvostek's sign, and other symptoms of spasmophilia, come and go, is sufficient evidence that some special cause of nervous irritability is present at one time and absent at another. We can only attribute this variability in the symptoms of tetany to the action of some specific poison generated in, or introduced into, the body in varying amounts, and the actual source and origin of such a poison is most likely to be in the gastro-intestinal tract. For tetany, almost without exception, is associated with gastro-intestinal disturbances, and occurs almost exclusively in infants and children who are artificially and improperly fed. It is true that tetany has been described in breast-fed infants, but such instances are extremely rare, and in order to account for them one must assume either that their mothers' secretion of milk, or the infant's power to digest it, is defective.

Prolonged suckling may have some share in the production of tetany, for three

cent of fifteen patients were at the least for twelve months, and one for eleven months. Seven of the fifteen were entirely bottle-fed from birth. The remaining four were suckled for three, four, five, and six months respectively, and subsequently were fed on cow's milk and barley-water, "petted" milks, and various patent foods, until they reached the age of fifteen to eighteen months, when children of the poor share their parents' meals.

Rickets as a Cause of Tetany.—Some have maintained that tetany and other convulsive disorders are merely the manifestations of rickets in the nervous system.

It is an undoubted fact that many of the subjects of tetany are also rickety. Nine out of fifteen cases were markedly so, but two of the remainder were under six months of age, when rickets can hardly have declared itself, and the others were not rickety at all. Also, the most pronounced type of rickets is often unassociated with spasms of any kind. The theory advanced by Kassowitz, that the pericranial thickening present in advanced rickets induces corresponding hyperemia of the cortex of the brain beneath, and so causes tetany and convulsions, cannot be accepted.

Yet there is no doubt that tetany, laryngismus, and convulsions, are most apt to occur in rickety children. The explanation is not that rickets is the cause, but the common gastro-intestinal disorders which accompany that disease. For, at all events, in early and primary cases of tetany the symptoms speedily disappear when gastro-intestinal disorders are treated, although the patient remains rickety as before. Improper food is the first factor in the causation of tetany, but it is impossible to say whether absence or presence of some constituent in the diet is responsible. Some cannot digest cow's milk in any shape, and others begin to thrive at once upon it. In general it may be said that excess of fermentable food, inferior in quality and badly prepared, is the usual cause of chronic gastro-intestinal disease in infancy and childhood, and that the consequence is the formation of intestinal toxins, probably bacterial in origin, which give rise to tetany. But no specific organisms nor toxin have been isolated as yet. In some cases excessive feeding alone, even when the diet is wholesome, will cause tetany, for the symptoms may speedily be removed by cutting down the diet.

Deficient Activity of Parathyroids and Disturbance of Calcium Metabolism.—A modern view as to the etiology of tetany is, first, that it is due to deficiency of calcium salts in the blood, and, second, that this deficiency depends on inactivity of the parathyroid bodies.

Sabatani found that electrical irritability of the cerebral cortex was increased when the calcium content was low, and decreased when it was high. Extirpation of the parathyroid glands in animals is followed by tetany, with lowering of calcium contents in the blood and increased output of the salt in the urine and feces (MacCallum and Voegelin). The tetany thus produced in animals is said to be immediately relieved by injection of a salt of calcium into the circulation. Hence it is concluded that the irritability of peripheral nerves in tetany is due to deficiency in metabolism of calcium salts, and that this deficiency depends on inadequacy of the parathyroids to fulfil their function. These alleged facts may be true as regards animals, but it is not certain that the tetany of children is akin to the tetany of parathyroidectomized animals. It is not certain that the calcium content of the blood is lowered in the tetany of human beings, or that the parathyroid bodies are in any way diseased, or that the symptoms are relieved by the administration of calcium salts. On the other hand, it is certain that (with the exception

of chronic cases, to which I shall presently refer) the symptoms of tetany are quickly relieved by giving castor-oil and bromide, and regulating diet without reference either to parathyroid activity or to the supposed deficiency of calcium content in the blood. It is quite possible that the calcium content may be lowered in cases of human tetany, but it is improbable that this is the cause of the symptoms.

The work of Parryth, Vincent and Jelly is strong evidence that the parathyroids do not possess independent functions, but that they and the thyroid are in reality one physiological apparatus. It is possible that extirpation of the glands lessens resistance to specific toxins present in the body at the time of the operation, or subsequently produced therein, and so gives rise indirectly to tetany, but evidence that the disease is due to thyroid or parathyroid insufficiency alone is not conclusive. The disease is probably caused by some specific toxin generated in an unhealthy or dilated intestinal tract.

Nervous inheritance seems to play but little part in predisposing to tetany. But Kassowitz noticed that some of the mothers of eclamptic children showed Chvostek's sign, and Thiemich collected twelve cases in which the mothers had suffered from laryngospasm and eclampsia in childhood, and "latent tetany" was present in several of their children.

The reason why tetany is more common in winter than in summer is unknown. Possibly confinement in a vitiated atmosphere indoors may in part explain this fact.

Acute and Chronic or Relapsing Tetany.—Both acute and chronic tetany are associated with gastro-intestinal disorder; but whereas acute tetany in overfed or ill-fed infants is readily curable, the reverse is the case in the chronic or relapsing form.

The writer does not agree with the statement that tetany only occurs in chronic intestinal disorders. It certainly may appear in previously healthy children after a few days' illness from vomiting, diarrhoea with copious offensive motions, and flatulent distension of the abdomen. Such cases are easily curable, at all events for the time being, and probably permanently, for after one or two attendances at the out-patients' department one sees them no more.

But there is little doubt that some—it is impossible to say how many—ultimately, if neglected, drift into the extremely fatal chronic or relapsing variety of tetany with dilatation of the stomach, or more frequently of the sigmoid flexure and large intestine.

It has long been known that tetany in adults is often associated with atony and dilatation of the stomach. Upwards of ninety such cases have been recorded, but the credit of drawing attention to the association in children of tetany and dilatation of the large intestine is due to Langmead. In the Clinical Society's Transactions for 1906-07, and Transactions of the Royal Society of Medicine for 1909, he collected nine cases, eight of them fatal, in which relapsing tetany, dilatation of the large intestine, unhealthy, offensive, copious, pultaceous, or porridge-like, green, frothy, and yeasty motions were associated. The ages of the patients were between two and a half and eight years.

TREATMENT OF TETANY.—The treatment of tetany consists in correcting obvious dietetic errors, and dealing with gastro-intestinal disorders which are their consequence. Sometimes excess of fluid or of fermentable patent foods causes tetany, in part, no doubt, by producing acute distension of the stomach. Lavage of the stomach is then indicated, though it should be remembered that in other

conditions this operation has induced tetany—probably because too much fluid has been used for the purpose.

Although diet is most important, it is difficult to lay down definite rules. For infants, good cow's milk, preferably citrated, serves in most cases, but in one the writer has noticed that all attempts to give it was followed by return of tetany, and the infant seemed only able to thrive on whey, cream, and albumin-water. In older children, suffering from tetany it is necessary to cut down starch, fatismaceous, saccharine, and fermenting articles of diet. The food should consist of finely minced chicken, fish, or meat, veal or chicken broth, whey, cream. A little dry toast may be given, but no vegetables or milk puddings. In infants, when vomiting is excessive, the stomach should be washed out, care being taken not to distend the organ with fluid. Bismuth with soda are the best drugs to give after a preliminary dose of calomel.

Diarrhoea, with loose, offensive motions, usually yields to castor-oil in 10- to 20-minim doses, made up in an emulsion, to which salol in 1- to 2-grain doses should be added. The writer has not been convinced of the value of calcium salts by personal experience, neither can he speak favourably of thyroid extract given with a view to supplement supposed thyroid inadequacy. It sometimes sets up violent diarrhoea and seems to aggravate tetany. But in some reported cases it has appeared to be of use. In the presence of laryngospasm and eclampsia, bromides may be added to the bismuth or castor-oil mixtures. The attacks of laryngospasm are best relieved by application to the front of the neck of sponges wrung out in hot water.

The treatment of dilatation of the colon associated with tetany is one of great difficulty. The subjects are usually emaciated and exhausted. The rational procedure is to wash out the stagnant, putrid, and fermenting contents of the bowel by means of copious irrigation. Boreic solution should be slowly and gently introduced into the bowel through a long tube, care being taken not to use force or to increase distension of the viscous. Such irrigation may be practised once or twice daily. By this means absorption of toxins may sometimes be prevented, and great, though often but temporary relief is afforded. Abdominal massage may also be of some service when these methods fail, and the colon is obviously permanently dilated and hypertrophied. Appendicostomy or excision of the bowel is the last resource.

2. FUNCTIONAL CO-ORDINATION NEUROSES.

Nystagmus, "Head-Nodding," Orycopasm, Spontaneous Ntans.—Nystagmus, or involuntary oscillations of the eyeballs, is usually associated in infancy with movements of the head. Excluding cases of nystagmus due to defective visual acuity resulting from microphthalmos, albinism, optic atrophy, cataract, choroido-retinitis, and keratitis, which are not associated with head movements, nystagmus, together with spasms nutans, is attributable to defective co-ordination between the action of the ocular and cervico-cranial muscles. The nervous mechanism at fault is probably that which regulates eye and head movements, and includes the semi-circular canals in its circuit. Ditter's nucleus (one of the red-nuclei) of the vestibular nerve situated in the external angle of the fourth ventricle and its connections with the oculo-motor nuclei beneath the corpora quadrigemina, and with the

anterior column of the spinal cord through the vestibulo-spinal tracts, are probably the seat of the disturbance.

Nystagmus with spasms nutans may be acquired in early infancy or be congenital and hereditary.

Acquired Nystagmus and Spasms Nutans are usually first noticed between the fourth and twelfth month. They occur most frequently in the winter months (December and January), and cease as a rule between the second and third year.

The character of the nystagmus may be horizontal, vertical, rotatory, oblique, and also convergent, as John Thomson has pointed out. It is usually binocular, but may be monocular. The head and eye movements usually occur together, but movements of each may precede or succeed those of the other. The nystagmus may increase or occur only when the head is stretched, and, when monocular, the head movements may cease when the affected eye is covered. The child often acquires the habit of looking sideways at objects as if it were hemianopic.

The movements of the head and eyes do not necessarily correspond; either horizontal nystagmus and head-nodding or vertical nystagmus and head-nodding may occur together. The movements are therefore not regarded as compensatory.

Exciting Causes.—A large proportion of patients, though not all, are rickety, and it is reasonable to suppose that rickets may interfere with the development of co-ordination. Randoltz was the first to draw attention to the occurrence of spasms nutans and nystagmus in dark houses and during the dark months of the year. Nystagmus has been attributed to darkness itself, or to eyestrain and fatigue of the retina owing to the child's attempts to fix a bright light in a dark room, and the head movements have been regarded as secondary to the nystagmus; but the conditions mentioned are not invariably present in all cases.

There is no reason for supposing that gastro-intestinal disorders or dentition are in any way responsible. Some of the children seem to be in perfect health at the onset of the movements, and health is not affected thereby.

Nervousness, and especially super-sensitiveness to noise, has been noted in some, and in these it is possible that irritability of the vestibular nuclei and semicircular canals may determine the head movements, and that the disturbance may extend to the vestibulo-motor nuclei, and so give rise to the nystagmus.

In a few instances nystagmus and head-nodding are associated with mental deficiency, especially Mongolian imbecility, and may be persistent; but in the majority the movements cease spontaneously, without special treatment, towards the close of first dentition. In a few cases relapses have been noted.

All that is necessary in the way of treatment is, in most cases, attention to general hygiene and provision of diet suitable to the ordinary child, and treatment of rickets if present. Antipyrin and bromides which have been recommended are unnecessary.

With regard to diagnosis, it need only be mentioned that nystagmus may be acquired in mid and late childhood in consequence of poli-encephalitis, hydrocephalus, cerebellar tumour, and Friedreich's disease, but such affections are considered elsewhere. Insular sclerosis is not a cause of nystagmus until early adult life is reached.

Congenital and Hereditary Nystagmus.—Nystagmus in these cases is observed as soon as the child begins to take notice or makes attempts at fixation of the eyeballs—i.e., in the third or fourth month. Unlike the transient form of nystagmus

already described, it persists through life. It is hereditary, and, like some other hereditary affections, is usually transmitted through the female to the male, but sometimes it is transmitted by either parent and affects both sexes (Nettelship). The nystagmus may or may not be associated with head movements. Intelligence and physical health are as a rule unaffected. Vision is almost invariably defective from ametropia (hypermetropic astigmatism). The irides are blue, the fundi pale, and the hair is fair, in most cases, and Nettelship regards the condition as a mild form of albinism. Nettelship has recently collected thirteen pedigrees of subjects of hereditary nystagmus. He divides them into two groups—(1) those with head movements and (2) those without head movements—and concludes that nystagmus and spasms sutans together are transmissible through either parent and to either sex, whereas nystagmus alone descends only through unaffected females to males.

The writer has, however, under observation a child aged three years, in whom nystagmus—horizontal and convergent—together with lateral movements of the head, were noticed at three months of age. His mother's sister's eldest boy is similarly affected, and also his maternal grandfather, who traces the condition to his own grandfather. The girls in this family escape, but their first male offspring are affected.

Congenital hereditary nystagmus tends to diminish in adult life, and treatment can only consist in improving visual acuity by means of suitable glasses.

“**Nystagmus Myoclonic.**”—Lenoble and Aubineau describe a form of congenital, familial, and hereditary nystagmus which they regard as an isolated myoclonus confined to ocular muscles. It occurs independently of any other ocular or auditive lesion, and is not necessarily associated with any mental or physical defect.

It is, however, in their opinion a minor and more-symptomatic variety of a group of myoclonus, including the “paramyoclonus of Friedreich,” Mervan's “fibrillary chorea,” certain “electrical choreas,” Gilles de la Tourette's “tic,” and Unverricht's “epileptic myoclonus,” in all of which nystagmus is exhibited, with stigmata of degeneracy and possibly alteration in intelligence. (See also Tics, p. 629).

“**Head-Rolling.**”—“Head-nodding” should be distinguished from “head-rolling,” which is a common habit in infants under two years of age. The back of the head is firmly pressed into the pillow, and steadily rubbed from side to side until the occiput may be worn bald. The movements only occur when the child is lying down. The subjects are often rickety. The probable explanation is that the head sweats, and contact with a hot and moist pillow causes itching, which is relieved by friction. A bran pillow, with a slight depression in the centre, should be provided instead of a soft and downy one. Inflammation of the ears is sometimes the cause, but in such cases fretfulness, irritability, and signs of pain, are present.

The movements are obviously voluntary, and so may be distinguished from spasms sutans (see p. 686).

In *Eclampsia Nervosa* sudden jerkings of the head to and fro or from side to side occur in series of fifty or more, and are accompanied by a dazed look or momentary unconsciousness. The colour sometimes changes, the pupils become dilated, the breath is held, or a few hurried, irregular respirations are drawn.

In *Salaam Spasms* the body is suddenly bowed forwards, and the hands are extended, palms downwards, in front of the face.

Eclampsia sutans and *salaam spasms* are forms of petit mal or epilepsy.

Defective co-ordination has been held to explain such affections as congenital laryngeal stridor, spasm of the pylorus (hypertrophic stenosis) or of the cardiac sphincter, causing dysphagia (Thomson).

Even hypertrophy of the bladder, associated with dilatation of the ureters and hydronephrosis, and idiopathic dilatation and hypertrophy of the colon, have been attributed to inco-ordination in the action of sphincters and detractors of hollow viscera.

Such explanations are not generally accepted, but they may be partially true.

Congenital Stridor is usually ascribed to a malformation of the epiglottis, which overhangs and obstructs the glottis, but the malformation may be secondary to spasmodic closure of the vocal cords, and caused by the child's efforts to inspire. In the writer's experience, two forms of congenital stridor may be distinguished—one is hoarse, low-pitched, and guttural, and due to obstruction by the epiglottis; the other is high-pitched and whistling, and depends on actual closure of the glottis. Normally the vocal cords separate in inspiration, but in cases of congenital stridor this co-ordination fails, and they remain closed.

The prognosis in congenital stridor is good. It ceases as a rule after the first year of life, which would not be the case were malformation of the glottis the sole cause (see also Chapter VI., p. 253).

Hypertrophic Pyloric Stenosis.—Normally the pyloric sphincter relaxes when the gastric detractors are in action, and in these cases it is supposed to remain obstinately closed whilst the stomach makes prodigious efforts, as shown by peristaltic waves, to drive its contents through.

The hypertrophy of the sphincter in this hypothesis is the result of spasm, but it is urged that the hypertrophy is congenital, and therefore unlikely to be produced by spasm *in utero*. Many therefore regard the hypertrophy as a developmental freak, and impute the symptoms to actual stenosis of the pyloric orifice. But Baiter has discovered post mortem that much hypertrophy of the sphincter may exist without clinical evidence of stenosis. Hence it would seem that pyloric spasm does play a part in producing the symptoms. Obviously, the spasm of a sphincter would be more forcible and effective when the muscle is hypertrophied than otherwise, and cases in which there is much hypertrophy would be the worst.

On the other hand, there appears to be another and less serious class of case in which hypertrophy only occurs in accordance with the degree and persistence of the spasm, and such cases may be those in which recovery takes place under medical treatment.

Miller and Wilcox have shown that hypertrophic pyloric stenosis has nothing to do with acid dyspepsia.

The cause of the spasm is unknown, except that it is in some way related to the food administered (see Chapter IV., p. 189).

Spasm of the Cardiac Sphincter.—Normally the act of swallowing opens the cardiac sphincter. Thomson explains certain cases of dysphagia in young children by supposing that co-ordination between the two processes is defective.

Spasm of the cardiac sphincter or lower part of the oesophagus is a troublesome and not uncommon affection in adults. Suddenly, in the act of swallowing cold aerated fluids, or a morsel of ill-masticated food, acute pain is felt beneath the sternum, and the sensation of a hard ball rising in the throat. After much effort flatus may be expelled or the morsel of food regurgitated, and the symptoms are

relieved, though they may recur on each attempt to swallow for a considerable time, and hysterical anorexia may be the consequence.

Little children are not infrequently brought to hospital with the history that they have suddenly declined to swallow, and scream at all attempts to make them. The difficulty is overcome by a little coaxing and persuasion to take food in small quantities, but it is possible that recollection of a painful spasm has caused the symptoms.

The dependence of congenital hypertrophy of the bladder, with dilatation of the ureters and hydronephrosis, upon inco-ordination between the sphincter and detrusor muscle, resulting in backward pressure, is hypothetically possible, but the presence of other malformations suggests a developmental rather than nervous origin for these conditions.

Congenital Idiopathic Dilatation and Hypertrophy of the Colon (*Hirschsprung's Disease*) is explicable by the hypothesis that inco-ordination exists between the sphincter ani and detrusor muscles of the bowel. Hypertrophy of the sphincter and resembling that of the pylorus in hypertrophic pyloric stenosis has sometimes been found. But such explanations are entirely hypothetical, though none more plausible has been suggested.

Other congenital defects of co-ordination of purely somatic origin are seen in the tremors and ataxy due to ill development of the cerebellum, or perhaps of the cerebello-spinal system. They are dealt with elsewhere (see also Chapter XIV, p. 286).

Acquired Co-ordination Neuroses.—These include stammering of the vocal organs, and certain cases of enuresis and retention of urine attributable to inco-ordination of the bladder and its sphincter.

They fall under the heading *Psycho-Somatic Neuroses*.

5. NEUROSES DUE TO FAULTY DEVELOPMENT OF CEREBRAL CENTRES.

Aphasia.—Children as a rule can say a few words at the end of the first year. At the eighteenth month they can name most common objects in one or two syllables, and can construct short sentences. At the age of three years their vocabulary has become much enlarged, and they should be able to talk fairly well. But some individuals are later in acquiring speech than others. In rare instances children, although obviously intelligent and healthy, may not utter articulate words until the fifth and even the seventh year, but express themselves by signs and gestures. They are not backward in other respects, and once they begin to talk they soon make up for lost time.

But in the majority of cases where no signs of speech appear by the age of eighteen months some mental defect should be suspected.

Backwardness in sitting, standing, walking, and in taking notice, is usually noted in such cases. But some imbeciles of the busy, restless type are unusually active on their legs, and interested for the moment in all they see. On this account their mental deficiency may be unsuspected, although at the age of three they can only make incoherent sounds in lieu of speech.

Partial Deafness may cause delayed speech and imperfect articulation of words. Absolute deafness, congenital or acquired, may give rise to deaf-mutism. In either

more than fifty per cent. of cases the deaf-mutism is acquired, and due to otitis media or (more rarely) to posterior basilar meningitis, or occasionally to poliomyelitis.

Congenital Deaf-Mutism is a fault in development of the centres of hearing or auditory apparatus, and is commonly a "familial" affection.

Congenital Word Deafness as a Cause of Mutism.—The term "congenital word deafness" is a misnomer, for, although in cases described as such there is failure to appreciate the significance of spoken words, the sense of hearing is otherwise unimpaired. This is shown by the fact that musical and other sounds are obviously perceived. At first the child appears to be a deaf-mute, taking no notice of spoken words and making no attempt to speak. But later it may be taught to repeat words and sentences by ear, parrot-wise, without attaching any meaning to them (*echo-lalia*). The defect is not in the auditory centre, but in the connection between it and the centres which interpret the sound of words heard, for words are repeated even when whispered.

Fortunately, the visual centres can usually be cultivated in such cases by instruction in lip reading. By this method, as in the case of deaf-mutes, the patient learns to estimate the mechanism employed in uttering words with their articulation.

Congenital Word Blindness.—It is convenient to consider this condition in association with so-called "congenital word deafness," for the two affections are analogous.

The congenitally word-deaf is not deaf, but fails to appreciate the meaning of word sounds; the congenitally word-blind is not blind, but is unable to recognize the significance of printed or written words and letters, and sometimes figures.

The defect in congenital word blindness is probably in the connection between the visual reception centres and the higher centres which interpret printed and written symbols, for the patient may be able to copy words, letters, and figures, although they are meaningless to him. Congenital word blindness varies in degree from mere slowness and difficulty in learning to read to complete alexia. Visual amnesia may account for milder cases, for there is less difficulty in recognizing Arabic numerals than Roman letters, probably because there are fewer of them to remember. But visual amnesia can hardly account for more complete cases of alexia, because in these the significance of symbols has never been grasped at all. One cannot be said to forget what one has never known.

Congenital word blindness may be mistaken for mental deficiency, or attributed to defective eyesight, but in genuine cases intelligence and visual acuity are normal. The difficulty is as great in reading the largest as the smallest type.

With perseverance and special training even the worst cases of congenital alexia are capable of considerable improvement.

The auditory centres may be trained to supplement the visual, just as in congenital word deafness the visual supplement the auditory, and in most cases of congenital alexia the auditory memory appears to be highly developed.

In the worst cases the meaning of letters, words, and numerals, may be learnt by tracing them in the air or on paper, or by use of the Braille raised type. Thus the muscular and tactile senses are requisitioned in order to overcome the defect.

Microcossia is probably due to defective development of the centres of hearing and their connections. It will be considered under Defects of Speech.

Loss of Acquired Speech may be due to deafness from various causes already mentioned. Temporary motor aphasia is not uncommon in severe cases of chorea, especially in the paralytic variety of that disorder. It may persist for many weeks, or even months, but recovery is invariable. Neurotic children may be temporarily stricken dumb by fright, and a child who has just learnt to talk may lose the power in consequence of some severe and prostrating illness, and be compelled to learn again. Aphasia due to gross cerebral disease (hemorrhage, occlusion of vessels, etc.) is rarely permanent in young children, because the speech centres on the right survive, and take on the function of those damaged on the left side. A syphilitic child aged between three and four, who was under the writer's care, became aphasic and hemiplegic on the right side. She recovered speech and use of limbs in a few months, but a year later, during an attack of pertussis, a paroxysm was followed by left-sided hemiplegia and aphasia, which remained permanent.

Mental Defects and Congenital Cerebellar Ataxia, due to mal-development of the cortex cerebri and cerebellum, are dealt with in Chapter XIV., p. 804.

B. SOMATO-PSYCHIC OR PSYCHO-SOMATIC FUNCTIONAL NEUROSES.

1. DISORDERS OF SLEEP.

Insomnia.—Normal infants up to the eighth or ninth month of age spend most of the twenty-four hours in sleep. Towards the end of the second year about twelve hours at night and from one and a half to two hours by day are given to sleep. From the second to the fourth year twelve hours at night and about an hour's sleep by day are sufficient. From the fourth year until puberty, nine to eleven hours by night is an average allowance. But some need far more sleep than others. The sluggish must "slumber again" if he is ever to be wide-awake and useful. A healthy child will obtain as much sleep as it requires if given opportunity and trained in regular habits. Otherwise, even a healthy infant may give trouble by keeping awake at inconvenient hours. The cause of recent insomnia in a previously healthy child may be indigestion from improper food or overfeeding, hunger, pain of any kind, irritating cutaneous disorders, an uncomfortable bed or night clothing, unhealthy condition of the house or bedroom, over-excitement, and febrile affections. Insomnia in neurotic infants may depend on similar causes, but the infants are specially susceptible to them. The neurotic infant is always a bad sleeper; he is with difficulty sent to sleep and readily awakened. He defies comforters, rockers, and lullabies, and enriches the vendors of elixirs, soothing syrups, and teething powders. The causes enumerated need only exist in miniature to deprive him of rest. Frequently they can hardly be said to exist at all; nervous irritability or general hyperæsthesia to stimuli is to blame.

Much depends upon firmness of management. The baby has to learn that the cot is his sleeping-place, and not his nurse's arms, but the cot should be made comfortable and attractive. He should become accustomed to slight noises, and to the presence of light, though not glaring light, in the sleeping-room.

As to position, most children can be trained to lie indifferently on the right or left side or on the back, but neurotic children often have individual peculiarities

in the matter which have to be respected. They adopt the attitude which suits them best. Many throughout life cannot sleep on the right side, although this position is favored by the majority. The reason is probably that the heart in neurasthenics, as Clifford Allbutt has pointed out, is, like other internal organs, badly made. It falls over to the right when the patient lies on that side, and causes a sense of weight and oppression which prevents sleep. Generally speaking, the attitude adopted is devised to supply warmth and support to the epigastrium. The child often lies semiprone on the left side, with his knees drawn up and hugging a pillow to his abdomen. This need for epigastric warmth and pressure in order to secure sleep is implied in a dictum of Hieronymus Cardanus, the sixteenth-century physician: "Always go to sleep with the hand on the pit of the stomach." Some peculiar attitudes during sleep are significant of cardiac or respiratory embarrassment. In other conditions, whether due to heart disease, naso-pharyngeal obstruction, asthma, or flatulent distension, the patient can only sleep when sitting up or with the head elevated on pillows. Restlessness and disturbed sleep are more common than actual insomnia in infants. In later childhood, cold feet and chronic indigestion, or mental and physical fatigue, may cause true insomnia. Fatigue promotes sleep in a healthy, but prevents it in a neurotic child.

Somnolency is not uncommon in active-minded children. Some whilst dropping off to sleep talk to themselves or their dolls, or go through imaginary games and lessons. Some will even answer questions and carry on conversation during sleep, but their utterances are irrelevant and "delphic," and they remember nothing of them next day. Parents should be warned against cultivating this faculty when it exists in their infant oracles.

Tooth Grinding occurs almost always during sleep. It rarely happens in waking hours except in imbeciles and in naughty children who practise it in order to annoy their elders. In imbeciles the teeth may become bevelled or worn flat by constant attrition. This habit during sleep is much more common in neurotic than in normal children, and is the result of dreams set up by any form of peripheral irritation, as in the case of dreams in general. Severe local pain in the shape of toothache or carache is probably not the cause, because, although tooth-grinders may be restless and wake screaming in night terrors, they do not as a rule wake complaining of toothache or any kind of pain. It is not a sign of pain in imbeciles, but a source of gratification. It is a common symptom in meningitis and other gross cerebral diseases during the periods of unconsciousness which precede the final stage of coma, but does not occur in intervals of consciousness, although headaches may be excruciating. Teeth-grinding is also a well-known symptom of pest in adults who sleep badly and suffer from night terrors, though not from toothache. Probably, therefore, teeth-grinding during sleep does not indicate local pain, but is a manifestation of a distressing dream in which anger or rage is the leading feature. For teeth-grinding is a primitive exhibition of these emotions. Its former significance is lost in imbeciles. Its occurrence in dreams may be stigmatic, for in dreams we are reduced in some respects to aborigines. Gastro-intestinal disorder is perhaps the most frequent cause, and the popular belief that teeth-grinding is a symptom of worms is occasionally correct.

Night Terrors and Nightmare.—The difference between night terrors and nightmare is only one of degree, and this depends on the temperament of the dreamer. Neurotic children suffer from night terrors, and normal children from nightmares. A stolid, unimaginative child may overeat himself and awake scream-

ing at night, but he is easily satisfied that "it is only a dream," goes to sleep again, and is none the worse next day. The neurotic child, however, is rendered temporarily insane by the vividness and horror of his hallucination, he may suffer severely from emotional shock at the time, and is sometimes haunted day and night by dread of its repetition. Typical night terrors are usually described in children between three and eight years. Older children may suffer from them, but one is comparatively seldom called to treat them on this account.

The attack occurs almost always within the first three hours of sleep. Except in cases of febrile disturbances, recurrent attacks in a single night are rare, but they may be repeated on many consecutive nights, or at weekly, monthly, or longer intervals. Piercing screams are heard, and the child is found sitting up in bed or crouching in a corner of the room with hands outstretched as if to shield himself. Sometimes he is trying to get out of the window or to open the door, and he may escape from the room and run screaming down the staircase. His face expresses wildest alarm, his eyes are widely opened with pupil dilated, and he gazes intently in the direction of the apparition which frightens him. He often gives a clue to its nature by shrieking "Black faces!" "Horrid man!" or some such exclamation. He may cling instinctively to anyone within reach, but does not at first look at or recognize persons. His attention is fully taken up by the imaginary object of his fears. The period of terror lasts from a few minutes to half an hour. The child presently recognizes his surroundings, but he sweats and trembles, turns pale, and seems utterly shocked and exhausted. He will beg not to be left alone in the dark again, and that his hand may be held. He soon dozes, but awakes and starts for a time before sleep becomes sound. Sometimes at the end of an attack he passes a large quantity of pale urine, but more frequently he has wetted the bed or the floor during the stage of excitement.

Next day there is usually some recollection of what has occurred, but is somewhat evanescent, even when the child has sprung out of bed, and may have answered questions more or less rationally, he has never been really awake, and he seems to remember nothing about it subsequently. Older children may seem to have forgotten their dreams, either because they are ashamed to speak about them or because they are too horribly complex for verbal description.

Silbermann drew a distinction between idiopathic and symptomatic night terrors. The former, he held, consisted in transitory visual hallucinations due to an abnormally excitable brain; the latter, in sensations of distress arising from digestive disturbance acting through the agency of the vagus nerve and giving rise to dyspnoea. But the distinction is too narrow. Visual hallucinations are certainly commonest in young children, but other hallucinations, such as those of hearing and common sensations, may occur in night terrors. Dyspnoea will certainly give rise to night terrors, but the dyspnoea may be due to other causes than indigestion.

It is probable that all dreams and night terrors are symptomatic in being reactions to dimly-felt bodily sensations or stimuli. They are idiopathic in the sense that the reactions to, or interpretations of the sensations which give rise to them are peculiar to the individual. Such idiopathy is represented in the nature of the dream itself and in its effect upon the dreamer. Exceptions may be taken to the view that all dreams are symptomatic on the ground that the nature of the dream, when it can be ascertained, seems to have little or no relation to the stimulus which is supposed to excite it. Why should indigestion, for instance, provoke visions of hideous faces, or of a gigantic toothbrush bristling with hostile intentions, instead

at sensations of mere pain or discomfort! Perhaps the explanation is that, in the semi-consciousness which obtains in dreams, a peripheral stimulus of any kind acts as a master-key which unlocks any storehouse of past impressions and sets them free. The ugly face or gigantic toothbrush dangling as an advertisement over a shop has excited only passing curiosity by day, but at night the awakened mental picture of it is disturbed by incongruous associations of ideas and invested with malignant attributes. For in night terrors, as in hysteria and delusional insanity, our ideas, impressions, and memories, are emancipated from control by judgment, sense of proportion, reason, and experience. The peripheral stimulus is no doubt unpleasant in character, and the dream becomes so too. Even a trivial event or experience by day may become a terror by night. But should the experience have excited alarm at the time, its after-effects will be quadrupled in dreams. Hence night terrors are always worst in nervous, timid children.

Young children often give a clue to the nature of their dreams by their exclamations, but this is not always so. A boy of seven would regularly wake the household by shrieking, "It's that coking thing again!" He vouchsafed no further details, but possibly the sight of some complicated piece of machinery had once alarmed him and figured in his dreams. The prominent features of repeated night terrors in similar cases known to the writer have been, "A house with blind eyes" (i.e., with blocked-up windows), "A flapper" (talc ventilator), and "A bomp" (far foot-warmer). Night terrors of this kind are not symptomatic of any morbid condition in particular, but others are highly suggestive of a special cause.

Dyspnoea, whether produced by naso-pharyngeal obstruction, by asthma, by a distended stomach, by burying the head in a pillow, or by an ill-ventilated bedroom, is responsible for dreams of being overwhelmed by avalanches or tidal waves. Actual restriction of movement accounts for the paraplegic form of night terrors in which one is on the point of being overtaken, but cannot stir a step. Dreams of bleak and lonely Arctic regions and polar bears may arise from feeling cold. Dreams of flying and of falling into a bottomless abyss suggest labyrinthine vertigo. Severe bodily pain does not excite dreams. It either prevents sleep altogether or awakes the patient.

Visual and auditory hallucinations are commonly associated with febrile disturbance. But pyrexial hallucinations are more variable and lasting than those of ordinary night terrors. Auto-intoxication may account for febrile delirium, and also for certain cases of night terrors associated with constipation, the poison generated having similar effects to those produced by the belladonna group.

Goodhart and Still found a rheumatic percentage in seventeen out of thirty-seven cases of night terrors, and, as previously mentioned, teeth-grinding, which is a form of night terror, is a common symptom of gouty dyspepsia. But the rheumatic or gouty diathesis and the neurotic are closely allied, and night terrors are perhaps more significant of the neurotic temperament than of latent rheumatism or gout. It is true that night terrors are extremely common in chorea, but usually in cases which show least manifestation of articular or cardiac rheumatism.

Eaurosis and night terrors are often associated. In such cases adrenergic vegetations, or hyper-acidity of the urine, or coli-uria, may be suspected.

Prepubertal Mycosis as a Cause of Night Terrors.—In children, usually upwards of six years of age, the hallucinations may take a peculiar form well described by Dickens as "an interminable sort of rope-making, with long, minute filaments

his strands, which, when they were spun round together close to my eyes, occasioned screaming."

Such dreams of many webs in intricate movement, or of indistinct gyrating objects which grow monstrous or diminish in size as they alternately approach and move from the field of vision, are probably of the nature of migrainous auras, for sufferers from migraine have often experienced them in early life.

Day Terrors (Pavor Diurnus).—Day terrors are more rare than night terrors, but the symptoms are much the same. The attacks usually occur in children aged between four and six years. Suddenly, whilst apparently wide-awake or in the midst of play, the child will break off and rush for protection to someone in the room, shrieking, "It's something black!" or "I can't see!" or "A fly's humming in my ear!" or "Someone is after me!" Consciousness of surroundings is maintained, but terror is intense. An attack usually lasts a few minutes, and sometimes ends in a burst of weeping. Eventually the child sobb itself to sleep, and often wakes subsequently with a headache. The attacks may occur in the same subject both by night and by day. The hallucinations may be of sight or hearing. Sometimes they seem to be due to vertigo, for the cry is, "I am giddy!" or "The houses and trees are falling on me!"

Probably, as suggested in some form of night terrors, a migrainous aura may be the cause of the symptoms, but they may also depend on disease of the labyrinth. In younger children paroxysms of screaming without apparent cause seem to be akin to day terrors. In some cases constipation due to atony of the bowel has seemed to be the cause. Still has attributed day terrors to mucous colitis and dysmenia. The relationship between night and day terrors and epilepsy has to be considered.

Epileptics may of course be subject to night terrors, but the subjects of night terrors in their most aggravated form are rarely epileptics. It is possible also, as in an instance known to the writer, that true nocturnal epilepsy may be mistaken for night terrors, and the supposed sequence of events may thus be explained.

Epilepsy, however, may be excited by fright and emotional shock, whether sudden or prolonged; and as such conditions are fully supplied by severe and repeated night terrors, the possibility that night terrors may be an occasional cause of epilepsy cannot be excluded.

Yet night terrors and day terrors are far more closely related to hysteria, hysterio-epilepsy, narcolepsy, and day somnambulism, than to epilepsy itself. The significance of day terrors is graver in this respect than of night terrors alone. The "terror" in some cases resembles an epileptic aura, but the writer has only known one instance in which a day terror was the prelude to convulsions and unconsciousness. The patient, an hysterical girl aged six, had repeated night and day terrors for several years, but was convulsed on one occasion only. Her brother suffered from day somnambulism.

TREATMENT OF DISORDERS OF SLEEP.—Insomnia and disturbed sleep in infants should not be treated by anodynes and hypnotics without attempts to detect and remove the cause. Hunger and indigestion from unsuitable diet, under- or over-feeding, are to be first excluded. The hygiene of the bedroom, comfort or discomfort of night apparel and bedding, need attention. Common local causes are catarrh, cutaneous irritability, and painful dentition, but the latter is most

frequently due to catarrh or digestive disorder. Pyrexia from any cause will prevent or disturb sleep.

Hydarg. c. creta or pulv. ribei c. soda may be given when the bowels are deranged. Liquor ammon. acetatis and spir. ætheris nit. are useful remedies for catarrh. When pain is present, Dover's powder or paregoric should be used in approximate doses.

Night terrors should be treated on similar principles, bearing in mind the peculiar temperament of the child who suffers from them. They are always evidences of mental and bodily ill-health. Removal of adenoids and tonsils, intestinal worms, threadworms, and scybala, correction of refraction errors, anti-rheumatic remedies, and tonics, will not in all cases provide a cure. It is necessary also to correct morbid tendencies to dwell on horrors seen and unseen, to protect active and sensitive, as well as dull, brains from educational overpressure, and to shield nervous children as far as possible from thoughts, words, and deeds, which are calculated to increase their emotional instability.

A dose of 5 to 10 grains of bromide of potassium or sodium, given an hour or so before bedtime, is often sufficient to insure a night's rest; chloral hydrate (2½ to 5 grains) may be added when there is much nervous instability. Bromides may also be given by day in combination with bismuth, gentian, rhubarb, or castor-oil, in gastro-intestinal disorders; with ferri et ammon. cit. in anaemia; with strophanthus, digitalis, citrate of caffeine, when cardiac stimulants are required; or with aur. romica, hypophosphites, and cod-liver-oil, in debility. Aceto-salicylic acid or salicylate of quinine are valuable in rheumatic cases. Citrate of potash often acts well in cases of nervous diarrhoea, and is also useful when night terrors are associated with enuresis and the presence of highly acid, concentrated urine or with colic-uria. It is almost unnecessary to add that children who are afraid of the dark should be allowed the consolation of a night-light. It is also to be remembered that many a night terror in young children has its origin in shadows on the wall.

Somnambulism is often associated with night terrors, but may occur independently in children upwards of five or six years of age. Sleep-walking in any case is indicative of an overwrought brain. It occurs in epileptics and in their families, but there is no necessary relationship between somnambulism and epilepsy. The habit may be prevented by wearing a belt at night fastened at the back, and having a ring attached, through which a bandage should be passed and tied beneath the bed. The bandage should not be tight enough to restrict movements in bed nor loose enough to allow excursions outside.

2. DREAMY MENTAL STATES.

Most persons of the poetic, artistic, literary, and philosophic type have been subject in early life to fits of reverie and absorption in which they are lost to the world whilst engaged in vain imaginings. Some seem to have the power to conjure up at will spirits from the vasty deep, or visions in accordance with the mood and fancy of the moment.

Voluntary indulgence in the habit is not harmful except from an educational point of view. But dreamy mental states may be involuntary and associated

with signs of mental exhaustion. Danger-signals are changes in disposition, irritability, fits of passion, gloominess, lethargy, forgetfulness and inattention, headache, disturbed sleep, spasmodic, and involuntary movements. In some cases dreamy mental states are attended by ill-defined dread or an inexplicable horror of surroundings. Claustrophobia, agoraphobia, a painful sense of unreality of personal existence and identity, a loss of sense of space, time, and orientation, may be accompaniments.

"I feel as if it wasn't me," said a child, aged eleven, when asked to describe her sensations to the writer. Adults complain of a distressing feeling of banality, of pre-existence and of prescience, and also of hallucinations of sight, hearing, smell, and taste, in connection with dreamy mental states. Evidence of this in children is chiefly based on confessions in after-life. Children of the age of eight to twelve will not as a rule own to sensations of the kind, lest they should be called silly, whilst younger ones can only show that they are frightened, without explaining why.

Fits of reverie and absent-mindedness should not be regarded merely as bad habits or moral offences, although it is true that a sharp word or sudden shake may check them at their onset. They may be indications of impending cerebral exhaustion. Intelligent teachers can usually distinguish them from wilful inattention and otiosity.

Petit mal and epilepsy are no doubt related to dreamy states, but the subjects of dreamy states are rarely genuine epileptics. So-called "post-epileptic somnambulism" and automatism are seldom preceded by a typical epileptic fit. They are examples of "highest level" seizures, and dreamy mental states seem to be mixed. Petit mal may be mistaken for mind-wandering, but in the former the child is usually quite unaware of any gap in consciousness—finishes a sentence, answers a question, or goes on with his occupation, as if nothing had happened; whereas in the latter he is quite conscious of having been "wool-gathering" when abruptly taken to task for this offence. Moreover, in petit mal, there is usually a change of colour, a momentary facial twitch, or a stagger, besides a vacant stare, to guide us.

Hysterical Somnambulism.—Without condemning dreamy mental states wholesale as morbid, they must, when frequent and excessive, arouse suspicion of impending mental trouble or hysteria.

They are but one stage removed from the "hysterical somnambulism" of Janet, a condition in which the attacks have nothing convulsive about them, but are a species of waking dream in which past experiences are lived through again and enacted with minutest detail. For example, a boy aged fourteen was admitted to hospital on account of fits of uncertain nature. During an attack he would become unconscious of surroundings, and go through an elaborate pantomime of seizing and throttling some imaginary being, shouting triumphantly meanwhile, "I've got him!" "Say your prayers!" over and over again. This would go on for half an hour or more; he would then fall asleep, and when questioned on waking, he would say he had had a bad dream of a monkey which once jumped on his shoulder, pulled his hair, and frightened him.

Similar "monodic" or "polydic" somnambulism may be seen any day in the disgraceful platform exhibitions of professional music-hall hypnotists, and it is curious that such histrionic displays are only regarded as "epileptic" when they occur spontaneously or in consequence of some emotional shock or physical

injury. Day somnambulism, or *vigilambulism*, as it is sometimes called, is more closely allied to hysteria than to epilepsy.

Dromomania may be a high-sounding name for playing "truant," but different causes of vagrancy should be recognized. Imaginative and romantic children will sometimes roam in search of adventure. Some, like St. Teresa, seek martyrdom; and some, like Maggie Tulliver, run away to be gipsy-queens because they do not reign at home. Some not only wander abroad at every opportunity, but account for doing so by inventing stories of ill usage and privation, in which they may appear to believe. Others (usually feeble-minded) seem to obey a nomadic instinct to stay out and sleep out whenever they get the chance. Finally, there are cases of true day somnambulism in which there is for a time complete loss of memory and personal identity.

The ordinary truant is easily detected as a rule by his general demeanour, and by the manner in which he has occupied his time of unenforced leisure. A short and sharp shrift awaits him in accordance with his deserts. But truancy is not abnormal, whereas there is a distinctly morbid or neurotic element in all the other forms of vagrancy or *dromomania*. It is only the highly neurotic and sensitive schoolboy who runs away to escape punishment or persecution. A single escapade may result from mere thoughtlessness or from habits of morbid introspection and selfish disregard for the feelings of others. When practised repeatedly in spite of remonstrance, and when associated with dishonesty, untruthfulness, immorality, and other acts of misbehaviour, the question of moral irresponsibility or feeble-mindedness arises. All these conditions may be passing phases of hysteria at or about the age of puberty, cured by temporary isolation and moral influences. When vagrancy is associated with evidence of feeble-mindedness, segregation from society should be permanent. Day somnambulism as a cause of *dromomania* has already been considered. The treatment of dreamy mental states in general depends on recognition of their cause. When associated with definite evidence of cerebral exhaustion, irritability, and hysteria in children, freedom from excitement and educational overpressure, healthy mental and physical occupation in wholesome country air, are the best remedies. But it should be remembered that dulness, boredom, and monotony, are as provocative of hysteria as over-excitement and fatigue.

3. TICS AND HABIT SPASMS.

Tics or "Habit Spasms," as they are usually called, are certain oft-repeated, and seemingly purposeless, movements of the face, head, body or limbs. They are motor manifestations of psychical irritability and excitability. Their occurrence implies super-sensitiveness and abnormally active response to external and internal stimuli. They are common in the "unrestrained" and rare in the "restrained" type of neurotic children. They make their first appearance usually between the fifth and tenth year, and may persist to any age. They are slightly more common in girls than boys.

Ætiology.—Predisposing causes are neuropathic inheritance and environment, as in other neuroses which characterize the neurotic temperament. Emotional excitement, pleasurable or otherwise, mental and physical shock, strain, and exhaustion, and any condition that lowers general health, such as chronic indigestion, will induce the psychic and nervous instability which are responsible for tics.

Exciting Causes are local and peripheral irritation of any kind affecting any part of the body, internal or external, but in most cases the causes are trivial in themselves and the events outlast the causes. Tics are not evidence of masturbation nor of worms.

VARIETIES AND CHARACTERISTICS OF TICS.—All tics are at first actions defensive and protective, half reflex and half purposive, against some irritating peripheral sensations. A blink, a sniff, a cough, or a wriggle, for instance, are natural protests against irritation of eye, naso-pharynx, or skin. But instead of ceasing after having served their purpose, they are repeated long after provocation has ceased, and then become tic. An irresistible impulse to repeat such simple acts of self-protection arises, and is gratified by their performance. But they lose their purposive aim, and become purposeless and ineffective caricatures of ordinary defensive acts and gestures. An occasional blink is replaced by a series of futile flickerings of the eyelids; a good honest sniff becomes a constant succession of little useless ones. Clearing the throat is represented by weird grunts, snorts, lucks, or yelps. Sometimes the sound suggests a combination of cough, hiccough, and exclamation, in one. An ordinary gesture of dislike for some passing taste or smell is repeated as a hideous grimace, in which the eyes are tightly shut, the teeth shown, and the tongue held out.

Sometimes the child seems to indulge in efforts to twist his mouth round his nose or ear, or moves his lips like a munching rabbit. Efforts to keep a loose cap in place or hair out of the eyes or from tickling the neck will cause rapid corrugation of the frontalis, or the child shakes his head as though he would shake it off. A tight or frayed collar is responsible for frequent elevation of the chin or twisting of the head from side to side. Elaborate writhes and wriggles result from a discomfort caused by ill-fitting clothes. Abdominal tics in which the diaphragm descends five or six times in quick succession, and the abdomen bulges, may be similarly explained. The movements invariably suggest to anxious parents that the patient has something alive inside, and inexperienced practitioners may impute them to pressure on the parasympathetic nerves by enlarged glands.

Locomotor tics consisting in sudden hops, twists, skips, jumps, or stamping on the ground, probably originate in some defensive protest against a passing local discomfort.

Tics are divided in order of severity into simple, in which isolated muscular movements are practised; co-ordinated, in which the movements are more elaborate and complicated; and convulsive, when particularly violent and spasmodic.

Psychical Tic (*Gilles de la Tourette's Disease*) is so called when, in addition to imperative impulses to perform ridiculous actions, there are explosive utterances of obscene words (*coprolalia*) or imitation of bystanders' gestures and words (*echolalia*, *echolalia*). The mental symptoms in the latter are, however, more in evidence than the motor.

Distinction between Tics and Spasms.—Tics are not spasms. Spasms are the motor results of local irritation somewhere in a spinal or bulbo-spinal arc. A spasm cannot be produced or controlled by act of will; its distribution is anatomical, not co-ordinated. It is not expressive of emotion; it is not preceded by an impulse or followed by a sense of satisfaction. It ceases when the irritating cause is removed. In all these respects tics differ from spasms.

Tics are incorrectly described as "Habit Spasms." Tics are not, strictly

speaking, habits. Habits are acquired by practice, but tics arise suddenly; yet in time they do become habits, and this has an important bearing on treatment.

Tics at first are conscientiously performed as an ineffective, exaggerated and abnormal protest against some passing local irritation; but in time they are performed unconsciously, and are no longer preceded by irresistible impulses and followed by a sense of satisfaction. At this stage they become habits which may be continued indefinitely. In the first stage the patient is aware of them, and the impulse to produce them is increased by drawing his attention to them, and lessened by distracting it; in the second stage the patient is unaware of their occurrence, which is chiefly when his attention is distracted, and they may be controlled by effort of will. Far more commonly, however, a tic does not become habitual; it ceases as suddenly as it began, only to give place to another. A child rarely indulges in more than one tic or antic at a time. He drops one only to acquire another, and gloomily announces the fact at breakfast time to his careworn next of kin. Variability is a characteristic of tics in childhood, and has to be considered in relation to treatment.

DIAGNOSIS.—It is important to distinguish between tics and the movements of Sydenham's chorea. In chorea the facial movements are a succession of normal though exaggerated expressions of emotion of every kind, quickly passing, alternating and coinciding with each other. Frowns and grins, expressions of grief, terror, surprise, mirth, joy, anger, and so forth, come and go in ceaseless sequence. The facial tics are not expressive of any emotion; one kind replaces another, but rarely occurs simultaneously. In tic the patient simply "pulls faces."

The bodily movements in chorea are normal primitive actions of flexion, extension, rotation, etc., though purposeless, needlessly vehement, alternating and clashing with each other.

In tic the bodily movements are meaningless, complicated motor tricks, gestures, or antics, which are but a fantastic parody of normal actions. As in facial tic, each bodily tic continues until supplanted by another. In rare cases tics and true chorea may occur in the same subject, and the diagnosis may be difficult. As a rule they are easily distinguished from each other. Tics are not evidence of rheumatism, latent or otherwise. The most inveterate "ticqueur" rarely develops endocarditis, whereas in true chorea it is common.

Myoclonus (*paramyoclonus multiplex*, *myokymia*) differs from the tics and chorea. It is rare, but may be met with in children upwards of five years of age. It consists in sudden simple spasms of individual muscles, not groups of muscles having associated action. Many muscles may be affected simultaneously and symmetrically on both sides of the body. They are spasms—unlike tics—and resemble those produced by electrical stimulation of muscle nerves. They do not resemble volitional movements, and cannot be controlled by will. The face is rarely affected. The muscular movements are sometimes accompanied by a "sawing," creaking or friction sound, audible through a stethoscope. Myoclonus is sometimes accompanied by epilepsy (Uvemann's *myoklonus epilepticus*); it is sometimes a familial and hereditary affection (*cf.* *Nystagmus myoclonicus*, p. 688).

Imitation Tics.—True tics are not the result of mimicry. If a child in a school or institution begins to "tic" or suffer from chorea, others will often imitate it, but the copy is always a bad one. True chorea does not occur in epidemics.

Willful Tics.—It should be remembered that tics may sometimes be kept up and practised by impish children solely for the purpose of annoyance or to gain some private end. A boy aged eight, under the care of Sutherland, after going through the whole gamut of blinking, grimacing, itching his shoulders, wriggling, and writhing, started—as in the case previously mentioned—a most aggravating form of yelping tic. The nuisance became so great that the hospital authorities begged that he might be sent home. Expostulation with the boy only led to floods of tears at first, and no cessation of the noises. He was only quiet when asleep. After about ten days, however, it was noticed that he only yelped when left alone. He looked well and cheerful, and a merry twinkle in his eyes suggested mischief in his madness. Accordingly, he was shown a strong family battery, and told that its application would cure him of making noises, but would be very painful. He heard the battery buzz, seemed much impressed, and ceased to yelp from that time forward.

Treatment of Tics.—Tics are psycho-motor manifestations of nervous instability, reflections of the patient's environment, reactions to irritant stimuli of any kind. The psychical or mental elements in the causation of tics are of more importance than their outward or motor expression. A neurotic child of the unrestrained type is always liable to tics if his environment is unsuitable. Some only exhibit tics at school, and some only when at home. Hard tasks, long lesson hours, teasing, bullying, and nagging, on the one hand, late hours, too many parties and pantomimes, on the other hand, may be responsible. In every case the first principle is to secure a quiet, unexciting, and generally wholesome, mode of life for the child.

The treatment of the motor manifestations of tics differs in accordance with the stage at which the complaint has arrived. Treatment appropriate to early stages is inappropriate to the later. In the early stage, when tics are consciously performed in obedience to an overwhelming impulse, it is worse than useless to try to check them by scolding or any form of punishment, or by a system of rewards and moral persuasion. Meigs and Feindel regard tics as due to volitional infirmity or weakness of will-power; but, after all, the will to resist may be strong, but the impulse to yield may be stronger.

"The evil done we may compute;
We know not what's remedied."

All tiqueurs bear witness to the desperate struggles made to control the impulses, and to their attempts to circumvent them in various ways.

For example, a boy aged nine, who caused the greatest annoyance by uttering shrill, puppy-like yelps at intervals of a few seconds, was told that if he made no noises for a whole morning he would be allowed to attend a hospital entertainment in the afternoon. For some hours not a sound was heard; but a nurse, peeping round the screen which surrounded his bed, discovered the little fellow half suffocated, with his head buried under the pillow, smothering his "yelps" as well as he could. He was not deprived of the promised treat, and the pleasure of it caused him to forget to yelp at the time, and he seldom did so afterwards.

Tics in the early, "impulsive" stage are increased by drawing the patient's attention to them. The treatment is, obviously, to take no notice of them, and to supply any kind of mental and physical occupation which will distract the patient's attention from them.

The tic "states," in which a variety of tics replace each other, should be regarded as expressive of an overwrought brain on the verge of exhaustion. If rest, and freedom from worry, excitement, and fatigue, are afforded, the tics may be left to take care of themselves.

The second stage of tic, in which they become "habits" no longer consciously performed and in obedience to an impulse, needs treatment on opposite principles.

At this stage they can be arrested by drawing the patient's attention to them and encouraging his power of control. They occur when the child is off his guard and believes himself to be unobserved, or at moments of excitement.

In such cases, Porot, Reissand and Péroz recommend elaborate forms of moral and physical drills and exercises. Porot would train the child to stand like a statue before a looking-glass, and teach it not to wince or jump when pricked or tickled or when bright objects are suddenly brought close to the eyes.

Such procedures are only admissible when tics have become "habitual," and it is ascertained by observation that mere attention is sufficient to control them.

Treatment of Local Causes of Irritation.—Tics are suggested, not caused, by local irritation. A spasm ceases when the local cause of irritation is removed. A tic may long outlast the irritation which suggested it. A facial twitch or blepharospasm which is cured at once by removal of a local irritant—*e.g.*, a decayed tooth—is not a tic at all.

It is customary in all cases of tic to search for and correct every minor abnormality of eyes, nose, throat and mouth, or prepuce, which may possibly produce local irritation. But when the most fastidious standards of perfection in all these departments have been satisfied, it often happens that the tics are worse than ever. Specialists claim to cure the particular form of tic which appears to come within their province, but are seemingly unaware of the variability of tics and their tendency to replace each other. A child may cease to blink, not because an infinitesimal error of refraction has been corrected, but because some vague bodily discomfort has suggested another form of tic.

True tics are not cured by correction or removal of minor and trivial sources of irritation. At the same time any serious defect in the shape, for instance, of conjunctivitis, astropia, adenoid vegetations, decayed teeth, which naturally lowers general health, should on no account be neglected. A mild form of follicular conjunctivitis frequently causes blinking, which in such cases is a spasm, not a tic.

Initiation tics should be stolidly ignored, or, if necessary, treated by firmness and douches of cold water.

When there is much mental excitement and irritability, bromides should be given to secure sleep and prevent night terrors. Antipyrin, in 3- to 5-grain doses, is sometimes useful. Arsenic and hypophosphites with or without iron and cod-liver-oil serve as tonics. Constipation and other digestive disorders need attention.

4. CONVULSIONS AND EPILEPSY.

A convulsion is defined by Hughlings Jackson as, "A sudden excessive discharge of many nervous arrangements representing movements at once, or nearly together, because the cells subserving such movements have become highly unstable."

Such instability may be due (1) to some inherent morbid condition of nervous cells which leads them to discharge superfluous energy from time to time, much as

an automatic flushing tank empties itself when full. Convulsions or epilepsy are called "idiopathic" in such cases. (2) The instability may be acquired. The nerve cells or neurons may be of average stability, yet rendered unstable by the violence of some toxin or by the intensity of a stimulus which reaches them. In such cases convulsions or epilepsy are described as "symptomatic."

Hypothetically the distinction between idiopathic and symptomatic convulsions and epilepsy holds good, but in practice it is often difficult to distinguish cases of convulsions attributable to minor causes of irritation from those in which none can be found.

In many the supposed causes of irritation are so slight in themselves that we cannot reasonably regard them as capable of exciting convulsions in subjects whose brain cells are of normal stability. Similar morbid conditions excite convulsions in some children, and not in others. Convulsions may attend a mild ailment in one child, and be absent during a serious illness in another. It follows, therefore, that an additional factor—namely, instability of the neurons—must be present in the former and absent in the latter. Convulsions which seem to be symptomatic may also be idiopathic.

Purely symptomatic cases are those in which the cause, whether in the shape of profound toxæmia or gross cerebral disease or injury, seems all-sufficient to irritate healthy neurons to the "dash" point. For instance, the neurons of a perfectly healthy cortex may be rendered unstable by such poisons as strychnine, arsenic, absinth, and alcohol, or by the toxins of tetanus and uremia, or by infection by *B. coli* and other micro-organisms.

A powerful emotional shock may excite fits in a person whose brain cells have shown no previous signs of instability. Asphyxia or severe hemorrhage may induce convulsions by depriving the neurons of oxygen, and so rendering them unstable. Gross lesions of the brain and meninges, such as hemorrhage, inflammation, embolism, thrombosis, tumors, fractures of the skull, or concussion, may all give rise to epilepsy or convulsions by direct irritation of healthy cortical neurons.

An indigestible meal, like an overdose of strychnine, may induce convulsions even in a healthy child. The constant irritation caused by an adherent prepucial band or polyp in the nose or external auditory meatus, the passage of a tooth through inflamed and tender gums, obstruction of the naso-pharynx by tonsils and adenoids, the presence of worms, may so lower general health that healthy neurons become unstable and fits are the consequence. But in the majority all these forms of irritation do not induce convulsions, but only symptoms which are speedily relieved by removal of their cause. It is only in the minority that peripheral irritation is the sole cause of convulsions. "Reflex" and "symptomatic" are commonly used as synonymous terms, but true reflex convulsions comparable to those which Brown-Séquard produced by pulling guinea-pigs' whiskers are distinctly rare.

In all cases of convulsions seemingly induced by minor forms of local irritation the idiopathic element asserts itself, and it is important, in connection with prognosis and treatment, to bear in mind "borderland" cases as well as the two main groups, idiopathic and symptomatic.

As to the cause of idiopathic or inherent instability of the cerebral neurons, one can only say that it is a matter of neuropathic inheritance. There is a history, not necessarily of epilepsy itself, but of some neuroses such as hysteria, neurasthenia, asthma, alcoholism, narco-mania, eccentricity, or insanity, in the families

of most persons who are subject to convulsive disorders which are not definitely symptomatic in origin.

Convulsions in infancy and childhood may be considered under the following headings :

Eclampsia Neonatorum.

Convulsions preceding and during the Period of Primary Dentition.

Convulsions occurring between Primary and Secondary Dentition, and later—Epilepsy.

Eclampsia Neonatorum—**EXCITING CAUSES.**—Convulsions at birth or within a few hours of birth may be excited by direct injury or by asphyxia due to super-tension of blood and venous turgidity arising from prolonged and difficult labour, especially in the case of first-born infants. The prognosis is far better in the latter than in the former. A large number of infants who become convulsed shortly after birth are subsequently found to be suffering from spastic hemiplegia, diplegia, and paraplegia. These conditions are usually attributed to supratentorial hemorrhage due to pressure on the cranium at birth. The occurrence of such supratentorial hemorrhage is undoubted, and in a few cases the clot has been successfully removed by operation. There is, however, little pathological evidence that the different forms of cerebral birth palsy are actually produced in this manner. After death, in cases of birth palsy, the cerebral conditions found are as a rule localized atrophy of the motor cortex, absence of development of cortical cells rather than definite evidence of hemorrhage. Pericnephaly with cystic formation in the meninges may be present. In extreme cases the whole brain may be shrunken and "walnut-like." Eclampsia and birth-palsy are not always due to congenital syphilis, but may depend on mal-development of the cerebral cortex.

Syphilitic infants often die in convulsions shortly after birth, but gross syphilitic intracranial lesions are rarely found in such cases.

Convulsions preceding and during the Period of Primary Dentition—**Gastro-Intestinal Disturbance.**—During the first year of life, improper or over feeding takes the first place amongst the exciting causes of convulsions, although convulsions at this period have from time immemorial been attributed to teething alone.

Dentition, however, is a painless process in a healthy child. It is only when the gums are tender and inflamed over a protruding tooth that pain is caused. Painful dentition results from painful gums. Indigestion makes the gums tender, and the baneful habit of allowing an infant to bite hard on septal rings and "comforters" bruises the gums and produces local or general gingivitis. Hence the convulsions during the teething age should be ascribed, not to dentition itself, but to the causes which make it painful, and these are gastro-intestinal disturbance and unhealthy gums.

Rickets is regarded by many as a potent cause of convulsions. In the writer's opinion, however, convulsions in rickets, like the other nervous associated with that disease—tetany, irritability of accessible nerves, and laryngismus—are chiefly caused by intestinal toxæmia due to improper feeding, for they speedily subside under suitable diet and treatment. One rarely sees convulsions in rickets apart from tetany and laryngismus, and the fits themselves may be partly asphyxial, in consequence of spasmodic closure of the glottis. Similar asphyxial convulsions are seen in cases of congenital stridor, which is the result partly of spasm of the glottis

and partly of congenital malformation of the epiglottis. Extensive obstruction of the naso-pharynx by tonsils and adenoids will sometimes give rise to nocturnal fits, which are, again, due to partial asphyxia. Convulsions in whooping-cough are also of asphyxial origin.

Infant Convulsions, in which the infant moans, distorts its face, clenches its fists, and draws up its legs, are due to colic. The attacks are infrequently culminate in general convulsions. It is not always easy to decide whether colic and convulsions are due to mechanical irritation of the intestines by masses of indigestible material or to intestinal toxæmia. Sometimes the child solves the mystery by voiding a quantity of curds or the fibrous portions of fruit and vegetables, which have usually been given to correct constipation. Diarrhoea with foetal, undigested stools and rise of temperature suggests a toxæmic origin of convulsions.

Allusion has already been made to eclampsia uterina and salivæ spasms, which, unlike "inward convulsions," are associated with temporary loss of consciousness.

Fits at the onset of exanthemata are rare. When they occur, it will usually be discovered that the child has previously been subject to them. The statement that convulsions in infants correspond to rigors in adults is, I believe, unfounded. True rigors are not uncommon in infancy, especially in cases of septic absorption—for instance, pyæmia and pyelonephritis due to infection by *Bacillus coli* or other micro-organisms.

Convulsions in Cerebral Disease.—Towards the end of the first and throughout the second year of life convulsions are commonly associated with various forms of non-tuberculous meningitis, post-hæmic, cerebro-spinal (epidemic or sporadic), suppurative, syphilitic, and hæmorrhagic (pachymeningitis hæmorrhagica). In the two latter affections a condition of tetanic rigidity is more common than one of clonic spasms. A considerable proportion of cases of non-tuberculous meningitis are due to otitis media, which is often unsuspected. After the second year, and between it and the sixth, tuberculous meningitis is far more common than other forms. Convulsions are more common towards the end than at the beginning of tuberculous meningitis, whereas the reverse is the case in other forms of meningitis.

Acute Encephalitis is usually ushered in by generalized convulsions and signs of widespread cerebral disturbance. The disease frequently follows in the wake of measles and other acute fevers. Polio-encephalitis is analogous to spinal poliomyelitis (infantile paralysis), which, again, sometimes commences with convulsions. The general tendency of acute polio-encephalitis is towards recovery, but more or less lasting damage is sometimes done to various areas of the brain and cerebellum, producing symptoms which localize the parts affected. Infantile hemiplegia, with convulsions of the Jacksonian type, are often the result of encephalitis involving the motor areas of the brain. When the prefrontal areas are affected, all degrees of mental enfeeblement, including complete idioy associated with frequent epilepsy, may be the consequence.

The subjects of *Hydrocephalus*, whether congenital or acquired, are often epileptic.

Thrombosis of the Cerebral Sinuses in marasmic infants is an occasional cause of convulsions; also embolism in cases of congenital heart disease.

Intracranial Tumours in any site give rise to generalized convulsions in consequence of the increased tension and anæmia of the brain which they produce, either on account of their size or of the hydrocephalus which attends them.

Epilepsy supposed to be idiopathic may be caused by a tumour in the cerebellum or other parts of the brain.

Uremic Convulsions are very rare in infants under two years of age. The nephritis which gives rise to them is usually septic, scarlatinal, or syphilitic.

Traumatism, in the shape of blows or falls on the head causing depressed fractures and concussion of the brain, is an occasional cause of epilepsy and convulsions.

Convulsions between Primary and Secondary Dentition, and later—Epilepsy.—After primary dentition is complete, convulsions may still be due to the same causes as in earlier infancy—namely, gastro-intestinal disturbance, laryngismus, whooping-cough, and various forms of non-tuberculous meningitis. But tuberculous meningitis, cerebro-spinal meningitis, sporadic or otherwise, poli-encephalitis, poli-myelitis, cerebral abscess from otitis media, cerebral tumours, hydrocephalus, and uræmia are more common causes of convulsions after than before the age of two years. Moreover, it is after the second year that psychic or emotional factors are concerned in the causation of eclampsia, and that typical epilepsy makes its appearance.

Relation between Epilepsy and Infantile Eclampsia.—Epilepsy is rarely diagnosed until primary dentition is complete, but there does not seem to be any fundamental difference between epilepsy and infantile convulsions. Infantile eclampsia is infantile epilepsy. Certainly, little babies do not bite their tongues in fits, nor do they experience "aurea," so far as one can judge, nor emit the hoarse scream or "lost soul's wail" which is characteristic of epilepsy in adults. But in many adults who are undoubtedly epileptics the seizures are in all respects like those of infants. The convulsions are generalized from the first, there is no cry nor any warning, the tongue is not bitten, and the sphincters are not invariably relaxed. We cannot refuse to recognize epilepsy because all the classical symptoms of the disease—known throughout all ages—are not present in an individual case, and so academic discussions on the diagnosis between infantile eclampsia and infantile epilepsy are unavailing.

Typical grand mal probably does not occur in infants under one year; but petit mal, consisting in momentary pallor, a dazed look, and slight twitching of part of the face or limb, often unilateral and followed by somnolence, may often be recognized within the first few months of birth. The real nature of such attacks, however, may be unsuspected, even when they are of frequent occurrence during many years.

The causes of epilepsy and eclampsia are the same. Epilepsy may be idiopathic or symptomatic. A third variety in which fits occur only under emotional stress may be termed "accidental."

Emotional Shock.—The writer has a good many cases in his notebooks of epilepsy following immediately or within a few weeks of some emotional shock. "Was frightened by a man" appears several times as the cause of fits in young children. In one, a boy aged fourteen, the first fit occurred immediately after seeing an unfortunate cat maddened by the pain of being accidentally scalded.

In many cases fits only recur after some emotional excitement, pleasurable or otherwise. In some, especially between puberty and coming of age, epilepsy appears in consequence of mental overstrain, worry, and anxiety, usually educational. Such cases are both preventable and curable. They are partly idiopathic and partly symptomatic.

Symptomatic epilepsy and convulsions may be due to meningitis, acute and chronic; to intracranial abscess or tumour; to congenital syphilis and cerebral lesions, the cause of birth palsy; to trauma, acute polio-encephalitis, or myelitis; to uræmia, and general bacterial infections.

SYMPTOMS OF TYPICAL EPILEPSY.—It is needless to describe in detail the motor features of an ordinary attack of epilepsy: not those of infantile eclampsia. A typical epileptic fit may begin with an aura which may be psychical, sensory, or motor, in each case indicating the part of the brain in which the discharge originates. Sometimes there is no aura or "warning" and the fit commences with a sudden fall with or without a scream or cry. The aura, when present, is followed by unconsciousness, and tonic succeeded by clonic spasms, localized or generalized. The immediate after-effect is sleep, which may be coma-like in deepness. The duration of an epileptic attack varies from a few minutes to several hours, in which the convalescent stage may be repeated over and over again. Recovery of consciousness is followed by headache, sometimes vomiting, and a feeling of general exhaustion. In some cases, automatism, hysterical violence and screaming, acute temporary mania, or transient paralysis of a limb or limbs, succeed a fit. Biting the tongue and evacuation of urine and feces are not essential for the diagnosis of epilepsy.

Aura occur in about one-half of all cases. Their importance is not only that their nature indicates the site in the brain of a local discharge, but also that they may occur without other obvious signs of epilepsy, and are therefore often misunderstood.

Varieties of Aura and Localization of Origin.—Aura may be psychical, sensory, or motor. Aura of special senses may be visual, auditory, olfactory, and gustatory. Aura of any kind are rare in children under five.

It is important to recognize that aura of all sorts may occur without other signs of epilepsy for many years. The discharge, so to speak, misses fire. Epileptic patients often say that they frequently experience the aura without a fit, but seldom a fit without the aura. A patient recently under the writer's care complains of frequent sensations resembling those produced by a "shocking coil," in his right arm and leg. They last a few seconds, and may occur many times during the day for several days in succession. So long, he says, as the sensations are confined to the right side he never has a fit, but if the left arm becomes similarly affected he knows that a fit is imminent.

Psychical aura in which there is a momentary feeling of bewilderment, a sense of unreality of one's existence or surroundings, and so forth, probably indicate a discharge from the prefrontal areas of the brain. One seldom hears of the elaborate and complicated psychical variety of aura in young subjects. It is only mentioned when actually associated with fits in later life. But the writer has been told by Langmead of a boy aged eight who said that always just before a fit he seemed to see a lady sitting at the window of a railway-carriage with a little dog upon her lap. The dog looked through the window and laughed at him, and he remembered no more. The writer has met with a boy, aged one year and eleven months, who had two or three fits daily, and evidently knew when one was coming on, for he would cry, "It's after me," and would try to crawl under the bed to get away from it.

Aura of Cutaneous Sensations—for instance, tingling, formication, or actual pain commencing in one or other part of a limb and spreading upwards—imply,

according to Harvey Cushing and others, a lesion of the post-central gyrus or post-Rolandic areas of the cortex.

In children of three and four and upwards, "warnings" in the shape of pains in the back or abdomen, or sensations of something crawling on their arms or legs, are not uncommon. Such auras, when they happen to be particularly painful or distressing, may be attributed to mere local affection, and their real import may be unrecognized. One of my patients, a boy aged twelve, had been sounded for stone in the bladder on account of frequent attacks of severe pain about the hypogastrium, which sometimes, it was said, caused him to faint away. In another lad, aged ten, who suffered from so-called "fainting attacks" preceded by epigastric pain and followed by vomiting, and sometimes hæmatemesis, gastric ulcer had been diagnosed. Another boy, aged sixteen, complained of waking at night with a horrible sensation that his eyes were going round to the back of his head. He would endeavour to "catch his eyes by twisting his head round"; then he would "give one long yetch and fall asleep again." In this case nightmare had been the explanation suggested. It is fair to state that the diagnosis of epilepsy was clear enough in all these cases by the time they came under observation. Yet, possibly, a careful investigation of the symptoms would have revealed their nature earlier.

The common epigastric aura seems to indicate instability on the part of the vagal or vasomotor centres.

Motor Aura, such as spasms and convulsions of any part of the face or limbs, point to a discharging focus in the pre-Rolandic gyrus (anterior Rolandic motor area). The exact site in Jacksonian epilepsy is particularized by the part—for instance, thumb or finger—in which the spasm begins.

Visual Aura, consisting in flashes of light, bright or coloured, often succeeded by total blindness, like the aura of migraine, point to the occipital cortex as the discharging centre.

Auditory Aura, in which sounds as of bells, steam-whistles, loud crashes, and sometimes voices, form the hallucinations, are attributable to a discharging lesion in the temporo-sphenoidal lobes. Sudden vertigo, in which the patient falls or complains that trees and houses are falling upon him, sometimes precedes a fit.

Olfactory and Gustatory Aura are rare, and probably proceed from the uncinate gyrus in the median surface of the temporo-sphenoidal lobes.

The writer has recorded elsewhere the case of a boy, aged eleven, who for two and a half years had been subject to attacks in which he would suddenly run to his mother or, if in school, to his teacher, exclaiming, "Horrid smell!" as "It's the smell!" He would appear much agitated, though not terrified, for a few moments, sometimes spitting and grimacing, and going through the action of trying to pull something out of his mouth. He would then behave as if he were silly, running to persons in the room, trying to kiss them, and talking unintelligibly meanwhile. This would go on for five or ten minutes, after which he would become quiet, turn pale, shiver, seem exhausted, and lie down to sleep, awaking subsequently with a severe headache, and remembering nothing except that he had experienced the disgusting smell. He also had attacks resembling those of petit mal at times, and sometimes, instead of the evil smell, he would complain that "something was running about in the back of his neck," but he had never had convulsions since infancy, when as many as seven fits a day occurred during dentition.

The parents and relatives of epileptics rarely mention petit mal as a symptom

unless questioned on the point. Advice is sought about fits, and not for the frequent attacks of momentary reverie, inattention, with dazed expression and slight spasms of face and eyes, which so often precede, it may be for years, an attack of typical epilepsy.

The relationship between night and day terrors and epilepsy has already been considered.

PROGNOSIS IN INFANTILE CONVULSIONS.—The immediate prognosis, so far as life is concerned, is fairly good. Even newly-born infants rarely die in the first attack. In fatal cases there is usually hyperpyrexia, with much cyanosis, cardiac and respiratory failure. The cause of convulsions, which in such cases persist more or less incessantly for twelve to twenty-four hours until death occurs, is probably some kind of profound toxæmia, and not a primary instability of nervous elements. No gross changes are discovered after death, except venous congestion of the brain.

The ultimate prognosis in infants who recover from a primary attack of convulsions should always be cautious and guarded.

The questions invariably asked are, "Will he be an epileptic?" and "Will his brain be affected?" Both are difficult to answer. If the child has obviously been overfed or inanitably fed, and the family history is good, the prognosis is probably favorable. If no fault can be found with the diet, and if the child seems in perfect health up to the time of and after the seizure; if, again, there have been for days together a series of slight transient general or unilateral spasms, with momentary loss of consciousness, the outlook both as regards mental condition and subsequent epilepsy is anything but hopeful. Unilateral convulsions render the prospect of recurrence likely, for in most cases, though not in all, some local lesion in the shape of encephalitis or vascular occlusion is the cause. A certain degree of hemiplegia often follows unilateral convulsions, and if it persists for more than forty-eight hours after the seizure the presence of a lesion of the opposite half of the brain must be assumed. The paralysis remains more or less permanent, and Jacksonian epilepsy may be predicted.

About 10 per cent. of infants who suffer from convulsions become epileptic in after-life. The percentage would be lower were cases of convulsions due to organic cerebral disease excluded.

The prognosis in cases of fits occurring during the period of primary dentition, which is also the time when rickets is developed, is on the whole good, for in the great majority some exciting cause in the shape of alimentary disturbances will be found, and may be corrected.

The hypothesis that the nervous acquire the habit of instability in consequence of frequent explosions is, in the writer's judgment, without basis. It is true that in some cases a *status epilepticus* is established in which the child is hardly ever free from fits, and the result is death or imbecility. Serious arachnoiditis (*meningitis externa serosa*) seems to be the cause. But the writer has notes of cases in which fits have occurred, not once, but many times daily for months on end, and then have ceased entirely. Obviously in such cases frequent repetition has not induced the habit of convulsions.

In all cases the probability that epilepsy may become latent should be entertained when giving a prognosis. In 34 of 237 cases, intervals of from two to thirty years elapsed between the seizures. In 2 the interval was two years; in 2 it was four to five years; in 3 it was six years; in 5 it was seven years; in 6 it was eight to ten years; in 4 it was twelve to thirteen years; in 5, fourteen

to fifteen years; in 2, seventeen to eighteen years; in 2 it was twenty years; and in 3 there was an interval of twenty-six to thirty years in which no fits occurred. In all these cases there is a definite history of convulsions in infancy or within the first few years of life, followed by epilepsy after the lapse of many years. The figures therefore show that, whilst it is impossible to pronounce any patient cured of fits who has ever suffered from them, there is at least a probability that he may escape recurrence for many years.

There is a remarkable fall in the incidence of convulsive seizures between the fifth and twelfth years of life, and a rise in both sexes from the time of puberty to adolescence. Medical men are apt to make too little, and the public too much, of the epochs of life in relation to disease. Parents of girls who suffer from epilepsy at puberty always attribute the fact to some abnormality in menstruation. The writer has seen a fair number of cases in which epilepsy coincided with the first menstrual period, and in some the fits seemed more frequent during menstruation than at other times. But as the incidence of epilepsy at puberty is practically the same in both sexes, he is inclined to regard puberty itself as the chief factor rather than menstruation.

SEX AND AGE INCIDENCE IN IDIOPATHIC EPILEPSY AND CONVULSIONS.—Of 237 cases of epilepsy and convulsions, 121 were male and 115 were of female sex.

Age Incidence.					Male.	Female.
Under 1 year	13	12
Between 1 and 2 years	16	17
" 2 " 3	9	5
" 3 " 4	6	2
" 4 " 5	2	2
" 5 " 6	3	2
" 6 " 7	4	1
" 7 " 8	1	7
" 8 " 9	2	2
" 9 " 10	5	1
" 10 " 11	5	4
" 11 " 12	3	2
" 12 " 13	3	7
" 13 " 14	2	11
" 14 " 15	7	8
" 15 " 20	18	15
Over 20 years	17	16

Omitting cases of convulsions occurring after the age of twenty years, it will be seen that 82 of the remaining 204 were under four years (46 males, 36 females). Between the fifth and twelfth years the numbers fall to 51 (29 males, 22 females); between twelve and fifteen years the numbers are 38 (12 males, 26 females); between fifteen and twenty years they are 35 (18 males, 15 females). These figures demonstrate the fact that from the fifth to the twelfth year there is a considerable fall in the incidence of convulsions in both sexes, and that a rise occurs between twelve and fourteen years and from fifteen to twenty years of age.

The prognosis in cases of epilepsy occurring only after emotional excitement is eminently favourable, provided that the patients are understood and properly managed. These "accidental" epileptics are neurotic, imaginative, active-minded, excitable, restless, and easily exhausted. Their family history is one of

eccentricity, insanity, genius, hysteria, asthma, migraine, mania, alcoholism, and suicide, rather than of epilepsy itself. A personal history of night terrors, somnambulism, headache, epistaxis, habit spasm, and fainting fits, is usually forthcoming in the accidental epileptic. The attacks to which they are liable may resemble closely those of typical epilepsy, and elaborate psychical aims are wont to precede them. But there is usually an hysterical element about them, and they are more correctly described as epileptic hysteria than as hystero-epilepsy.

TREATMENT—Eclampsia Neonatorum.—Convulsions in the newly-born may be asphyxial, due to supervenosity of blood and to venous turgidity, arising from protracted and difficult labour, especially in primiparae. Cyanosis is frequently present in such cases, and the treatment, provided that the infant is obviously strong and plethoric, consists in the application of leeches, one to each mastoid process, or in actual venesection, $\frac{1}{2}$ to 1 ounce of blood being abstracted. Pallid, puny, and anæmic infants who suffer from convulsions shortly after birth should be treated by warmth, stimulants, and saline injections.

Cerebral due to Supraventricular Haemorrhage at Birth.—Prolonged and difficult labour suggests this cause. If other evidence, in the shape of convulsions, unilateral convulsions, and persistent hemiplegia, is present, craniotomy and removal of the clot are clearly indicated. A few cases have been reported in which this operation was successfully performed.

Convulsions during the Periods of Proliferation and Dentition.—In the absence of traumatism, organic disease of heart, lungs, and kidneys, congenital syphilis, scitis media, and the various forms of meningitis—simple, basilar, purulent, and tuberculous—convulsions are usually traceable to improper food and overfeeding. When the gums over an erupting tooth are swollen, painful, and inflamed, lancing may be beneficial; but it should be remembered that gastro-intestinal disturbance, and not the erupting tooth, is the probable cause of the convulsions.

TREATMENT OF CONVULSIONS IN PROGRESS.—Hot baths, with or without mustard, are of doubtful efficacy, but may relieve colic when present. Ice should be applied to the head when there is hyperpyrexia, and the patient should be placed in warm water, which should be gradually cooled to 60° F., or even 40° F. In all cases the lower bowel should be washed out with saline solution (1 drachm to $\frac{1}{2}$ pint of warm water). Then a rectal injection of chloral and bromide, 3 to 5 grains of the former and double or treble the amount of the latter, in 2 ounces of water for a child of six months of age, should be given. Inhalation of chloroform may be used to allay spasm until some of the injection has had time to become absorbed. Inhalation of nitrite of amyl is recommended by some, and hyalobromide of hyoscyne in hypodermic injection of $\frac{1}{16}$ to $\frac{1}{32}$ grain is said to be useful in protracted cases of convulsions. It is not, however, free from danger in debilitated infants. Injections of morphia ($\frac{1}{16}$ grain) for a child of six months are preferable to those of hyoscyne, but should never be repeated within twelve hours.

When cyanosis is present, leeching, or even venesection, may be employed, provided that the infant is fairly strong.

Emetics are never advisable, but the stomach should be washed out if there is any suspicion of its containing poison or irritants of any description. Mustard is the most dangerous of all emetics for a young infant.

Whilst treatment is in progress, the probable exciting cause of the convulsions

should be ascertained by observation and inquiry. Whatever this may be, a full dose of castor-oil or calomel, if it can be swallowed, never does any harm, and may have the good effect of preventing recurrence of fits.

TREATMENT OF CHRONIC EPILEPSY.—The general treatment of a confirmed epileptic consists in attention to ordinary hygiene. Diet should be simple and plainly-cooked ordinary mixed food. There is no merit in depriving epileptics of meat in moderation. A purin-free diet is not a panacea. If successful in isolated cases, the reason is usually that the patient has been accustomed to eat voraciously, as many epileptics do, and the comparative starvation of a "purin-free" diet is therefore beneficial. A salt-free diet has been recommended, and possibly does good in some cases of uræmic epilepsy. Needless to say, alcohol, strong tea, or coffee, should be strictly forbidden. The bowels should be carefully regulated.

Removal of Sources of Peripheral Irritation.—As already stated, purely reflex epilepsy is very rare. It is without dispute that a mass of cocconit in the intestines, or a considerable blockage of the naso-pharynx by tonsils and adenoids, or the constant irritation of balanitis due to a narrow adherent prepuce, or a serious error of refraction which induces eyestrain and frequent headaches, may be factors in the causation of epilepsy and convulsions, for this is true of any condition which undermines health. But to wage a systematic crusade against every slight abnormality which may be found, whether in eye, nose, ear, throat, or elsewhere, in the hope of curing epilepsy, is, in the writer's judgment, unreasonable. He would correct, with any degree of hopefulness, only such abnormalities or morbid conditions as seem sufficient in themselves to cause obvious discomfort and derangement of health.

The epileptic colonies, with their system of mechanical daily routine of occupation, regularity of meals and hours, are well suited for the ordinary chronic idiopathic epileptic whose physical strength is sufficient, and whose mental calibre is under average. The colonies are not equally suitable for the neurotic, highly-strung, excitable, and usually intellectual, if eccentric, individuals who have been described as occasional or accidental epileptics. One cannot set them to dig and delve for the term of their natural lives.

It must be remembered that there are many historical examples of distinguished persons who have been occasional epileptics. So, although it is often necessary to impose for a time a quiet and monotonous mode of life on some neurotic child or young person who has shown minor or major signs of epilepsy in consequence of mental overstrain and excitement, it is not advisable to condemn such patients to the existence of automata for ever. It is better to order their lives with due and sympathetic regard for their mental and emotional proclivities, taking care not to overstrain the one or harass the other. Frequent change and variety of mental and physical occupation are better than dull routine. Anything calculated to induce undue excitement, pleasurable or otherwise, should be avoided. The possibly unfavourable influence of their companions, teachers, and one must sometimes include parents, should be carefully considered. It is a common experience that children and young people who are always having fits at home lose them when moved from the family circle.

Bromides may lessen the severity and frequency of attacks, but will not cure epilepsy. When given in injudicious doses for long periods they do more harm than good. A common mistake is to increase the dose after every fit, whereas the proper course would be to lower it.

Bromides do not seem to have a selective action on different areas of the brain. Hence, if given in large doses for long periods, they may, whilst reducing the explosiveness of motor areas, at the same time lower the inhibitory or controlling functions of other realms. The result of giving bromides continuously and in excess is general mental and physical decrepitude, besides impairment of digestion and repulsive eruptions. The epileptic who is not treated at all is better off than one who is overloaded with bromides.

Still, bromides in moderation have an undoubted influence in lessening the frequency and severity of fits. The dose should not exceed 60 grains per diem for an adult, or 15 grains for a child below the age of puberty, and this maximum should be reached only by slow degrees. If the patient seems no better whilst taking the maximum dose, strychnine and other tonics should be given instead for a week or two, after which bromides should be cautiously administered again. It does not seem to matter which salt of bromide is used, if not given in excess. Any salt in full doses is apt to disagree, and therefore it is best to mix them. The reported efficacy of bromides of strontium, gold, and zinc, is, in the writer's opinion, due to the fact that they are usually given in smaller doses than the bromides of potassium, ammonium, and sodium, and are commonly prescribed after the latter have upset health and digestion. Arsenic undoubtedly has some effect in preventing bromide acne.

Digitalis and belladonna are useful adjuncts to bromides when the circulation is poor, but in the writer's experience belladonna has no special effect upon petit mal.

The headaches which so often distress epileptics may be relieved by cannabis indica, caffeine, citrate, phenacetin, and phenazone.

The writer has never found borax of use when given alone, but it seems to increase the efficacy of bromides.

Time of Administration.—In cases of nocturnal epilepsy, a full dose of bromides should be given a couple of hours before the fit is expected. Fits which occur on rising in the morning should be treated by giving a light meal and a dose of medicine half an hour before getting up. In diurnal epilepsy, bromides should be taken at regular intervals three or four times a day.

The writer wishes he could share the optimism of others with regard to the efficacy of bromides. Authorities are agreed that idiopathic epilepsy is curable in only 10 per cent. of cases, and that only those who have been free from fits for at least ten years can be pronounced cured. As has been mentioned, intervals of as many as thirty years may occur between attacks whether bromides have been taken or not. We are all familiar with the epileptic who says he always has a fit when he tries to do without the medicine. On the other hand, the faith of patients who have imputed their lengthy immunity to gallons of medicine religiously consumed is sometimes rudely shaken by a fit.

In regard to such substitutes for bromides as *adonis vernalis*, *solanum carolinense*, tincture of *simula*, each has its day, and is useful, probably, in giving the patient a respite from bromidism.

Operative treatment should always be advised when epilepsy is directly traceable to traumatism, and signs of injury to the skull are evident. Operation is again indicated in cases of *status epilepticus*, in which the patient is incapacitated by forty or fifty or more fits in a day. As already mentioned, localized meningitis *serosa externa* may be the cause. A number of such cases in which the subarach-

told systems have been opened and drained, with great relief to the patient, has recently been recorded. The statement, however, that "about 8 per cent. of all cases of epilepsy are amenable to surgical treatment" is perhaps unduly favourable.

5. HEADACHES AND MIGRAINE.

Headaches.—Apart from those caused by tumours and other forms of gross cerebral or meningeal disease, persistent or recurring headaches may be due to local causes, such as hypermetropia, astigmatism, strain of accommodation, asthenopia; nasal or naso-pharyngeal obstruction and catarrh; diseases of the middle ear or auditory meatus; and decayed teeth.

The chief general causes are bad atmospheric and noisy surroundings at home or at school; anæmia, dyspepsia, constipation, rheumatism, and uræmia. But many headaches are purely of nervous or emotional origin.

The locality of headaches is not of great diagnostic value. Bitemporal or frontal headache is frequently due to hypermetropic astigmatism. Occipital headaches may sometimes be traced to decayed molars. Headache due to disease of the nose or nasal sinuses is usually frontal or supra-orbital. Headaches proceeding from the other causes mentioned may affect any site. The "casque" headache of neurasthenia and hysteria is rare before puberty.

Migraine (Hemicrania).—The headache in migraine is characterized by its excruciating severity and by the physical prostration which accompanies it. Other headaches, with the exception of those due to tumour, abscess, and meningitis, are seldom so severe, but are more frequent and lasting than those of migraine. In migraine the headache is not invariably unilateral.

Migraine is distinguished from other forms of headache by the peculiar disorders of vision and sensory disturbance which precede it. Hemianopsia, macropsia, or micropsia, are common. Stationary objects may look steamy and misty and in movement, or like an unfinished "jig-saw" puzzle. Dark clouds with luminous borders, flickering lights, and "fortification spectra," may be seen. There may be hallucinations of hearing, taste, and smell. Numbness and tingling of the fingers, usually on one side, may occur, and articulation may be difficult. All these premonitory symptoms or "aura" may perplex and alarm the child, and, as already mentioned, they may explain some cases of day and night terrors in young children, in whom as a rule migraine is not suspected. Typical attacks of migraine, however, are rare under the age of puberty.

The prodromata may last from a few moments to half an hour, and subside as the pain comes on. The headache increases in intensity for several hours, until at its height the patient lies groaning and whimpering, protesting against the slightest sound, and hiding his face from the light. He will not speak above a whisper, and sometimes not at all, so that he may be supposed to be unconscious. But he never is unconscious, nor does he shriek with pain and grate his teeth as in meningitis. The extremities are often cold, and the patient may shiver. The pulse is weak and slow, or hard, small, and quick; the features are pale and haggard in some, flushed, with injection of the conjunctivæ and throbbing of the carotids, in others. Nausea and refusal of food are common, but vomiting is infrequent except towards the end of an attack. In an hour or two the acute symptoms gradually subside, the

patient usually falls asleep, and wakes hungry, bright, and well. He remains exempt from recurrence, which may be at weekly or monthly or longer intervals.

In some cases transient paresis of the third or some other ocular nerve follows each attack of migraine (*migraine ophthalmoparalytique*). In one case known to the writer, permanent paresis of the ocular sympathetic on the right side appears to have been the result of repeated migraine.

Migraine rarely affects general health; sufferers are often physically and mentally active. It is certainly a family affection and one of the neuropathic inheritances. Some regard migraine as a mark of the "superior degenerate," but in most cases of true migraine intellectual superiority is more apparent than degeneracy.

The pathology of migraine is unsettled; but whatever may be the exciting cause, the symptoms are certainly associated with vasomotor or sympathetic disturbances, and varying degrees of local cerebral anemia and congestion will account for them. The posterior part of the pituitary body may be at fault, for this organ exercises powerful control over blood-pressure in general, and over the cerebral circulation in particular (De Bert Fischer, Wiesbaden).

Spitzer attributes migraine to temporary internal hydrocephalus due to congenital narrowing of the foramina of Monro, and consequent intermittent pressure on the choroid plexuses. Were this so, acquired and permanent internal hydrocephalus would be a more common disease than it is, considering the relative frequency of migraine.

The more probable view is that migraine depends on the accumulation of toxic materials in the blood which irritate the sympathetic system. The nature of the toxic materials is unknown; probably it varies in individuals. In some the "purin" bodies, or other common articles of diet, seem to act as poisons. In others no special cause can be ascertained. To speak of migraine as a "nerve storm" does not convey much meaning. It has been suggested that migraine and epilepsy are closely allied, but the association of the two disorders is certainly very rare.

Headaches due to Defective Eyesight.—Hypermetropia, myopia, and astigmatism, are well-known causes of headache, photophobia, lachrymation, and conjunctivitis, produced by spasm of the ciliary muscles and fatigue of the internal recti. The symptoms are relieved by atropin, and by supplying suitable glasses when major degrees of ametropia are present. But similar symptoms in using the eyes for close work may occur in neuroathenic patients whose refraction is apparently normal. The condition is one of asthenopia, which is but part of general neurasthenia. It is true that certain enthusiastic oculists claim that all the symptoms of neurasthenia are due to uncorrected ametropia, and can be cured by correcting infinitesimal errors of refraction. The writer is unable to share this optimistic view. At the same time, rest for the eyes is as important for neurasthenics as rest for the brain and other parts, and glasses which reduce the necessity for accommodation in asthenopia may be beneficial. Yet glasses are but a *pis aller* in the case of the neurotic scholar child who is incapacitated by headache directly he uses his eyes, although his vision be normal. Rest from his labours will cure his headaches, and his asthenopia too.

As regards migraine, one rarely meets a sufferer who is not wearing spectacles or pince-nez, or one who has been cured by wearing either.

Headaches due to dyspepsia, anemia, constipation, rheumatism, need treatment on ordinary principles. Rheumatic headaches are usually relieved by aspirin or salicylate of sodium. An examination of the eyes for errors of refraction and

optic neuritis, and of the urine for albumin, should never be omitted. Other local causes, such as decayed teeth, otitis media, naso-pharyngeal obstruction, and disease of fronto-nasal sinuses, should be borne in mind as possible causes. Faulty hygiene of sleeping- and class-rooms is sometimes responsible. If the conditions cannot be improved, the patient should be removed from them. Headaches traceable to educational overpressure, worry, discontent, and unhappiness, in neurotic children, cannot be relieved by drugs or by any treatment short of alleviating or changing the circumstances.

Migraine seems to be less dependent on definite ill-health, unsuitable modes of life, and local causes, than are other forms of headache. Subjects predisposed to migraine are as liable to attacks in the playing-field as in the school-room. Sedentary brain workers often have their worst attacks during holidays, when open air and exercise have made them feel robust and well. It may be that exercise in such cases floods the circulation with waste products and with toxins which have accumulated in the intestinal tract. On the other hand, migraine is sometimes traceable directly to mental work, vitiated air, want of exercise, constipation, and unwholesome diet, and may be cured or greatly lessened by attention to such details.

TREATMENT.—Treatment should aim at elimination of deleterious substances from the body and limitation of their supply. This may be attained by giving an occasional saline purge and by regulating diet and mode of life. Possibly, too, irritability and morbid reaction of the sympathetic, on which the phenomena of migraine appear to depend, may be lessened by certain drugs. When pallor and cordlike contraction of arterioles prevail, the nitrites, especially liq. trinitrin., in doses of $\frac{1}{4}$ to 1 minim. may be given in the intervals between attacks. When there is flushing, throbbing of cervical and temporal arteries, such drugs as strychnine and ergot are beneficial. When opposite conditions of the sympathetic—namely, constriction and dilatation—are present at the same time, liq. trinitrin. may be combined with ergot and strychnine. Liq. strychnine (1 to 2 minims) with ext. ergotæ liq. (10 minims) may be given to a child twelve years of age three times daily. In some cases quinine with hydrobromic acid and nux vomica is of service.

In highly emotional children with rheumatic symptoms, salicylate of soda, with bicarbonate of ammonium, sal volatile, and inf. gent. co., should be administered. Citrate of iron and ammonium agrees as well as any patent preparation of iron, and may be given with ammon. brom., nux vomica, or arsenic, when anæmia is present. It should be remembered that quinine, iron, and nux vomica, may give rise to headaches, particularly in neuroathetic persons, who often show marked susceptibility to their action.

The bowels should in all cases be carefully regulated. Fresh air, moderate exercise, and healthy surroundings, are essential. The diet should be plain, not over-abundant. Excess of sugar and starch in some cases, and of nitrogenous food in others, seem to increase proclivity to migraine.

During an actual attack of migraine, the patient should be kept warm, reclining in a quiet, shaded room. The less he is disturbed by pressing food and medicine upon him the better. All sufferers from migraine agree that, once an attack has commenced, nothing can stave it off. But some relief may be obtained by a dose of antipyrin or phenacetin with citrate of caffeine when given at the onset. Pulv. guarana, in 12-grain doses, for a child of twelve has been recommended, but a cup of tea is perhaps as efficacious.

6. ENURESIS.

Enuresis as a neurosis seems to be the result of super-sensitiveness of the lumbar centres of micturition, and of deficient inhibitory control on the part of the higher cerebral cortex over the lower centres in the spinal cord. It is one of the indications of general nervous instability, and may be congenital, hereditary, and familial, or acquired. When nocturnal only, enuresis may be due to unsuspected epilepsy.

Ætiology.—Cases in which nervous instability is the sole cause are the most intractable, but in the great majority some exciting cause is present as well. In a few, want of proper training may keep up the habits of infancy. Every child has to be taught to control its bladder and make known its desire to empty it. It usually learns to do so before the age of two years. If incontinence is habitual after the age of three years, it is a morbid manifestation. When constant day and night, it is usually associated with some form of mental enfeeblement or with spina-bifida occulta, in which case it is incurable. It may be a symptom of pressure on the cord by tuberculous or other disease in its neighbourhood.

The chief exciting causes of enuresis, when not of purely nervous origin, are—Adenoid vegetations; local conditions within and without the bladder which render it and the spinal centres irritable; constitutional defects, such as diabetes mellitus and insipidus; and, far more rarely, chronic interstitial nephritis. Chorea is a common cause of inability to control the bladder.

Adenoid vegetations are a cause of nocturnal enuresis, but probably never give rise to diurnal incontinence. Enuresis may be a part of, or the result of, night terrors, which are common in the subjects of adenoid vegetations. But night terrors in such cases are probably set up by partial asphyxia, which stimulates the respiratory centres and vasomotor centres in the medulla. Thus, an increased flow of urine takes place into the bladder, which empties itself because the cerebral centres of control are inhibited by fright.

Causes of Irritation within the Bladder.—Stones, cystitis from tuberculous disease, and new growths, need only be mentioned here as possible causes. Cystitis due to infection of the urinary tract by micro-organisms, especially by *B. coli*, is not an uncommon cause of enuresis, nocturnal and diurnal, and of frequent and precipitate micturition (pollakiuria—*cf.* article Enuresis, p. 533).

The urine infected with *B. coli* is highly acid, specific gravity, 1010 to 1020, clear on passing, but on standing becomes neutral or alkaline and turbid, the turbidity being unaffected by heat or reagents. It has a characteristic offensive odour, and stains napkins or underclothing yellowish-brown. Pus cells and a trace of albumin may be found. On centrifugalizing and staining the deposit with methylene blue, the bacilli are seen with an oil-immersion lens.

The **TREATMENT** consists chiefly in rendering the urine alkaline by means of increased doses of citrate and acetate of potash. Five grains of each may be given every four hours, and the amount increased by 20 grains daily until 160 to 180 grains are taken in twenty-four hours. The alkaline treatment should be continued for several weeks, as cessation is often followed by renewal of the symptoms. The addition of urotropin in 5 to 10 grain doses often seems to be of service, although *B. coli* is said to flourish in urotropinized urine. Benzoate of sodium and of

ammonium in doses of 5 to 10 grains are sometimes useful. Hyoscyamus in 10 to 15 minim doses allays vesical irritability. To prevent reinfection, it is desirable to attack the *B. coli* at their source in the bowel. Constipation should be treated, and the rectum washed out with boric or saline solution. The usefulness of salol and other so-called "intestinal disinfectants" is problematical. Vaccine-therapy is unnecessary in uncomplicated *B. coli* infection, but when pyuria with pyrexia and evidence of pyelo-cystitis is present, a vaccine should be prepared from a pure culture of the organism isolated from the patient's own urine. Three millions is a suitable dose for a child one year old, and 25,000,000 for an adult. The dose should be repeated in two days' time, and the interval gradually extended in accordance with the patient's progress.

Hyperacidity of the urine, with deposit of urates, uric acid, or oxalates, is also associated with nocturnal and diurnal enuresis, especially in nervous or rheumatic subjects. Restriction in nitrogenous food, and administration of citrate of potash, is often efficacious in such cases. Salicylate of soda sometimes causes painful and frequent micturition.

Enuresis may also be caused by affinity of the urine without cystitis. The urine is of low specific gravity, 1000 to 1005; it may contain a trace of albumin. Polyuria is usually present, and a characteristic feature is that the child, on being waked at night to pass urine, does so in considerable quantities, but after a short interval is found to have passed as much again in bed.

In some cases excess of saccharine or farinaceous food seems to be the cause of this form of enuresis, for it may be cured by supplying a rigid antidiabetic diet for a few days (Percy Lewis). Polyuria, whether due to diabetes mellitus or inappetens, or to chronic interstitial nephritis, is in itself sufficient to cause enuresis.

Irritability of the bladder may still produce incontinence night and day, although the original cause of irritation has been treated or removed. The presence of even small quantities of normal urine in the bladder is sufficient to excite it to contract. At short intervals there is a sudden call to micturate, which must be obeyed at once.

The treatment of an irritable bladder, which will contain only small quantities of urine, consists in training it to hold more. The child should be kept in bed, and instructed to micturate at intervals gradually increased. Diet should be bland, and excess of farinaceous, saccharine, and nitrogenous articles should be avoided. Water or barley-water may be given freely. It is a mistake in any case of enuresis to restrict fluids except shortly before bedtime. Tincture of hyoscyamus in doses of 10 to 15 minims or more is a valuable vesical sedative. It may be given with citrate of potash and infusion of buchu.

Atony of the sphincter is not a common condition, but occasionally causes incontinence. The urine drips or trickles away without frequent and distressing desire to pass it. Such cases in both sexes may sometimes be cured by passage of a sound. Electricity in the form of faradism, one pole being placed on the perineum or in the urethra, and the other on the back or suprapubic region, has been recommended. Direct massage of the neck of the bladder by means of a finger introduced into the rectum has been advocated. There are obvious objections to the method.

Treatment by epidural injections of saline solutions has been practised in France and America. It is supposed to act by irritating the cauda equina, and thus conveying impulses to the limbic centres, which are thought to be incited

thereby to exert tonic or inhibitory influences over the bladder. The site of the injection is the membrane closing the lower end of the sacral canal, which is indicated by a triangular depression situated at the posterior termination of the trianglular fossa. Injections of 1½ to 5½ or more drachms of normal saline sterilized solution are made with an ordinary syringe, having a needle 1½ inches in length. The needle is inserted at the point of election, and thrust forwards and upwards into the sacral canal. The patient stands or lies on one side with thighs flexed. No anæsthetic is used. It is admitted that the method is by no means uniformly curative; its effects, if any, are probably psychical.

In inveterate cases of irritable contracted bladder, gradual expansion of the organ by injection of weak boric solutions, combined with local application of electricity, is said to be successful.

Hypnotic suggestion is a very few carefully selected cases of enuresis of purely nervous origin may be recommended.

Cause of Irritation outside the Bladder.—Of these, phimosis, balanitis, and vulvitis, may be mentioned; but in some cases the incontinence may be the cause, and not the result, of irritation. Oxyurias and constipation may sometimes give rise to enuresis.

GENERAL PRINCIPLES OF TREATMENT.—Whatever may be the cause, the inconvenience occasioned by nocturnal enuresis may be lessened by waking the child at set intervals to pass water. Accidents commonly happen when the child lies on his back; therefore he should be taught to sleep on one side. A "bobbin," or some hard object strapped to the small of the back, will wake him should he turn over on it. Restriction of fluids just before bedtime, and of stimulant diuretics, such as tea, coffee, mustard, pepper, spices, meat, and meat juices, and in some cases sugar and starch, should be enjoined. Enuresis when a part of night terrors needs treatment appropriate to the latter (q.v., p. 633).

Punishment of any kind is rightly condemned in any case of enuresis occurring in a child upwards of three or four years of age; but in younger children, particularly those who are wilful or lazy, one or two slappings will often break the habit. Medical men are not usually consulted until this primitive method of treatment has failed. Rewards for not wetting the bed will sometimes stimulate powers of control over the bladder.

Children who invariably wet the bed should not be sent to a boarding school, and inveterate cases should be removed to a nursing home or hospital, where serious attempts made to train them to overcome the habit are often successful when home treatment has failed. Constipation should be relieved. Evacuation of the bowels in the evening before bedtime will sometimes cure nocturnal enuresis.

The utility of some drugs has already been mentioned. It would be impossible to enumerate all the drugs which have been found invaluable by some and useless by other practitioners. Consideration of the various causes of the complaint will suggest appropriate remedies.

Atropine and belladonna should not be given by routine in every case. Children who wet the bed occasionally should not be poisoned indiscriminately with belladonna for months together. The physiological action of belladonna in full doses is to cause temporary paralysis of the bladder and to lessen secretion of urine. In some cases such results are beneficial, in others not. Belladonna is useless when sphincter control is weak, also in cases in which the tendency to enuresis is kept up by local and constitutional conditions. It is seldom useful when enuresis is

both diurnal and nocturnal. It is useless in *B. coli* infection. It is most valuable in cases of habitual nocturnal enuresis. The drug should then be pushed to full doses (10 to 40 minims of the tincture for a child of ten to twelve), and be given at night only, with 10 grains of one of the bromides; or atropine may be given in 1-minim doses of the liq. atropine sulph., 4 grains to 1 ounce, gradually increased until the physiological effect is produced. If no good effects follow a few weeks of belladonna treatment, the utility of its continuance is doubtful. The disadvantages of keeping a child for months in a state of chronic belladonna intoxication is obvious. Few parents can be induced to persevere with the treatment after one or two experiences of children rendered half blind, staggering, delirious, and unable to swallow, in consequence. The ill effects can, however, be reduced by giving belladonna or atropine at night only. The writer has found better results from giving hyoscyamus, citrate of potash, and buckwheat, than from the belladonna treatment alone; but in some such cases it is probable that he was treating a *B. coli* infection unawares.

Cantharides has been recommended when there is weakness of the sphincter. Lycopodium, in doses up to a drachm of the tincture, may be useful as a bladder sedative. Ergot in atony of the bladder, antipyrin as a general nerve sedative, valerianate of zinc in $\frac{1}{2}$ to $\frac{3}{4}$ grain doses, rhiz. aromatica in doses of 10 to 15 minims of the liquid extract, as a stimulant, diuretic, and tonic, may all be useful when the conditions which indicate their use are present. Arsenic, strychnine, and ox. veronica and iron, may be required. In a few instances, where there is mental and physical evidence of thyroid insufficiency, thyroid extract may be beneficial.

Nervous Retention of Urine is not uncommon in boys. The writer has known it to occur in father and son. The father throughout his life could never use a public urinal, nor pass water if anyone was by. The son suffered much at school from the thoughtless teasing and practical jokes to which he was subjected by his school-fellows on account of a similar failing. Fortunately, no further ill consequences ensued; but in such cases, as the affection cannot be overcome by drugs or strength of will, common-sense instruction should be given, in order to enable the patient to conceal it. Hysterical retention of urine in young girls never needs active treatment by passage of a catheter.

Incontinence of Faeces.—Inability to control fecal evacuations may be the result of paraplegia due to myelitis, injury to the lumbar portion of the spinal cord, or disease of corresponding lumbar vertebrae or spina bifida. Incontinence of faeces is a common result of stuporous conditions and exhaustion in the course of any prostrating illness. In severe cases of chorea both bladder and rectum may be incontinent. Occasional incontinence of urine and faeces may be evidence of unsuspected epilepsy. Idiots of the lowest grade cannot be taught to control their sphincters, but incontinence in this particular can hardly be described as incontinence. Apart from these causes of fecal incontinence, atony of the anal sphincter may render it so weak that it cannot resist the slightest impulse to empty the rectum. The condition is analogous to atony of the sphincter vesicae; the anus is patulous, and little or no contraction of the sphincter ani occurs on introduction of the finger. Sometimes atony of the sphincter ani and fecal incontinence are associated with polypus or prolapse of the rectum.

Most frequently, however, incontinence of faeces is the consequence of the diarrhoea of purely nervous origin which besets those of neurotic temperament during any period of emotional distress or anxiety throughout their lives. It is

attributable to disturbance of the abdominal sympathetic system. Nervous diarrhœa and incontinence are common in children who worry over lessons and babble of them during sleep. They will eat but little breakfast, and diarrhœa, and sometimes vomiting, occur directly afterwards; they hurry lest they should be late for school, and sail themselves on the way or when they get there. Incontinence in such cases is, of course, only a natural consequence of nervous diarrhœa. Treatment consists in rendering school life less formidable to neurotic children, or in keeping them at home. In some instances solid excrement may be voided into the clothing. Mental deficiency, or epilepsy, or an obscure form of psychopathia, may in some cases be the cause.

Hysterical retention of feces arising from mysophœbia has been occasionally recorded in young children (Thienisch).

7. HYSTERIA.

Schneider regards hysteria as the persistence in adults of the childish type of reaction to the facts of life, and his view is consistent in many ways with other modern hypotheses as to the nature of hysteria. Janet, for instance, traces all the phenomena of hysteria—mental and physical—to "restriction of the field of personal consciousness." On the mental side the hysterical person is wayward, capricious, fickle, unreasonable, impulsive, because, owing to amnesia of former impressions and events, she is unable to guide and govern action, to balance motives, and the consequence of yielding to them, in the light of past experience. The idea of the moment becomes a fixed obsession, and reigns supreme, unrestrained by the simultaneous presence of other ideas which should counteract or modify it. If a desire cannot be gratified at once, the consequences are emotional outbursts and actions contrary to decorum and common-sense. Such restriction in the mental field of judgment, criticism, analysis, and memory, places the hysterical person precisely in the position of the spoiled and passionate neurotic child, whose experience of life is too short to teach him that he cannot always have his own way.

On the *physical side* there is similar restriction of the field of personal consciousness. Just as the field of vision in hysteria is restricted so that the patient does not perceive objects at the periphery, so also there is similar restriction in regard to perception of other peripheral stimuli—hence *anæsthesia*, loss of muscular sense, and *pseudo-paralysis*. Only a certain number of the multiple sensations and stimuli which make up personal consciousness are assimilated and responded to.

If, as Babinski holds, "suggestibility and persuasibility (*Pithiabilité*) are the criteria of hysteria," all children are hysterical, for the greater part of training and education in early life depends upon the child's suggestibility. Owing to lack of experience and to its simplicity, the normal child accepts suggestions without demur and believes all that it is told. Suggestion and counter-suggestion or persuasion are in constant use in every nursery. Were not normal children amenable to persuasion, not even the most Spartan of methods could tame them. But, as a matter of fact, hysterical adults and neurotic children are singularly obdurate to suggestion and persuasion. They are notoriously difficult to hypnotize, and their suggestibility chiefly applies to suggestions made by themselves. Hysterical affections can be cured by persuasion, but the difficulty in curing them lies in the

selection of the method of persuasion adopted and of the person who exercises it. This same difficulty arises in the case of neurotic children. A fall on the foot suggests to one child that it has hurt itself, but it is speedily cured by the counter-suggestion of "kissing the place to make it well"; whereas another child who meets with a similar accident requires all the counter-suggestion and persuasion that parents, nurses, and physicians, can devise to convince it that the injury is not incapacitating and lasting. Suggestibility and persuasibleness are indeed characteristic of hysteria, but the terms can only be used paradoxically. Hysterical adults, and children, too, are only amenable to abnormal forms of suggestion and abnormal methods of persuasion.

The hypotheses of Freud and Breuer again support Schreyer's contention, that hysteria is a persistence or recrudescence of puerile reactions to the facts of life. They trace the origin of hysteria to events occurring in very early childhood. Stated briefly, the essentials of Freud's hypothesis are—

1. The occurrence of "conflicts" between desires or impulses and the moral censure which forbids their consummation, or even the entertainment of the idea of yielding to them.

2. The result of such "conflicts" is that a "complex," or system of ideas having an "emotional tone" and a conative trend towards thought or action in some definite direction, becomes suppressed or banished from the conscious mind (repression).

3. The repressed complex, which may be either a spontaneous thought or based upon an actual incident—usually of a disagreeable or revolting character—and is invariably related to sexual instincts, becomes buried in the subconscious mind.

4. But though forgotten, the "complex" remains like a foreign body or an impacted wisdom-tooth in the subconscious mind, and asserts itself in various hysterical symptoms, such as emotional outbursts, tremors, convulsions, anaesthesia, paralysis and contraction of limbs, and in various painful subjective sensations which have no obvious relation to their hidden cause.

5. The different modes in which a repressed complex finds vent, or "evasion of the censure" is practised, are called "transference," "symbolization," and "displacement."

The censure, for instance, of a suppressed illicit and erotic desire may be evaded by lavishing undue affection on cats and canaries (transference), or the desire may be "symbolized" in an exaggerated interest in marriages, births, and scandals. Such interpretations of a celibate's fondness for animals, and interest in ordinary daily events, seem to be unnecessarily harsh. In "displacement" the censured complex finds expression in some subjective sensation which is not under the ban of the "censure." For instance, the pangs of unrequited love may find expression in some subjective sensation remotely associated with the object of affection—e.g., a haunting odour of a cigar.

Postponing criticism for the present on Freud's views, which have been given in barest outline, attention may be drawn to their consistency with what we know of the operation of the childish mind. Emotional conflicts do take place in early life, especially in children of the "restrained neurotic type." In our own childhood they were called "struggles against temptation," the "conative trend" being, in books of the period, generally towards jam. Suppression of the complex was a moral triumph, and the end to the victor in the shape of subsequent hysteria was not contemplated. The example of a repressed complex

given seems trivial, but other more formidable and yet analogous instances may be adduced. Suppressed fear may dominate the whole of a neurotic child's existence: fear of animate or inanimate things, fear of darkness, of the supernatural or of particular persons, dread of misdeed to others or to himself, dread of doing wrong, fear of punishment or of being past forgiveness. The child may know that his fears are unwarrantable, and despise himself for yielding to them. He would die rather than concede to them, and succeeds by prodigious efforts in suppressing obvious manifestation of his secret troubles.

Other repressed complexes may be anger, jealousy, desire for revenge, sense of injustice, disappointed ambition, self-depreciation. Such pent-up emotions in childhood find expression in bursts of tears, fury, or spitefulness and cruelty, without apparent cause.

Sullen moroseness, callousness, moping, unsociableness, "dromomania," may be evidence of suppressed complexes. Curious outbreaks of insubordination, immorality, or dishonesty, untruthfulness, unfounded accusations against friends and companions, immodesty, neglect of personal cleanliness, self-inflicted injuries, are other methods by which a repressed complex manifests itself. All these phases of hysteria are common enough at or about the age of puberty.

In younger children pent-up emotions may be said to display themselves in such complaints as night tremors, vomiting, indigestion, anorexia, henteric diarrhoea, exsthesia, cardiac palpitation, pallor, flushing, fainting, and convulsive disorders.

The process of "symbolization" as a means of "evading the censure" is characteristic of childhood. Maggie Tulliver hammering nails into her wooden doll's head, and grinding it against the wall, to express her animosity towards her disagreeable aunt, and young Rawdon Crawley squaring up to Lord Steyne's hat in the hall, are familiar examples of symbolization in fiction, and are true to Nature too.

Transference, exemplified in the spinster's devotion to pets, which Freud unkindly attributes to unsated sexual desires, may be seen any day in the child who treats her doll as a baby, and exercises stern discipline over her younger brothers and sisters, in obedience to maternal instincts.

In short, whether we accept "restriction of personal consciousness" (Janet), or "Pithiatisme" (Babinski), or "repression of emotional complexes" (Freud), as the foundation of hysteria, we find that all these conditions are present and normal to a certain extent in early life.

Hysteria is shown in exaggeration and abnormal exercise of normal functions. The difference between the normal and neurotic individual in this respect is mainly one of degree. Without restriction of personal consciousness—or the power of disregarding counteracting stimuli—habits of attention and concentration would be impossible. Yet reverie, concentration and absorption in occupation, are only one step removed from dream states, automatism, loss of memory and identity, which are manifestations of hysteria.

The various methods of ventilating repressed emotional complexes are rational, or may be morbid and hysterical in accordance with their vehemence, eccentricity, and inappropriateness. Ill treatment of a doll, for instance, is a harmless outlet for a personal grievance compared with cruelty to a pet animal or to younger children. Any normal child may sulk if slighted, but the hysterical child becomes melancholic, not merely sulky. The normal child seeks affection by bestowing it in moderation upon others—the hysterical by extravagant demonstrations, or by

leaving illness and injury, or perhaps by buying gifts for others with stolen money. Much of the misconduct and startling immaturity of neurotic children of both sexes must be regarded as evidence of repressed emotional complexes seeking an outlet. The seeds of hysteria are sown in early life, and Freud's insistence on this fact is of the utmost importance. Recognition of "repressed complexes" in young children, and appropriate management, may save them from hysteria and many other functional nervous disorders.

Freud's views are, however, open to criticism in certain points. It seems unnecessary to assume that a suppressed complex which asserts itself in hysterical manifestations is banished from the conscious mind. Most people remember their emotional conflicts, however much they strive to forget them. If a disagreeable incident or complex of ideas is actually forgotten, and only elicited by psycho-analytic methods, it is possible that it has nothing to do with the symptoms, although the patient may readily accept the psycho-analyst's assurance that it is their cause. Evidence of the nature of a repressed complex rests entirely on the patient's statements. Many are not disposed to agree with Freud that a repressed complex is invariably of sexual nature. It is true that hysteria and a variety of functional neuroses may result from the patient's conviction that secret sexual offences have ruined him body and soul. But patients are seldom reticent on this point, and when a tedious process of psycho-analysis is necessary to elicit confession, it is highly probable that the "suppressed complex" is in reality suggested by the inquirer.

Freud soon abandoned his original contention that hysteria is the out come of actual sexual assaults experienced by little children, and has elaborated a theory of hysteria based upon the evolution of the sexual instincts in infancy and early life. He maintains that stages of auto-eroticism and homo-sexualism are normal in infancy, and depend on the process of development of the oral, anal, cutaneous, and genital erogenous zones respectively. Normally the genital zone proper becomes predominant, whilst the remaining erogenous zones are subsidiary and subordinate. The energy belonging to the repressed sexual impulses derived from zones other than the genital is diverted into non-sexual channels by a process called "sublimation," by which is meant the educational development of social charms and graces. The evolution of normal sexual activity takes place between the ages of five to ten years, and should be complete at puberty. But if during the prepubertal period some erogenous zone, such as the oral or anal, becomes *over-developed* at the expense of the genital zone proper, tendencies to sexual perversion occur. Impulses towards them may be suppressed instead of being "sublimated" into other forms of activity, and find expression in various manifestations of hysteria. It is, however, impossible to admit that all cases of hysteria are founded on "psychopathia sexualis." It is well known that profound hysteria may be the immediate result of trauma or emotional shock of any kind, and that any sudden emotional shock may cure it. Both facts are incompatible with Freud's views.

Freud and his school point with triumph to the success of their treatment in proof of the correctness of their theories. But similar successes may be claimed by the exponents of any method of treatment, provided that it is based on "suggestion" or on the application of some unwonted and unexpected stimulus. Ancient physicians, who believed that hysteria was due to wanderings of the womb, no doubt cured many patients by persuading them that they had succeeded in tethering

the organ. Could the patient be persuaded that hysterical symptoms are due to a neglected corn on his toe, the chiropodist could vie with the psychotherapist who attacks a corn in his patient's mind.

The secret of curing hysteria lies in supplying suddenly, or otherwise, extraordinary stimuli to take the place of normal stimuli, which are unappreciated by the patient because, as Janet holds, his personal consciousness is restricted. The efficacy of the stimuli may depend on their novelty, unexpectedness, the *entraineement* of their application, and, above all, on the personality of the physician or quack who applies them. According to Boerhaave, an Arabian physician once cured a Caliph's young wife, who was suffering from hysterical hemiplegia, by suddenly offering a gross affront to her modesty. A single shock from a ten-and-sixpenny battery will cure the simple village maiden of hysterical motion, paralysis, or amblyopia, whereas a Wimshurst machine, with a plate as large as a cart-wheel yielding a 6-inch spark, may fail to relieve the highly educated person similarly affected, unless she credits the man behind the machine with occult and supernatural powers. The stimulus must be sufficient to open the floodgates of the sympathetic, and send the blood coursing through the body, brain, and limbs. In some the effect may be produced by one or two pails of cold water or the application of a faradic wire brush; others require the elaborate and impressive ceremonial provided at shrines, grottoes, or music-halls.

In the writer's opinion, hysterical symptoms cannot be imputed to restriction of personal consciousness, suggestibility, imitation, or to manifestations of "repressed complexes" alone, though all these conditions in an exaggerated and abnormal form are characteristic of hysteria, and are as such the essence of the neurotic temperament. By the neurotic temperament is meant individual peculiarities in regard to perception of, storage of, and reaction to, the varied stimuli on which conscious personality depends. Hyperaesthesia, anaesthesia, dysaesthesia, and symaesthesia, in respect to stimuli in various alternations and combinations, may account for hysteria, neurasthenia, and functional neuroses in general. Volition, attention, the emotions, and all intellectual processes, have a physical basis in bodily changes induced by stimuli, and these changes are chiefly brought about by the action of the sympathetic nervous system. Anomalous conditions of the sympathetic in the direction of excess or defect of action in response to stimulation account for the subjective sensations as well as the objective physical signs which are not under direct control, and are therefore misinterpreted by the mind of neurotic subjects. Delusions, illusions, and hallucinations, are the result of misinterpreted stimuli.

No broad gulf separates hysteria and neurasthenia. Neurotics of either type are liable to both, but on the whole the "restrained" type is more apt to develop hysteria, and the "unrestrained" is more prone to neurasthenia. In neurasthenia there is super-sensitiveness with excessive reaction to all kinds of sensations and stimuli; in hysteria hyperaesthesia and excessive reaction occur in regard to some particular sensations and stimuli, whilst others are unappreciated and give rise to no reactions.

Hysterical Paralysis and Contractures.—Children before the advent of puberty do not exhibit such stigmata of hysteria as anaesthesia, restriction of visual fields, and hysterio-genetic zones. Babinski maintains that these conditions do not exist even in adult hysterical patients, unless their presence is suggested by medical men. This may be true in respect to the hysterio-genetic zones of Charcot, but it

is certain that hysterical patients often discover spontaneously that their field of vision is restricted, and when unaware of the fact they cannot know by intuition that it will be revealed by the perimeter. The chart of the visual fields characteristic of hysteria cannot be the result of suggestion. Anaesthesia no doubt can be induced by suggestion, but the patient often discovers it for herself, and reveals it to others by turning her limbs into pin-cushions and needle-cases. The absence of anaesthesia in infantile hysteria has been attributed to the simplicity of the child mind. The hysterical adult, it is alleged, argues that if a limb is paralyzed it must be dead, and therefore insensible, whereas the child is incapable of such feats of ratiocination, and is only aware that it cannot move its leg or arm. It seems more probable that the anaesthesia precedes the imaginary paralysis, and that the adult argues (if she does argue) that the limb is powerless because anaesthetic. It is anaesthetic and apparently powerless because of the state of partial ischaemia which prevails. Hysterical palsy is, in fact, a form of ischaemic paralysis dependent upon overaction of the sympathetic vaso-constrictor system. The evidence of this is that the limb does not bleed when pricked. In the neurotic child, however, the sympathetic system, although unstable and irritable, has not acquired the extraordinary super-sensitiveness and hair-trigger-like action which is developed in the hysterical adult. A similar condition of ischaemia probably occurs in the child, but it is insufficient to produce bloodlessness and anaesthesia, though enough to cause the limb to "go to sleep."

Hysterical paralysis may be associated with flaccidity or rigidity. The onset may be sudden or gradual. The type of paralysis may be monoplegic, diplegic, or paraplegic. The flaccid variety may affect one or both legs. Under passive movements, if the child's attention can be disengaged or if it is taken by surprise, the muscles can be felt to contract, thus showing that they are not paralyzed. Sometimes all voluntary movements are freely performed when the patient is lying down, but a condition of "astasia abasia" is present. If the child is placed in erect position, it treads on the inner or outer side, or even the dorsum, of the feet, and at once collapses on the ground if unsupported. When one lower limb only is affected, the patient will not rest upon it, but trails it like a dead weight behind it, with the toes or dorsum of the foot scraping the floor. In less pronounced cases many curious methods of progression are adopted. A girl aged ten, for instance, walked exactly like a tight-rope dancer, sliding her feet in a line in front of each other, swaying her body and waving her arms to preserve her balance, an expression of intense anxiety on her flushed face. A day or two later she could not walk across the room, but got to the other side by hugging the walls all round, or made short tottering charges from point to point, seizing each article of furniture on the way, apparently just in time to save her from a heavy fall. In a short time she imitated in an exaggerated fashion the "cross-legged" gait of another child suffering from congenital spastic paraplegia. Another girl, aged seven or eight, behaved precisely as if wearing skates on the ice for the first time, slipping and sliding her feet away from each other, treading on her outer or inner ankles, falling in all directions, yet never seeming to hurt herself.

When rigidity is present, there is almost always complaint of pain. The knee is most commonly affected. It is kept rigidly extended or semiflexed, and all attempts to move it are met with resistance and piercing shrieks. In advanced cases the thighs are firmly flexed on the abdomen, the legs on the thighs, and the feet extended so that the soles fit the curves of the buttocks. The attitude may

be maintained for months together. At first it may be relaxed during sleep, but in time it may become constant. The skin may slough in the flexures of the joints and cause foul, unhealthy ulcers. The degree of contracture is far more extreme than is ever seen in any known organic disease. It is difficult to explain on any hypothesis, but the writer is inclined to believe that it originates, like the ties, in an "act of defence." It resembles the almost involuntary drawing up of the whole limb, which follows dipping one's toes into unexpectedly hot or cold water. Possibly some painful or disagreeable sensation in the sole of the foot suggests it. Whether flaccidity or rigidity be present, the paralysis or contracture is probably suggested by pain of one kind or another.

In hysterical contracture of the upper extremities the position is usually one of extreme flexion at one or other joint. The crucial test of its being functional, and not due to organic contraction, is that by coaxing and manipulation the spasm may be relaxed, and the fingers, hand, and forearm, brought into the same plane. In cases of true hemiplegic rigidity this is impossible.

In hysterical paralysis of any kind the extremities are usually cold and blue, and angio-neurotic oedema may be present. The tendon-jerks are nearly always active or exaggerated, and the patient gives a peculiar cry or start of alarm and surprise when they are elicited. The knee-jerks and Achilles-jerks are never absent except in organic disease of nerves or spinal cord. True ankle-clonus is never present in functional paralysis. The plantar response may be flexor or absent, or usually active with drawing up of the entire limb and expansion of all the toes, but the slow "Babinski" extensor response of the great toe is definite evidence of organic disease.

DIFFERENTIAL DIAGNOSIS OF HYSTERICAL PARALYSIS AND CONTRACTURE.—

The unqualified diagnosis of hysteria should never be made until all other possible causes of the symptoms have been excluded. Symptoms typically hysterical in their exaggeration, instability, and apparent subsidence, may yet have an organic basis in cerebro-spinal or other diseases. To regard functional disorder as organic is excusable, to mistake organic disease for functional is unpardonable from the patient's point of view.

Localized pain is probably at the bottom of all hysterical paralysis and contractures in childhood. The pain may be due to rheumatism or a slight strain, or to incipient tuberculous disease. For example, a highly emotional child aged ten had walked with a stiff knee for several weeks. She was coaxed and persuaded to bend the knee, to kneel and walk normally, and seemed cured for the time; but three months later she was found to have tuberculous disease in the neighbourhood of the hip-joint.

Provided that caution is exercised, there is not much danger of mistaking cerebral tumour or incipient meningitis, acute rheumatism, poly-myelitis and polyneuritis, tuberculous and other forms of arthritis, for hysteria.

TREATMENT OF HYSTERICAL PARALYSIS AND CONTRACTURES.—As previously mentioned, the principle of treatment of hysterical paralysis is to supply some unexpected or novel stimulus to take the place of unfelt normal stimuli, or to counteract gradually those which have supplanted and excluded all others. The method of "taking by surprise" is often strikingly successful. A sudden and peremptory order to get up and walk, or a sharp application of faradism will sometimes cure at once. But it is unwise to attempt such miracles in the presence

of parents or relations. Removal from the surroundings in which the disability has occurred is essential. Treatment should not convey any idea of harshness or severity. Mild fardium, massage, passive movements, and appropriate exercises for the affected limbs, should be employed.

If the child seems apathetic, dreamy, and lethargic, cold douches are the best remedy. If exacting, quarrelous, excitable, and desirous to attract attention, it should be quietly ignored in the intervals of routine treatment, and should not be allowed to regard itself as an object of special interest or importance. A little good-natured teasing and badinage is useful at times. Obedience should be enforced by firmness, kindness, and encouragement.

Few cases of hysterical paralysis resist treatment on these lines in an institution; the problem is how to deal with them when cured, for a return to former surroundings often brings about an immediate relapse.

TREATMENT OF CONTRACTURES.—Examination under an anæsthetic is usually necessary both for the purpose of diagnosis and to avoid giving pain. When the spasm is fully relaxed, the limbs should be put up in splints, and if the lower extremities are affected an extension apparatus should be applied. The apparatus should be removed on the second day for the purpose of massage and passive movements and electrical treatment, and should only be re-applied if the spasm shows signs of return. They usually subside in a few days, and the remainder of the treatment consists in re-educating the child to stand and walk, or to use its arms again.

Functional Amaurosis.—The restriction of visual fields common in hysterical adults is not, in the writer's experience, met with in prepubertal years. A form of functional asthenopia, with loss of conjunctival reflex and sensibility and deficient use of the muscles of accommodation, has been described, but the condition has to be distinguished from diphtheritic paralysis. Absence of knee-jerk, nasal voice, and loose ineffectual cough, will complete the diagnosis of the latter affection. Functional asthenopia, not dependent on errors of refraction, is referred to in the section on headaches (p. 716).

Acute Amaurosis.—Apart from cases of sudden blindness occurring in hydrocephalus and intracranial tumours, in which change in the optic nerve may be observed, acute amaurosis may follow encephalitis, in which the optic discs are usually normal. The prognosis is fairly good. The condition has been attributed to toxic affection of the cortical visual centres in the occipital lobes.

Post-Eclamptic Amaurosis seems to be due to temporary exhaustion of the visual centres. A similar condition of exhaustion of other cerebral areas may produce transient aphasia or hemi-paralysis. Post-eclamptic amaurosis due to exhaustion of visual centres is as a rule not hemianopic, but affects the whole of the field of vision. Sight is usually restored in a few hours.

Uremia has to be borne in mind as a cause of transient blindness.

Amaurosis following Prolonged Blepharospasm has been described. After long disease of one or both eyes the child seems literally to have forgotten how to see, and before vision can be regained we must re-educate his cortical visual centres. Leber regards amaurosis in such cases as this as due to functional derangement of the cortical centre of vision. Another cause of transitory amaurosis is nigraia.

Hysterical Ptosis.—Hysterical ptosis is really blepharospasm. The eyelids are not flaccid, drooping, and powerless, as in myasthenia gravis or paralysis of the levator palpebrarum. The eyebrows are not raised nor the frontalis muscles corrugated in the effort to keep open the eyes. The orbiculars are tightly contracted and the eyelids quiver.

Hysterical ptosis is not uncommon in children of seven or eight years of age and upwards. The onset is usually sudden and dramatic. In the midst of a meal or some other occupation the patient becomes violently agitated, and declares that she cannot see. The doctor, hurriedly consulted, finds the patient clinging closely to her mother, sobbing quietly, and dropping tears from beneath her fast-closed lids. His approach is greeted with agitated shrieks and pathetic appeals to mother not to leave her. But on the parent's departure the child becomes quiet and docile. When told to walk, she gropes her way with outstretched hands but invariably avoids all obstacles. Isolation, and a little coaxing and persuasion, soon effect a "cure."

The sudden onset suggests some temporary defect of vision, perhaps a migrainous aura, which convinces the patient that she is blind.

Hysterical Mutism is rare before the age of puberty, but here it may be remarked that many children who develop hysteria also show signs of premature sexual development, with or without precocious menstruation. In one form of hysterical aphonia the voice is not raised above a whisper. The patient is usually tolerant of laryngoscopic examination, and approximation of the vocal cords is seen to be imperfect. Cough is forcible and noisy. A slight catarrh or much shouting and singing is sometimes responsible. After a few days' rest the voice usually returns.

In more typical hysterical mutism the patient is dumb. When asked to speak, she gazes morbidly and appealingly about her, means, but utters no words. The behaviour is as histrionic as in cases of hysterical blepharospasm. A shock from a faradic battery usually restores speech, but unless the patient is removed from her surroundings relapse may be expected.

Functional Dysphagia may occur in neurotic infants at or about the time of weaning. Sometimes the difficulty is with semi-solids, and sometimes with fluids. Sometimes it is due to the child throwing back its head in order to avoid swallowing food it does not like. The difficulty is overcome by patience and perseverance. Sometimes it is due to hurry, and may be cured by feeding slowly and in small quantities at a time, or the child may have suffered from the discomfort of food going the wrong way, and dreads that it may do so again. In some instances it may be akin to the stage fright, due to inco-ordination or stammering of the muscles of deglutition, which prevents the debutante from eating and drinking in face of an audience.

Reference has been made to dysphagia in young infants, apparently due to spasm or absence of relaxation of the cardio sphincter, leading to regurgitation of food before it reaches the stomach. It may be necessary to treat it by passage of bougies and feeding by nasal tube. It seems to be analogous to spasm of the pylorus (John Thomson).

Hysterical Dysphagia is characterized by prodigious, though unavailing, attempts to swallow. The patient complains that she cannot get the food out of her mouth, and that it goes down, but returns before it is half swallowed, or that a ball sticks in her throat to meet it as she swallows. The difficulty is with solids or

semi-solids. Treatment is by isolation, encouragement, and, as a last resource, forced feeding.

Hysterical Anorexia, as already stated, may be the consequence of painful spasms of the cardiac sphincter or lower part of the oesophagus. (See Co-ordination Nervosa, p. 689).

8. NEUROTIC HABITS.

Body-Rocking is sometimes practised by neurotic and eccentric children. Whilst sitting they sway themselves backwards and forwards twenty or thirty times a minute. Sometimes they make a creaking sound meanwhile, and appear to rock themselves to sleep or into a dreamy mental condition. They are usually queer, odd children in other ways, but not necessarily deficient in intelligence. Similar movements are, however, frequent in imbeciles. Bears, elephants, and monkeys, commonly practise swaying movements of the kind, and the habit in horses is known to groomers as "weaving." Body-rocking in children is a harmless practice in itself, but it may lead to, or be, a form of masturbation, especially in little female children.

Head-Banging is not uncommon in children when in a fit of temper. In other cases the child seems to derive some unaccountable pleasure from striking the head with his fist or against the wall or floor. It is an alarming habit, but injury does not seem to result from it. It is not as a rule associated with headache or pain, but may be an attempt to relieve suffering caused by meningitis, earache, or intracranial tumour.

See **Head-Rolling and Teeth-Grinding** *cf.* pp. 688 and 693.

Pica, or dirt-eating, occurs as a neurosis in children between the first and second year and upwards. They will consume earth, cinders, plaster from the walls, slate pencil, paper, or wool off the blankets. They crave for such articles, and refuse ordinary food, and, as might be expected, suffer from colic and diarrhoea, and become thin, sallow, and unhealthy-looking. The habit may be set up by constipation or indigestion, and rarely by worms. It is usually curable by depriving the child of its favourite comestibles, procuring free evacuation, and giving tonics to improve digestion. Unless signs of mental deficiency are present, it is not of serious import.

Hair Pulling and Swallowing are habits of children older than those who practise *pica*. The hair may be torn out in tufts, leaving bald patches, or the ends be nibbled off. When swallowed it may form hair-balls in the stomach, as in the case of cattle and Angora cats. Preventive treatment consists in shaving the head.

Nail-Biting from time immemorial has been regarded as a sign of bad temper, and the habit is certainly rare in persons of placid and equable disposition. In Freud's parlance, it may be an example of "transference" or symbolization of a "repressed complex"—an outward display of pent-up irritability. Perseverance in the old-fashioned use of bitter aloes by day, and gloves by night, will break the habit. Inveterate cases may be cured by "crowning" some of the back teeth, so that the incisors cannot meet closely enough to allow nail-nibbling.

Picking and Scratching.—Cutaneous hyperæsthesia is a characteristic of neurotic children. They are liable to urticarial, erythematous, and eczematous affections,

and also to fœrmication and itching without manifest eruptions, which cause them to scratch and peck until sores are produced on the skin. The presence of parasites should, of course, be excluded. Boutts of itching, without apparent local cause, and also urticaria, are common on warm damp days, especially in spring and autumn. Some mild form of intestinal toxæmia is probably the cause, and the old custom of giving "spring" or "cooling" medicines in the shape of rhubarb and magnesia is often beneficial. The itching may be allayed by warm alkaline lotions or by dilute solution of liquor carbonis detergens. Underclothing should be soft, loose, and non-irritating. As previously mentioned, "tics" may be suggested by subjective fœrmication.

Thumb-Sucking.—Inveterate thumb-sucking has become less common since the advent of the "comforter." Prolonged sucking of either is responsible for projecting teeth and narrowed jaws, and sometimes "air-swallowing." In addition, the thumb may become sodden and misshapen in consequence of action. Yet babies must have something to suck; it is almost the only pleasure they appreciate, and it is cruel to deprive them of it. The question of thumb versus comforter is difficult to decide. Comforters are baleful, septic things. A baby's thumb can be kept fairly clean; a comforter cannot. On the whole, preference may be given to the thumb.

The origin of thumb-sucking is lost in antiquity. Perhaps, however, when practised in moderation, it may aid in the development of salivary glands, promoting digestion in the baby, and enabling parents and nurses to obtain a few hours' sleep.

Some children seem to heighten the soothing effect of sucking the thumb by caressing the lobe of the ear or scratching the side of the head at the same time. Possibly the auricular branch of the vagus is stimulated in this manner, and some physiological effect on gastric circulation is produced which acts as a soporific.

It has been stated that thumb-sucking is an allotropic form of masturbation. The two processes are not strictly analogous, yet, in accordance with Freud's views, thumb-sucking may be regarded as an "auto-erotic" manifestation; and if so, excessive stimulation of the "oral erogenous zone" may perhaps affect injuriously the evolution of other zones on the development of which normal sexual instincts depend.

However this may be, it is desirable on other grounds that the habit of thumb-sucking should be broken during the second year. The difficulty in doing so is often considerable in the case of neurotic children. Even when apparently cured for months together, any trivial illness or emotional excitement which renders them wakeful at night will cause a relapse. The writer has known a child in her eighth year who, when feeling unwell or unable to sleep, would lie for hours bravely struggling against temptation, but at length she would get out of bed to wash from her thumbs the bitter aloes which deprived her of her solace. She was cured by a dentist, who supplied a gold-wire regulator contrived to press painfully on the roof of the mouth whenever the thumb was sucked.

In such cases, when preventive measures, such as muffling the hands or applying light splints to the upper extremities fail, or cause much mental discomfort to the child, bromides in 10-grain doses half an hour before bedtime should be given. Treatment by mental therapy may be recommended in inveterate cases, but it usually implies removal from the family circle, for neurotic children are seldom amenable to home-made suggestion.

Mercurismus, or "Cud-Chewing."—This curious habit of regurgitating and chewing food may date from early childhood, or may only be developed between the tenth and twentieth years. It may be acquired by imitation, or follow stomach trouble or some other illness. In some cases it is hereditary, and has been traced backwards through five generations by Brockbank (1907). The habit is purely involuntary, but is sometimes under control of the will. It is practised a few minutes after a meal, and may continue for an hour or more, most commonly after ingestion of meat. The process is not unpleasant unless the stomach is out of order. It may to some extent be influenced by diet and by slow mastication, especially of meat. The practice was known to the ancients, and was associated with the presence of rudimentary horns on the forehead by Fabricius ab Aquapendente in 1618. The superstition was no doubt connected with belief in fauns and satyrs. Sachs, however, took the trouble to investigate the matter, and found one ruminator among 100 cases of "cornea cutanea."

Air-Swallowing ("Aerophagia") may result from suction of air through a perforated "comforter." More commonly the habit is acquired in the effort to relieve indigestion by eructation of wind. Neurotic dyspeptics are sometimes unaware that the air which they belch at will has been previously swallowed, and is not generated in the stomach. Treatment consists in dissuading the patients from attempting to bring up wind.

Holding the Breath (Hysterical Laryngospasm).—Children aged between two and four years, when in a violent passion, sometimes hold the breath until they become purple in the face, strike out wildly with their clenched fists, dance on the floor, or throw themselves down and kick. The attacks are alarming at first, because no cry is uttered and the child seems on the point of asphyxiation, but after half a minute or so he regains his breath and screams lustily. Probably laryngospasm occurs as the result of hysterical emotion. A little cold water should be thrown in the face. It is important to avoid fussiness and any appearance of consternation; otherwise the child will be encouraged to hold his breath deliberately on slight provocation. The tempestuous application of a slipper is the best remedy in such cases.

The emotional condition in which hysterical laryngospasm and voluntary holding the breath occur serves to distinguish them from laryngospasm associated with tetany (q.v., p. 682).

In **EXPIRATORY APNŒA** (Neumann) there is no inspiratory stridor, but the chest becomes fixed and respiration is arrested. The patient fights for breath, may lose consciousness and die. Or after a few moments respiration may be restored. According to Neumann, the attacks resemble the expiratory apnœa which is sometimes fatal in laryngismus stridulus (laryngospasm), but take place in older children. They may be the cause of death in status lymphaticus (q.v., p. 548).

Masturbation is a habit which may be acquired by infants of both sexes, even within the first year of life. It may be the consequence of local irritation, such as balanitis, or vaginitis set up by oxyurides. Sometimes unscrupulous nurses or companions are responsible, but injustice in this matter should be avoided. It is more common in female than in male infants, and in the latter it is seldom practised to the extent of producing orgasm. In girls friction is applied by the hands or by the heel of the foot, but most commonly by "thigh-rubbing." The thighs are tightly crossed, and the child rocks itself to and fro or from side to

side on its buttocks, becomes excited and flushed, holds the breath or grunts; perspiration breaks out, and is followed by pallor and exhaustion. These signs of sexual orgasm are sometimes mistaken for "fits" or epilepsy. When practiced openly and persistently after the age of three years, the habit is usually a sign of mental deficiency; but neither mental deficiency nor insanity, nor, indeed, any of the lurid pathological consequences attributed to masturbation in a previously healthy subject, is in reality so produced. Secret indulgence is more harmful morally than physically. Imbeciles sometimes masturbate many times a day without ill effect on physical health and vigour. The sunken eyes, dilated pupils, sallow cheeks, reverie and lassitude, which are often regarded as evidence of masturbation, are simply signs of neurasthenia. It is true that sexual neurasthenics often attribute all their woes to self-pollution and doubtless they may be rendered worse thereby, but the typical sexual neurasthenic is often the picture of physical health. His mind suffers, not his body.

The treatment in infancy is preventive. The child should be watched whilst awake, and the process should be checked on every occasion. Some form of mechanical restraint should be devised, and be worn in bed. The feet may be tied in opposite corners of the lower end of the bed, and the arms and hands confined by a sleeveless garment with a waist-belt. In all cases local causes of irritation should be cured if present. In older children the habit is less easy to cure. The results should not be exaggerated, nor the child treated as a moral outcast or pariah. It is usually sufficient to speak plainly about the filthiness of such practices, and to warn the child that persistence in them may unfit him for games and other pleasures. A healthy child usually discovers this for himself. It is inadvisable to caution young children against bad habits unless they are known to exist. The warning may excite curiosity, and lead to committal of the offence itself. At the school age, however, every child should be put on his guard against obscenity and contamination by *Opelias excoel.* What are called "straight talks" on sexual subjects may be useful to children of both sexes at the eve of puberty. The physiology of the sexual system may be explained, but laymen would be wise not to dwell on the pathological results of immorality. Injudicious terrorism may lead a neurotic boy to believe that a nocturnal emission is a sign that sin has found him out. Evidence of puberty in girls may similarly be misinterpreted. If "straight talks" were left to school doctors, sexual neurasthenia would be less prevalent than it is. The ordinary healthy schoolboy need only be told that if he abstains from evil habits he will play a better game at football, and need not worry himself about past delinquencies.

Cold baths, hard bed, early rising, regular exercise (except climbing ropes and poles), plain diet, should be enjoined. But the neurasthenic who believes that he has ruined himself body and soul by even more transgressions requires much consideration, assurance of recovery, and encouragement to occupy mind and body in healthy pursuits and recreations. Endeavours should be made to convince him that nocturnal pollutions are natural events.

With regard to drugs, bromides are useful for infants who masturbate, but should not be given to older children.

Salix nigra has been advocated. Tonics, such as hypophosphites, quinine and iron, if *anæmia* is present, should be prescribed. The bowels should be regulated and digestion attended to if impaired. *Balanitis* calls for circumcision.

3. DELAYED WALKING—LOSS OF ACQUIRED WALKING POWER.

Functional Paralysis.—If a child cannot stand or walk by the age of two years, it is either mentally deficient or some morbid condition of the brain or cerebellum or spinal cord or muscles is present. Excluding imbecility and diplegia due to cerebral or cerebellar disease, the commonest cause of delayed walking is rickets. But rickets in such cases shows itself less in osseous changes and epiphyseal enlargement than in general laxity of tendons, ligaments, and muscles (hypotonia). There is excessive motility in all the joints, so that the child can imitate all the gestures of a professional contortionist. There is no paralysis, but the muscles are weak and flabby, and the child cannot support its weight when placed on its feet.

Treatment is by massage and passive exercises of the limbs. The old-fashioned "go-cart" is useful in teaching such children to walk without risk of inducing deformities. The prognosis is good.

The condition has to be distinguished from amyotonia congenita (Oppenheim), which is in reality a congenital form of myopathy (see p. 899) characterized by progressive muscular wasting, loss of tendon-jerks, and eventually by deformities due to musculo-tendinous contractures.

Loss of Acquired Walking Power.—Sudden or gradual inability to stand or walk may depend on pain, paralysis or weakness, or hysteria. If sudden, infantile paralysis or paraplegia from spinal caries may be suspected. If gradual or not immediate, rickets, rheumatism, diphtheritic paralysis, scurvy, epiphysitis, and incipient tuberculous disease, should be borne in mind. The distinctive features of most of these diseases, and also of gross cerebral disease, such as tumour or hydrocephalus, which may cause sudden or gradual inability to stand or walk, need not be emphasised here.

Pain is a common cause of "going off their feet" in little children. Inability to stand or walk has to be distinguished from reluctance to make the attempt because pain is caused thereby or dreaded. If the effort to stand or walk is painless, the child will usually make it although it may be obviously paralyzed.

Rickety children at the age of two or three often tire themselves by running about at play, and next day refuse to put one or both feet to the ground, and cry piteously if compelled to do so. On examination there is obviously no paralysis, but passive movements of ankle, knee, or hip, excite resistance and screaming. No sign of inflammation of the joints can be discovered. Simple strain of certain tendons or muscles from over-exertion explains the loss of walking power, for with a few days' treatment by rest and gentle massage the pseudo-paralysis passes off.

Muscular rheumatism or fibrositis in children upwards of three years of age may produce similar effects. In infants under eighteen months rheumatism may be excluded, and pseudo-paralysis is usually due to scurvy or epiphysitis. *Chronic pseudo-paralysis* is described elsewhere. *Pseudo-paralysis from want of exercise* is common at the end of tedious illnesses. The child has forgotten how to walk, and must be taught again to do so. Such delays to convalescence might be prevented by massage and passive movements of the limbs when children are confined to bed from any cause for lengthy periods.

10. DEFECTS OF SPEECH.

Stammering and Stuttering (Dyslalia).—In Germany the word "Stammeln" (stammering) implies defective articulation (lalling and lisping or parolalia), and by "Stottern" (stuttering) is meant inco-ordination of the muscles concerned in the production of speech (dyslalia). In this country "stammering" and "stuttering" are synonymous terms, answering to the German "Stottern." Stuttering may be regarded as a subvariety of stammering. Stuttering consists in reiteration of the initial consonants or syllables in words. Stammering consists in arrest of utterance due to spasms and inco-ordination of the muscles concerned in the production of speech. There is no essential difference between them. Inco-ordination is present in both, but spasm is more marked in stammering than in stuttering. In stuttering, owing to imperfect vocalization and inspiratory force, utterance of initial syllables or consonants is weak and abortive. The stutterer is aware of this, and repeats the syllable or consonant over and over again in the effort to make it audible, but he does not realize that an intake of air is a necessary preliminary. Stuttering is less common than stammering, and more amenable to treatment by respiratory exercises. In stammerers the spasm may affect the articulatory muscles—namely, those of the lips and tongue—or the muscles of the glottis, and the respiratory muscles, including those of the abdomen and the diaphragm. Inco-ordination may exist between the different sets of articulatory muscles or between those of the respiratory muscles, or between the articulatory and the respiratory muscles, and thus prevent the equable flow of vowels and consonants. It is necessary in all cases first to ascertain the part of the mechanism which is at fault, and in order to do so the physiology of speech must be understood. Vowel sounds are produced in the glottis by vibrations of the true vocal cords, which are set in action by currents of air passing over them. The different forms of vowels, *a, e, i, o, u* (*ah, eh, ee, oh, oo*), arise from modifications of original tones produced in the larynx, by alterations in the capacity and resonance of the oro-nasal cavities. Consonants are produced by interruption of the current of air in some part of the air-passages above the larynx.

Three "stop positions," as they are called, are formed—

1. By the lips, as in producing *b, p, m*.
2. By the tip of the tongue and upper teeth, as in producing *t, d, n*.
3. By the anterior part of the tongue and palate, as in producing *l, k, g, s, z*.
4. By the posterior part of the tongue and soft palate, as in producing *h, k, or ch, g, j, r, ng*.

Consonants are divided into voiced oral and voiceless oral. Voiced oral consonants, such as *b, d, g, m*, are accompanied by sound produced in the larynx each time the consonant is uttered; in the voiceless oral, such as *p, t, k*, the larynx is silent. The consonants *f* and *v* are labio-dentals; *f* is a voiceless and *v* a voiced oral. *L* and *r* are voiced oral consonants; *l* results from partial contact of the tip of the tongue with the fore part of the palate, the sides of the tongue remaining free. *R* (trilled) is produced by vibration of the tip, not the sides of the tongue; when *r* is strongly "burred" the uvula also vibrates. *M, n, ng*, are called "voiced nasal resonants," in the production of which the soft palate is depressed, air is prevented

from passing through the mouth by closure of the lips or by approximation of the tongue to the palate, and escapes through the nares.

Bearing these elementary facts in mind, it is usually easy to ascertain which part of speech mechanism is at fault. Spasms may affect the articulatory or the vocal organs. Inco-ordination in the great majority of cases is between the oral and the laryngeal mechanisms. The patient does not know that consonants are formed within the oral cavity, and vowels within the larynx, and that clear articulation depends upon harmonious action between the vocalizing organs and those of articulation. He is not aware that consonants are merely interpolations into vocalized words, and makes frantic efforts to pronounce the consonants without the vowel sounds which should precede, accompany, or follow them (Cutburt).

His energy is devoted to the oral mechanism of speech; the laryngeal and inspiratory mechanisms are ignored. He has no notion of governing the exit of his breath; he emits it as a gust without making use of the glottis as a waste-preventer, and he attempts to speak when breathless or tries to talk without first inhaling air. In some cases the difficulty is in vocalizing initial vowels. Spasm of the false vocal cords which control exit of the breath takes place, and the patient stands speechless, with widely opened mouth and an agonized expression, for several seconds together; then, when spasm relaxes, he pours forth his words in one continuous exhalation until he becomes breathless (*q.* "As you Like It," Act III, Scene 2).

Ætiology.—Stammering is a functional disorder, and may be associated with other functional troubles, such as night terrors, enuresis, tics, the offspring of the neurotic temperament. Any emotional stress in the shape of fright, worry, anxiety, or any lowering of general health, may predispose to stammering. Local morbid conditions of the mouth or naso-pharynx which interfere with respiration may be partly responsible for stammering. Phimosis may be an indirect cause. Morbid stammering does not occur until about the fifth or sixth year, when powers of fluent speech should have been acquired. It frequently arises from imitation, and is not an hereditary affection. Boys are more apt to stammer than girls.

Treatment.—Attention should be paid to general health and needed local defects. In undertaking the treatment of stammering, it is first necessary to ascertain which part of the mechanism of speech is at fault. Is the trouble with the consonants—explosives, voiced and voiceless; with the fricatives; with the nasal resonants, or with the vowels? Alliterative sentences illustrating all the speech sounds can easily be constructed and used as text.

In the great majority the fault is respiratory. The patient must therefore be instructed, in the first place, how to increase the vital capacity of his lungs by respiratory exercises, especially those which occupy the diaphragm and abdominal muscles. He must also be taught to control the exit of his breath, first without, and secondly with, vocalization of vowels. He must intone the vowels, and be shown how to introduce the consonants meanwhile, as, for instance, in uttering the sounds *e-ah*, *o-ah*, *a-ah*, etc. He thus learns that the voice need not be intermitted in order to interpolate consonants, and that vocalization of vowel sounds aids in giving utterance to the consonants over which he stumbles.

Exercises in the methods of drawing and controlling the breath without reference to their practical bearing on speech production should first be mastered. Exercises

in vocalizing vowels should follow, and, finally, instruction in the art of forming consonants in conjunction with phonation should be given. Slowness and deliberateness of speech should always be insisted upon. The difficulty with most stammerers is that they cannot be induced to practice in daily life the principles which enable them to speak without stammering in the presence of their teacher. Timid, nervous stammerers cannot, and those who are callous and indifferent to their affliction will not, be treated at ordinary schools. Private tuition is almost always necessary.

Lalling and Lipping (Paralalia).—Lalling (from the German "Lallen," to speak as a child) and lipping consist in substitution of consonants for each other. Both conditions are characteristic of "baby language," which may be persistent in the feeble-minded and imbeciles. Minor degrees of lalling and lipping, in which the difficulty is only with sounds such as *s* and *r*, do not, of course, indicate deficient intelligence. *S* is commonly rendered *th*, *r* as *w*, *l*, or *y*, *th* as *f*, *w* as *v* or *r* as *m*. Defective ear or defective education accounts for some of these peculiarities; sometimes they are due to malformation of the mouth or teeth. The most extreme form of lalling and lipping occurs in so-called *idioglossia*.

Idioglossia is the name given by Hale White and Golding Bird to a form of speech so peculiar that the patient seems to have invented a language of his own. On careful analysis, however, the peculiarity is found to consist in substitution of vowel sounds for consonants, and of the various consonants for each other, and in slurring over syllables which are difficult to articulate. It resembles "baby language" in this respect, but differs from it in that the grammatical construction of sentences is correct; inflections are used, every sentence is complete, and each syllable of a sentence is represented by a separate sound. Pronunciation of vowel sounds is in all cases good, and many are frequently used in lieu of consonants. The consonants chiefly employed are labials, linguo-dentals, anterior linguo-palatals. Posterior linguo-palatals, *k*, *g*, and next to them *f*, *t*, *r*, present most difficulty, and the consonants *i*, *e*, *a*, commonly take the place of them. In a patient under the writer's care the consonants *b*, *p*, *d*, *l*, *k*, were freely used, and also the various vowel sounds. *T* and *d* were substituted for each other. *F*, *v*, *th*, *s*, *sh*, *z*, *zh*, *c* (*sch*), did not appear. *K* and *ck* (*kand*) were present. *G* was variable, but usually pronounced *st*. *N* and *m* were variable, but usually had a vowel sound, *ah*. *Ny* was absent, *l* only appeared as a terminal, *p* and *b* only as initials. As examples, "iron" was pronounced "dahi," "fly" as "pahi" (*a* as in "ah"), "sheep" as "tér," "slipper" as "tér," "Yes" became "dái" or "hái" (as in "day"); "dog" was "dol," "Go" was "do," but "God" was correctly pronounced.

Although, as might be expected, the difficulty in many cases is with sounds of which the mechanical production is obscure—*e.g.*, gutturals and posterior linguo-palatals—*idioglossia* is not a species of motor aphasia. For on investigation it is found that the patient can pronounce all the consonants which he omits, and apply them correctly if urged to imitate the visible methods by which they are vocalized.

The defect seems to be one of audition. Without being deaf in the ordinary sense, the patient is unable to discriminate between sounds. His ear does not detect any difference between his own jargon and words correctly spoken. In favor of the view that *idioglossia* is primarily a fault in audition, analogous to color blindness, is the fact that in typical cases there is complete absence of an ear for music. This is not so in all cases which have been described as *idioglossia*, but many atypical conditions have been included under this heading. A physical disability

to pronounce certain consonants, such as *s*, *w*, *th*, *l*, and *r*, is extremely common, and does not constitute idioglossia. The term should be confined to cases in which, by use of the oro-visual method, it can be shown that the patient can accomplish word and letter sounds by aid of eye and sense of touch, but not by ear alone. Idioglossia may therefore be classed with congenital word deafness, word and colour blindness, all of which affections depend on anomalous development of the centres of sight or hearing. Intelligence is not defective, and may be above the average in cases of idioglossia. On the other hand, the condition may occur as part of general cerebral mal-development in imbeciles, but the speech of imbeciles is merely a persistence of the infantile type in which "falling" or lipping is normal. Idioglossia must also be distinguished from defective articulation due to local malformation or morbid condition of the nose, throat, tongue, and teeth. Amongst them may be mentioned naso-pharyngeal obstruction, congenital insufficiency of the soft palate which gives rise to the speech characteristic of paralysis or spasticity of the palatal muscles. Delayed descent of the upper central teeth after the temporary teeth have been shed may cause faulty articulation of ligno-dentals. Tongue-tie, being a rare cause of defective speech, is the first to be suspected by mothers and nurses.

TREATMENT OF IDIOGLOSSIA.—In all true cases of idioglossia correct articulation is possible if the child's attention can be gained sufficiently to make him imitate the mechanism necessary for the production of various sounds. If his eye and attention wander, his ear is no help to him. He must therefore be taught to speak precisely as a deaf-mute is taught on the "oral" method, by aid of vision and sense of touch. It is necessary first to ascertain which sounds are most faultily produced. Then monosyllabic sentences should be constructed in which the consonants occur both as initials and as terminals, and the patient should be made to imitate their mode of production.

Much perseverance and patience are required, as relapses are common when tuition is lax and intermittent. It may be necessary to isolate the patient from other children, because they soon learn to understand him, and he therefore takes no pains to speak correctly. The visual memory is often defective, and should be trained by lessons in drawing from memory. Attempts should also be made to cultivate his ear by "sol-fa" exercises.

CHAPTER XIV

ORGANIC DISEASES OF THE NERVOUS SYSTEM

F. E. BATTEN

CLINICAL EXAMINATION.
MENINGITIS.
INTRAORBITAL ABSCESS.
GENERAL EPENDYMITIS.
FOCAL-ENCEPHALO-MYELITIS; POLIO-MYELITIS.
INTRACRANIAL TUMORS.
VASCULAR LESIONS OF THE CENTRAL NERVOUS SYSTEM.
SINUS THROMBOSIS.
GENERAL EPILEPSY.
INFANTILE HEMIPLEGIA.
HYDROCEPHALUS.
HEMATOMYELIA, HYDROMYELIA, SYRINGOMYELIA, CENTRAL GLIOMATOSIS.
PARALYSIS OF THE CRANIAL NERVES.
DIFFUSE SPASTIC PARALYSIS.
BIRTH INJURY TO THE BRACHIAL PLEXUS.
VON RECKLINGHAUSEN'S DISEASE.
TOXIC NEURITIS.
PROGRESSIVE SPINAL MUSCULAR ATROPHY (WERNICKE-HOFFMANN).
OTHER FORMS OF PROGRESSIVE MUSCULAR ATROPHY DUE TO SPINAL DISEASE.
AMACROTIC FAMILY IDIOT (WARR-TAY-SACH).

GENERAL DEGENERATION WITH SYMMETRIC CHANGES IN THE MACULA.
PROGRESSIVE LENTICULAR DEGENERATION.
SYNDROMES OF THE CENTRAL NERVOUS SYSTEM.
DEGENERATED SCLEROSIS.
MYASTHENIA GRAVIS.
HEMIPARESIS OF THE FACE.
FAMILY PERIODIC PARALYSIS.
FAMILY AND HEREDITARY ATAXIA.
CEREELLAR ATAXIA.
PARAPLEGIA.
TUMORS OF THE SPINAL CORD.
FAMILY SPASTIC PARALYSIS WITH ARDROPHY.
FAMILY AND HEREDITARY SPASTIC PARAPLEGIA.
NEURITIC TYPE OF PROGRESSIVE MUSCULAR ATROPHY.
NERVOUS MANIFESTATIONS DUE TO SPINAL CURVES.
NERVOUS MANIFESTATIONS ASSOCIATED WITH SPINAL RIFIDS AND SPINAL MENINGOCYCLE.

CLINICAL EXAMINATION OF THE NERVOUS SYSTEM.

It is important to carry out in a systematic manner the examination of a child who is suffering from some affection of the nervous system.

It is needless to say that the first thing to do is to get "on good terms" with the child. This is easily accomplished by some people; others experience difficulty.

Much can be gained by the general inspection, though it is not a good thing to stare at a child; it is resented, "war is declared," and peace is with difficulty restored.

It is as a rule unwise to try to hold a child during examination; let it lie on a bed or on the mother's or nurse's lap. If it struggles, let it have the freest play for its movements. The same systematic examination should be adopted in the examination of the nervous system of the child as in the adult.

The head should be examined, and the cranial nerves in their order. The *first nerve, that of smell*, is rarely capable of being examined in a child. The *second, that of sight*, is tested by noting if the child will follow objects—in older children by test type; and in these a rough idea as to field of vision can be obtained. The *fundus of the eye* may now be examined, but in practice it is often better to leave this till the end.

The examination of the *fundus* is most easily accomplished by placing the child on its side, a lamp being held behind its head, and the light reflected from the ophthalmoscope on to the eye. An electric ophthalmoscope is of an immense advantage in examining the *fundus* of a child. No attempt should be made to hold the head nor to lift the eyelid (except in a comatose child). Most children will sooner or later open the eye, and an examination of the *fundus* will be obtained. The right eye of the child should be examined with the right eye of the observer, and when that is completed it should be turned round in bed so that the other eye is uppermost, and the left eye of the child examined with the observer's left eye.

The examination of the *fundus* of the eye will often afford valuable information as to the condition from which the child is suffering. If difficulty is experienced in obtaining a view of the *fundus*, an examination by the indirect method should be made, when opacities of the cornea, lens, or vitreous, are more easily seen, and may account for the difficulty in obtaining a view of the *fundus*.

Optic neuritis, optic atrophy, tubercle of the choroid, choroiditis, hæmorrhages, and other changes of the *fundus*, may be found.

If the above method be practised and adopted, there are very few children in whom a good examination cannot be obtained, even without the use of homatropine, though for a complete examination dilatation of the pupils is needed.

The *third, fourth, and sixth nerves* are examined together by testing the movements of the eyes, noting any weakness of the levator palpebrarum, the action of the pupil to light and on accommodation. An Argyll-Robertson pupil is a rare manifestation in a child, but, when it occurs, is, as in the adult, suggestive of syphilis. It occurs also in lesions of the corpora quadrigemina. The inequality of the pupils may be noted. Such inequality may, apart from paralysis of the third nerve and local conditions of the eye, be due to the affection of the sympathetic on one side. The action of cocaine may be tried, for in cases of paralysis of the dilator fibres due to affection of the sympathetic the pupil fails to dilate when this drug is applied to the eye. The direct and consensual reaction of the pupils to light should be tested.

In cases showing hemianopsia, the effect of light thrown on one or other half of the retina should also be observed (Wernicke's sign). In a case of hemianopsia due to a lesion of the chiasma or optic tract, the pupil will fail to react when the light is thrown on the blind side, whereas if the hemianopsia is due to a lesion above the external geniculate body the pupil will react readily to light.

The contraction of the pupil to light and its rapid dilatation whilst the light is still being thrown on the eye, and the oscillation of the pupil called "hippus," are present under a variety of circumstances, and these signs are by no means indicative of organic disease of the nervous system.

An eccentric pupil is one in which, owing to the uneven contraction of the iris, the pupil becomes eccentric, generally in an upwards direction. This has been noted in cataract and other disease, but, although of interest, has no particular diagnostic value.

Defective upward and downward movements of the eye whilst the lateral movements remain good is a characteristic symptom pointing to the involvement of some portion of the nucleus of the third nerve, and commonly occurs in mid-brain lesions, especially in those of the corpora quadrigemina.

Just as loss of upward and downward movements of the eyes is characteristic of affection of the third nerve nucleus, so is loss of lateral movements characteristic of a lesion of the nuclei of the sixth nerve.

It is important to bear this fact in mind in considering the causes of paralysis of the external rectus muscles of the eyes. For example, if weakness of the external rectus on the right side is associated with weakness of the internal rectus of the left eye for conjugate movements to the right side, whilst it can be shown that there is no weakness of the internal rectus on convergence, it is probable that the lesion is placed in the nucleus of the sixth nerve, or interrupts the fibres in the posterior longitudinal bundle as they pass from the sixth to the third nerve nucleus of the opposite side.

An affection of the fifth nerve gives rise to both motor and sensory symptoms.

The motor symptoms are shown by weakness of the temporal and masseter muscles in closing the jaw, and the deviation of the jaw to the paralyzed side on attempting to open the mouth; the sensory symptoms by anesthesia over the distribution of one or all of the divisions of the fifth nerve on the face and mucous membrane of the mouth and nose, and by the abolition of the corneal reflex.

The corneal reflex should be tested by directing a current of air on to the cornea from a small india-rubber bellows.

The seventh nerve supplies the muscles of the face, and also in part its course carries the taste fibres. It is tested by asking the child to shut the eyes, raise the eyebrows, show the teeth, and noting the movements of the face whilst laughing or crying; and, if the child is old enough, testing the sensation of taste on the anterior two-thirds of the tongue.

If the lesion is in the nucleus or peripheral portion of the seventh nerve, there is marked weakness of the movements of the upper portion of the face—i.e., the closing of the eyes and raising of the eyebrows—as well as in the movements of the lower portion. If the lesion is distal to the geniculate ganglion, and proximal to the entrance of the fibres of the chorda tympani, taste is affected. If the lesion is situated above the nucleus of the seventh nerve, then the weakness of the face is only marked in the lower part of the face—i.e., on showing the teeth. There may be, and often is, a little weakness of the orbicularis, but never the complete paralysis seen in a lesion of the peripheral portion of the nerve. The loss of emotional expression whilst voluntary movement remains good indicates a thalamic lesion as the cause of the weakness.

A peripheral lesion of the seventh nerve will abolish the corneal reflex, but the stimulus to the cornea is felt, and if sufficiently irritating the head will be drawn away. This should be compared to the absence of the corneal reflex from affection of the sensory portion of the arc by involvement of the fifth nerve.

In supranuclear lesions of the seventh nerve the facial muscles react normally to electrical stimulation; in peripheral lesions the electrical reactions are altered.

The eighth nerve consists of two parts: the cochlear, concerned with the appreciation of sound; the vestibular, concerned with the sense of position of the body in space.

The hearing may be defective owing to affection of the auditory apparatus.

In such a case the bone conduction of sound will be normal, whilst the aerial conduction will be defective. Normally the tuning-fork should be heard by aerial conduction after it has ceased to be heard by bone conduction.

The test is made by placing the foot of the tuning-fork over the mastoid, and when it has ceased to be heard the prongs are held just outside the auditory meatus. If this sound is not recognized, the defect is probably in the auditory apparatus, and not in the nerve. The test is a difficult one to apply in children, and is only of limited application.

The vestibular portion of the nerve is investigated by the injection of cold and warm water into the meatus, so as to impinge on the membrana tympani. If the vestibular nerve is intact, nystagmus is produced to the side investigated, either spontaneously or on lateral deviation of the eye. Failure to produce nystagmus indicates vestibular disease. Rotation of the patient similarly produces nystagmus.

The ninth nerve supplies sensation to the palate and taste to the hinder third of the tongue, and affection of this nerve also gives rise to weakness of the palate and loss of the palatal reflex.

The tenth and eleventh nerves supply the pharynx, the larynx, the sternomastoid and trapezius muscles.

The twelfth nerve supplies the tongue, and affection of this nerve gives rise to wasting and weakness of the intrinsic fibres of the tongue.

In supranuclear lesion of the twelfth nerve there is weakness of the tongue, so that when protruded it is turned toward the paralyzed side, but there is no wasting.

MUSCLES OF THE TRUNK AND LIMBS.—Having completed the examination of the cranial nerves, the examination of the muscles of the trunk and limbs should be undertaken.

The capacity of the child to perform movements should be tested, any weakness, wasting, or hypertrophy, of muscles being noted.

The power of the back and abdomen should be tested, and the action of the diaphragm and thoracic muscles noted.

The child should be asked to attempt to raise himself into a sitting position, and the movements of the umbilicus in relation to the costal angle noted. The umbilicus should remain stationary: if, however, it moves up, it indicates weakness of the lower segment of the muscles; if downward, weakness of the upper segments of the abdominal muscles. The child should be asked to get up from a lying position, and should be seen to walk.

Goat will often furnish most valuable information as to the nature of the disease.

The co-ordination of the arms should be tested by making the child touch the tip of his nose with the first forefinger, the eyes first being open and then closed.

The co-ordination of the legs is tested by asking the child to place the heel of one leg on to the knee of the opposite leg. The hypertonus (increased tone, rigidity) and hypotonia (loss of tone, flaccidity) of the limbs should be observed, and at the same time the sign pointed out by Kernig may be noted—viz., the inability of the observer to extend passively the legs beyond a right angle to the thigh, the thigh being fixed at a right angle to the trunk.

If there are signs suggestive of cerebellar disease, special methods should be adopted to bring out the inco-ordination. The child should be asked to hold the arms out and rapidly separate and approximate them. If the lesion be unilateral, the

inco-ordination of the one side as compared with the other is very marked (*dysad-
douchokinesia*).

In cerebellar disease the child should be asked to crawl along the floor; it will then be noticed that the buttocks tend to fall over to one side or the other. If the buttocks tend to fall over to the left (i.e., inging corkscrew direction), the lesion is probably in the left lobe of the cerebellum; if to the right (i.e., an outgoing corkscrew), a right-sided lesion is probably present.

Increased tone (*hypertonia*) commonly occurs with lesions of the upper neuron, and hence frequently occurs in cerebral disease. Diminished tone (*hypotonia*) occurs with lesions of the lower motor neuron, and also especially in association with cerebellar disease.

Tremor.—*Spontaneous tremors* are seen in a variety of nervous diseases.

Intention tremors occur both with cerebral and cerebellar disease.

Clonus movements are indicative of loss of cerebral control.

Athetosis is a manifestation of cerebral disease, probably due to a lesion of the basal ganglia, and only occurring when the pyramidal tracts are relatively intact.

Rhythmical tremor is seen more especially in association with lesions of the mid-brain, the red nucleus, and optic thalamus.

Fine tremor occurs in children as a familial complaint.

REFLEXES.—The examination of the reflexes often furnishes most valuable information as to the nature of the disease.

The corneal and light reflex has already been dealt with. The *facial reflex*—i.e., the contraction of the orbicularis palpebrarum—on tapping the side of the face over the malar bone, is seen in many cases of affection of the upper motor neurons. It is often present to a very marked degree in cerebral diplegia. A similar movement is present in cases of tetany, but it probably depends on a direct excitability of the muscles to a mechanical stimulus.

The *lip reflex*, to which John Thomson has called attention, is of interest, but has no special diagnostic value.

The *palatal reflex* may be absent under a variety of circumstances in which the arc of the reflex is interrupted. Most children have a most active palatal reflex. An active palatal reflex in association with a paralysis of the palate is indicative of an upper motor neuron lesion, and occurs in association with a bilateral hemiplegia or diplegia.

The *arm-jerks* can normally be obtained in children. Sometimes they are active. To the increased activity of the arm-jerks alone no special significance can be attached, but if the jerks on the one side are greatly exaggerated as compared to the opposite side, it is indicative of some pyramidal lesion—e.g., hemiplegia. The absence of the arm-jerks occurs in lesions of the lower motor neuron—e.g., poliomyelitis, injury to the brachial plexus, and diphtheritic paralysis.

The *abdominal reflex* is not present in young babies; it generally develops between the second and sixth months of life, and is usually active after the fourth month of life.

The abdominal reflex should be tested by stroking the abdomen obliquely in its four quadrants, and noting the movements of the umbilicus towards the part stroked. The gentle stroke of the finger is often sufficient to elicit it, but in other cases the point of a pin is needed to bring out the reflex.

The absence of the abdominal reflex on one side is of great diagnostic value, as

indicative of affection of the pyramidal tract. There are, however, certain exceptions to this rule. In some cases of cerebral diplegia the abdominal reflex may be found to be quite active, and, again, in some cases of infantile hemiplegia it is found that the abdominal reflex is as active on the paralyzed as on the sound side. The rule, however, that absence of the abdominal reflex on one side indicates affection of the pyramidal tract on that side holds good.

The abdominal reflex may be absent owing to a lesion of the lower motor neuron, such as a poliomyelitis of the thoracic segment of the spinal cord.

The cremasteric reflex, like the abdominal, develops during the first year of life.

The *knee-jerk* is present in babies at birth, and it may be said are never absent except under pathological conditions. Many persons fail to obtain the knee-jerk in infants and young children because of the manner in which they attempt to elicit it. The best method of testing the knee-jerk in childhood is to place the child in the lying position, and to hold the thigh in the left hand and support the leg on the forearm of the observer, and when complete relaxation of the muscle is obtained the patella tendon should be struck with the percussor.

The knee-jerks may be absent in various conditions independent of any disease of the nervous system. Thus, in pneumonia, broncho-pneumonia, severe enteritis, and other toxic conditions, they are not infrequently absent. Their presence or absence is to some extent indicative of the severity of the poison. In some cases of enteritis which have been prolonged, it will be noticed that the knee-jerks are absent for a considerable period after the subsidence of the acute symptoms.

In diphtheria the disappearance of the knee-jerks whilst the patient is under observation should make one especially careful as regards active movement on the part of the patient. The return of the knee-jerk is a good sign. Exaggeration of the knee-jerk, not infrequently precedes the disappearance in cases of diphtheria.

The *ankle-jerk* is best tested by placing the child so as to lie on the abdomen, flexing the leg at the knee and holding the foot at right angles to the leg. A tap on the tendo Achillis gives rise to a contraction of the gastrocnemius and soleus, and produces extension of the foot. The ankle-jerks are usually absent when the knee-jerks are absent, but there are exceptions to this rule—e.g., pseudo-hypertrophic paralysis, Friedreich's ataxia.

Plantar Reflex.—The method of obtaining this most important reflex is as follows: The leg should be flexed, and complete relaxation obtained. If possible, the attention of the child should be directed away from its foot. The foot is stroked gently on the outer side of the plantar surface, from the heel towards the little toe; the movement of the big toe is now observed. Normally, in children above two years of age the great toe is flexed; in babies the toe is sometimes flexed, but more often the big toe is sharply extended, and in association with this the little toes are also extended.

It is the slow extension of the big toe which is of important diagnostic value, and even in quite young children, when such a response is obtained, considerable value may be attached to the observation.

In older children it not infrequently happens that an extensor response will be obtained on testing the plantar reflex on the outer side of the foot, whilst on the inner side, with pressure over the flexor tendon, a flexor response is obtained. It should be remembered that the plantar reflex, like other reflexes, will in the early stages of disease vary according to the condition of the patient. A child

who has been absolutely at rest may show neither ankle clonus nor an extensor plantar response, but a little exertion will bring out both these phenomena.

Again, with varying conditions of intracranial pressure the plantar reflex will change its character. This is frequently seen in cases of cerebellar tumours where, either from direct or indirect pressure due to hydrocephalus, the pyramidal tracts are affected.

The quick withdrawal of the foot and rapid extension of the toes on powerful stimulation of the sole of the foot is of no diagnostic value, but a carefully-examined plantar reflex is of the greatest service in the diagnosis of nervous diseases.

SENSATION.—The various forms of sensation should be tested in a child. In infants, that of pain is practically the only one that can be examined, but in older children tactile, pain, heat and cold, sense of position, appreciation of weight, size, and nature of objects, can all be investigated.

Loss of sensation may be due to a cerebral, to a spinal, or to a peripheral lesion, the distribution and character of the loss of sensation furnishing evidence as to the locality of the lesion.

MENINGITIS.

INTRODUCTION.—Meningitis is one of the most common affections of the nervous system in children. The inflammation may attack the dura mater, the pia mater, or the arachnoid. The former is rarely, the two latter commonly, affected. The inflammation may arise secondarily to some well-marked lesion elsewhere within the body, or without any such obvious focus.

A classification of meningitis on a pathological or bacteriological basis is the most satisfactory, and the following, suggested by Sir William Osler, is that which has been found most practical.

No distinction will be made between primary and secondary infection. In some cases the focus from which the infection has arisen may be obvious, in other cases it may not be found, but such a distinction has no great clinical value.

CLASSIFICATION:—

<i>Meningoencephal Meningitis</i>	(A) Epidemic cerebro-spinal meningitis. (B) Sporadic cerebro-spinal meningitis, formerly known as "posterior basal" meningitis.
<i>Pneumococcal Meningitis</i>	Due to infection by the pneumococcus arising from the ear, nose, orbit, lungs, pleura, pericardium, peritoneum, joints, or from a point of entry which may not be obvious.
<i>Traumatic Meningitis</i>	Due to injury.
<i>Tuberculous Meningitis</i>	Due to infection by tubercle bacillus.
<i>Pyogenic Meningitis</i>	Due to infection by staphylococci and streptococci.
<i>Rheumatic Meningitis</i>	Due to <i>Diplococcus rheumaticus</i> .
<i>Other Form of Infection</i>	Due to <i>Bacillus typhosus</i> , <i>B. enteritidis</i> , <i>B. influenzae</i> , gonococcus, <i>Actinomyces streptothrix</i> .
<i>Syphilitic Meningitis</i>	Due to infection by <i>Treponema pallidum</i> .
<i>Severe Meningitis</i>	Due to nephritis, acute specific fevers, mumps, and certain otitic infections.
<i>Meningism</i>	Symptoms of meningitis without demonstrable meningitis.

Meningococcal Meningitis.—There are two well-recognized forms of infection by this organism—the first occurs from time to time in an epidemic form; the second occurs sporadically, and is always present in England to a greater or lesser degree, especially during the earlier months of the year.

(A) *The Epidemic Form.*—Meningitis in an epidemic form occurs from time to time in this country. The last considerable epidemic occurred in Edinburgh, Glasgow, and Belfast, in 1907, and in the previous year in America.

SYMPTOMATOLOGY.—The disease often begins suddenly with fever, vomiting, pain in the head and the occipital region, and stiffness in the back of the neck. The head becomes retracted, and opisthotonos is present. The patient may be delirious, or may lie in a lethargic condition. A cutaneous eruption is often present, which somewhat resembles herpes, and in some cases this may become hemorrhagic. It is this symptom which has given to this disease the name "spotted fever." There is often very marked cutaneous hyperæsthesia.

Ocular paralysis in its various forms may occur, but optic neuritis is rare. Blindness is sometimes present. Deafness is a more common symptom, and may be followed by permanent deafness.

DIAGNOSIS.—The diagnosis is easily made by means of lumbar puncture and examination of the cerebro-spinal fluid. The fluid is always turbid, and contains a large excess of leucocytes, most of which are polymorphonuclear. The *Diplococcus intracellularis* of Weichselbaum is present, and can after suitable staining be seen both within and outside the leucocyte. Cultures can usually be obtained.

TREATMENT.—The treatment by intraspinal injection of Flexner serum obtained from a horse which had been immunized against meningococcal infection has proved most effective in diminishing the mortality from the disease. As shown by Flexner and Robb, the mortality, which without treatment amounts to 80 per cent., has by means of this line of treatment been reduced to 26 per cent.

(B) *The Sporadic Form.*—Meningococcal meningitis commonly occurs in infants and young children; 76 per cent. of the cases occur in children under a year old, and in the majority of these cases during the first six months of life.

The disease may arise quite suddenly in perfectly healthy children without any preceding illness. It is due to infection of the meninges by the meningococcus, but the path by which the infection reaches the brain and spinal cord is unknown. The nose, mouth, ears, and intestine, have been regarded as possible channels of infection, but none of these have yet been proved to be so.

The disease more frequently occurs during the winter and spring months of the year. Out of a series of eighty-five cases, fifty-four occurred during the five months January to May.

SYMPTOMATOLOGY.—The illness usually starts acutely with rise of temperature, vomiting, and restlessness. The child screams with pain and resists any movement of the head and neck. Convulsions may be an early symptom, and twitching of the arms and legs occurs. The child lies on his side with the neck rigid or the head somewhat retracted. The arms and legs are usually flexed in the earlier stages of the disease. The fontanelle is almost always bulged and tense. The child frequently lies with the eyes open and the eyelids retracted, and, though not unconscious, takes but little notice of objects placed before his eyes. He sometimes appears to be quite blind. The pupils remain active to light, even in cases where

the blindness seems to be complete. Squint may occur, but is relatively uncommon in this form of meningitis.

Hearing is usually acute. In some cases there is hyper-acuity; the slightest noise makes the child scream, whereas a bright light moved before the eyes produces



FIG. 71.—MENINGOCOCCAL MENINGITIS DURING THE ACUTE STAGE.

Some purpuric spots can be seen on the skin of the infant.

no effect. This symptom stands out in rather striking contrast to the deadness seen in association with the epidemic form of the disease. The child will in the



FIG. 72.—CHILD WITH ACUTE MENINGOCOCCAL MENINGITIS, SHOWING MARKED RETRACTION OF THE EYELIDS AND STARING APPEARANCE OF THE EYES, OFTEN ASSOCIATED WITH BRISSEN.

Compare the position of the eyes with the downward displacement seen in hydrocephalus (Fig. 115).

earlier stages of the disease usually drink and swallow well. The abdomen is not retracted. Respiration is but little accelerated, and the pulse is regular and rapid.

Rashes, to which so much attention has been fixed in the epidemic form, are less frequently seen in a sporadic form. A patchy erythema is not uncommon, a vesicular rash is sometimes present, and rarely a purpuric rash. The purpuric rash is peculiar; there is usually a vesicle, around which hemorrhage has occurred, with induration of the skin and subcutaneous tissue.

Joint swelling is in some cases present. The infection is generally periarticular rather than articular.

The course of the disease is most variable. In some acute cases death takes place in less than a week after the onset of the illness; in other cases the child, after remaining well nourished, but in a semi-conscious condition, for several weeks, gradually begins to waste. The head becomes more retracted, the back becomes arched so that the occiput and heels nearly meet, the arms become stiff and rotated inward, and the child dies in an emaciated condition. In other cases the child



FIG. 22.—MENINGOCOCCAL MENINGITIS DURING THE CHRONIC STAGE, SHOWING MARKED RETRACTION OF THE HEAD, OPISTHOTONOS, AND CONTRACTION OF THE LIMBS.

may recover after weeks of such illness, but in an idiotic or mentally defective condition. In other cases, after a few weeks' illness, complete recovery, both as regards their physical and mental condition takes place. Hydrocephalus, cerebral diplegia, and mental deficiency, are the result of this disease.

The course of temperature is subject to the most marked variations. In most cases it oscillates between 100° to 103° F., and lasts for a period of three weeks or more; in other cases there is a continuous high temperature between 103° to 104° F., lasting for many days, such as to suggest the possibility of typhoid fever, in other cases, again, there is a markedly swinging temperature, with a morning rise to 103° or 104° F., and with a fall at evening to 97° or 98° F.

The blindness which is present in this disease is a striking feature. It is often present as an early manifestation, and is complete. It is not due to optic neuritis or atrophy. Optic neuritis is a rare manifestation, the optic discs and the vessels of the fundus generally appear perfectly normal.

The child will sometimes remain permanently blind after general recovery has taken place. In some cases the blindness will persist for many weeks, and then sight may be recovered perfectly. The longest period that I have seen a child blind and recover good vision is four months.

Pseudo-glaucoma is a rare condition of the eye in which the vitreous becomes opaque and has the appearance of a glaucoma of the retina. Recently Coates and Forbes have supplied proof that the condition is sometimes due to meningococcal infection.

Kernig's sign may or may not be present. In late stages it is commonly present; in the early stages it is often absent. It is a sign of but little diagnostic value.

The blood generally shows a leucocytosis of 15,000 to 20,000 per cubic millimetre during the acute stages.

The cerebro-spinal fluid in the early stages of the disease is always turbid, and may vary from slight turbidity to thick pus. Sometimes it forms a clot spontaneously. It usually fails to reduce Fehling's solution.

On examination of the cell deposit, this is found to consist chiefly of polymorphonuclear leucocytes, with a smaller number of lymphocytes and other cells. The proportion of polymorphonuclear leucocytes varies with the stage of the illness, but in the acute stages is always between 65 per cent. and 90 per cent. In the later stages of the disease the fluid becomes less turbid, and the proportion of lymphocytes is increased. The fluid always contains an excess of albumin. The average amount, as estimated by Aufrecht's albuminometer, in normal cases is about 0.400 per cent.; in meningococcal meningitis it averages 0.44 per cent., and may amount to over 1 per cent.

In films made from the cell deposit, numerous intra- and extra-cellular diplococci can be found, and on culture a growth of the *Diplococcus intracellularis* can be obtained.

PATHOLOGY.—This form of meningitis is due to the invasion of the meninges by meningococci. Any portion of the membrane of the brain may be affected, but the disease usually falls with greatest intensity on the base of the brain, and especially on the region of the fourth ventricle around the base of the cerebellum. The meningitis may extend forward so as to involve the optic chiasma, but as a rule it is much less in this region than in cases of tuberculous meningitis.

The inflammation may in some cases spread over the whole vertex of the brain, and the surface of the hemisphere is seen to be covered with a thick green lymph on which engorged veins stand out prominently. The pus is held in the meshes of the pia arachnoid, so that washing does not readily remove it from the surface of the brain. The fluid in the lateral ventricle may be purulent, but, on the other hand, is in some cases perfectly normal.

The spinal cord invariably shares in the meningitis of the brain, being as a rule most marked on the dorsal surface of the cord in the cervical and lumbar regions, whilst the thoracic region remains fairly clear.

In those cases in which the disease has been of long duration the membrane of the brain presents an opaque appearance, as if the surface had been dented by flour.

Adhesions between the membrane and the brain in the region of the fourth ventricle are commonly present. This may lead to an obliteration of the fourth ventricle, or to the distension of the membrane, so as to form a "cistern."

containing a large amount of cerebro-spinal fluid. This "cistern" may or may not be in direct communication with the fluid in the lateral ventricle of the brain.

The microscopical changes in the brain tissue itself are very slight, and the infiltration of the meninges is almost confined to the subarachnoid space.

The organism which gives rise to this disease is the *Diplococcus intracellularis*, and was, so far as the sporadic cases are concerned, first described by Still. It is closely related to the *Diplococcus intracellularis* of Weichselbaum, but presents variations in regard to growth, fermentation of certain sugars, and possibly also in the opsonic reactions.



FIG. 74.—MENINGOCOCCAL MENINGITIS.

The whole vertex of the brain is seen to be covered with coagulation, on which the dilated, congested, and thrombosed veins stand out.

DIAGNOSIS.—The diagnosis depends upon the examination of the cerebro-spinal fluid obtained by lumbar puncture and identifying the organism.

The most characteristic symptom of this form of meningitis—viz., retraction of the head—may be present in other diseases, and is seen in association with otitis media, pneumonia (especially an apical pneumonia), retropharyngeal abscess, thrombosis of the longitudinal sinus, mumps, and enlarged cervical glands. When investigating a case in which retraction of the head is a symptom, all the above should be borne carefully in mind. The diagnosis of meningitis from otitis media or thrombosis of the longitudinal sinus is sometimes most difficult, and can only be made by an examination of the cerebro-spinal fluid and a careful consideration of all the symptoms.

PROGNOSIS.—The prognosis, both with regard to life and with regard to complete recovery, is by no means so unfavourable as in other forms of meningitis.

Probably about 50 per cent. of cases survive, and of these 15 per cent. make a complete recovery; the remaining 35 per cent. are left with blindness, hydrocephalus, and varying degrees of mental deficiency.

The duration of life in the acute cases is short, the disease running its course in one to three weeks. Other cases run a prolonged course of months, the child gradually passing into a condition of extreme emaciation.

TREATMENT.—The treatment of meningococcal meningitis by means of anti-meningococcal serum is that which is most likely to be attended by success. This success has been definitely proved in the case of the epidemic form, but the results are not so conclusive in the sporadic form.

The method of injection is as follows :

A lumbar puncture is performed, and the cerebro-spinal fluid allowed to escape until all the excess of pressure is relieved. The amount which escapes may be 50 to 60 c.c., and an endeavour should always be made to obtain at least 10 c.c., which is the amount of serum to be injected.

The serum to be injected should be warmed by placing the bottle into water at the temperature of 130° F. When the serum has been sufficiently warmed, it should be drawn up into a syringe also carefully warmed, and should be slowly injected through the needle which has been used for lumbar puncture. As soon as all the serum has been injected, the needle should be withdrawn and the puncture closed with collodion. The feet of the bed should now be raised so that the lumbar region is raised above the cervical region of the cord, in order that the serum, which is of a higher specific gravity than the cerebro-spinal fluid, may gravitate from the lumbar region towards the cervical region.

The anti-meningococcal sera which are on the market are those of the Lister Institute, of Flexner, Kolle and Wassermann, and Ruppel.

The injection should be repeated every day, and the cerebro-spinal fluid which is withdrawn on each occurrence should be carefully examined, for it furnishes the most useful record of the progress of the case. The cerebro-spinal fluid, which on the first examination has been turbid with numerous polymorphonuclear cells and numerous meningococci, becomes gradually clearer, and the meningococci fewer and more difficult to cultivate; and eventually the meningococci disappear and the fluid becomes clear, and contains a larger proportion of lymphocytes and less albumin.

The question of how many injections should be given will be raised. I have given six on six successive days with a favourable result. It is certainly well to continue the injections until the temperature has reached the normal. In successful cases this occurs in two to three days; in other cases the temperature remains raised for several days. It is of great importance to deal with the case in its early condition, and not after the disease has been in progress for two to three weeks.

With regard to other methods of treatment, it is important to keep the child as quiet as possible, and not to move it more than is absolutely necessary. A water-bed is often a great comfort. The child usually swallows well during the earlier stages of the disease, but in the later stages nasal feeding becomes necessary. Troublesome vomiting should be treated by washing out the stomach. The complete recovery from the results of this disease is only effected very gradually, and it is often many months, and even years, before the child is again perfectly

normal; periodic headache, attacks of vomiting, and irritability, being the most marked symptoms.

Pneumococcal Meningitis.—The division of pneumococcal meningitis into two classes—viz., primary and secondary—is an artificial one. In the so-called "primary pneumococcal meningitis" the source of the infection is not obvious, whereas in the "secondary" the meningitis is the terminal stage of a general pneumococcus infection from an empyema, purulent pericarditis, an infected joint, an otitis, or other source. The type of meningitis is, however, similar.

SYMPTOMATOLOGY.—The clinical features of a pneumococcal meningitis are those which are seen with other forms of meningitis—viz., drowsiness, vomiting, rigidity of neck, and convulsions. The symptoms differ according to the distribution of the disease. In some cases very few cerebral symptoms are present, or may only be present shortly before death occurs. A pneumococcal meningitis is not infrequently found unexpectedly on post-mortem examination where there were no symptoms during life to point to such a condition.

MORBID ANATOMY.—The appearance of the surface of the brain in acute pneumococcal meningitis is very striking. The whole vertex and base are seen to be covered with a thick, purulent, greenish exudation, which lies either in the meshes of the pia arachnoid or between it and the dura mater.

The lateral ventricle may contain pus. There is almost invariably a meningitis of the cord, which is especially marked on the dorsal surface and in the cervical and lumbar region, leaving the thoracic region comparatively little affected.

DIAGNOSIS.—The diagnosis of pneumococcal meningitis is made by examination of the cerebro-spinal fluid obtained by lumbar puncture and evidence of the presence of the pneumococcus. The cerebro-spinal fluid is always turbid, contains an excess of polymorphonuclear cells and excess of albumin.

PROGNOSIS.—It is probably true that pneumococcal meningitis is invariably fatal. It is possible that recovery may occur in a case with a circumscribed patch of meningitis due to a pneumococcal infection, but when once the whole cerebro-spinal system becomes invaded there can be no likelihood of recovery.

TREATMENT.—Relief of the intracranial pressure may be afforded by lumbar puncture, but no permanent benefit is likely to result. The injection of an anti-pneumococcal serum (Painé) may be tried. It may be effective in cases where the disease is limited to the meninges, but could not be expected to have any effect in cases in which the pneumococcal meningitis is a terminal manifestation.

Tuberculous Meningitis.—Tuberculous meningitis most commonly manifests itself during the second and third years of life, and in this respect stands in contrast to meningococcal meningitis, which is most common during the first year of life. It is due to the invasion of the meninges of the brain by tubercle bacilli, which are almost certainly carried into this situation by the blood-stream.

Tuberculous meningitis sometimes arises in children who would seem otherwise to be in perfect health, but in such cases one never fails to find on post-mortem examination some tuberculous focus in the body, generally in the thoracic or abdominal glands, from which the infection has arisen. In other cases the source of the infection is more obvious—viz., from the lungs, abdomen, ear, joints, or

benes. The acute specific fevers, and especially measles, would seem to be the exciting cause of the onset of tuberculous meningitis. Injury to the head also seems to precipitate an attack.

SYMPTOMATOLOGY.—Tuberculous meningitis commonly has a slow onset, even in those cases in which the symptoms are said to have come on acutely. A careful inquiry it is found that malaise or headache preceded the acute onset of the symptoms for some weeks.

Headache, loss of appetite, screaming at night, irritability, fever, and vomiting, are the earlier manifestations of the disease. The child wishes to lie down, dislikes the light, and from time to time screams either with pain in the head or on any slight disturbance. Constipation is a marked feature both in the earlier and later stages.

Gradually the child passes into a drowsy condition, from which it can only with difficulty be aroused. It will sometimes lie with the eyes open, taking no notice of surrounding objects or persons. A squint is often present. The pulse

at this stage becomes slow and irregular. Respiration is shallow, with an occasional long-drawn inspiration. This may assume the "Cheyne-Stokes" or gasped character.

The temperature is generally raised one or two degrees, but no characteristic feature can be said to be attached to the temperature of a case of tuberculous meningitis. Stiffness of the neck is a common manifestation, but marked retraction of the head is rare, and only occurs in those cases in which the meningitis is found to have a distribution around the fourth ventricle and base of the cerebellum.

Tremor of the limbs and spas-



FIG. 7A.—TUBERCULOUS MENINGITIS.

Photograph of a child during the later stages of the disease. The left hand was in constant rhythmic movement, and only a blurred image of it appears in the photograph.

modic twitching of muscles often occurs. An unusual manifestation is that of movements which exactly simulate those of chorea. These may be unilateral in distribution, and disappear after a few days and give place to the general rigidity which is a manifestation of the later stage of the disease.

Comas occur in all stages. They may be the first symptom of the onset, but are more frequent during the later stage of the disease.

The ocular symptoms are most variable. Almost any form of ocular paralysis may be present, but paralysis of the external rectus, giving rise to an internal strabismus, is undoubtedly the most common. Ptosis, paralysis of the muscles supplied by the third nerve, may be present, and such paralysis may be unilateral or bilateral.

The pupils may be dilated and fail to react to light, or may oscillate in a marked degree when light is thrown upon the retina. Hemianopia as a symptom of tuberculous meningitis has been noted, but only in older children.

Optic neuritis is not a common manifestation in the earlier stages of the disease.

but in the later stages some swelling is commonly present. Haemorrhages may occur, but are rare.

Tubercles of the choroid occur in a small percentage of cases of tuberculous meningitis, probably in about 7 per cent.; but since they are most easily missed in ophthalmoscopic examination of the fundus, this percentage may be too small. The presence of tubercles in the choroid is greatly in favour of a tuberculous invasion of the meninges, but they may be observed in cases of miliary tuberculosis in which no meningitis occurs.

Lumbar puncture and examination of the cerebro-spinal fluid is essential in all cases of tuberculous meningitis. The fluid usually spurts out under considerable pressure, it is clear or slightly turbid, has no visible deposit before centrifugation, but often forms a fine flocculent clot. It contains an excess of albumin (from 0.1 to 0.3 per cent.). The normal sugar is nearly always absent. It contains a high proportion of lymphocytic cells, 70 to 80 per cent. being of this nature; very exceptionally the polymorphonuclear are in excess. Tubercle bacilli can almost always be found in the fluid, though a careful examination for such is sometimes needed.

Von Pirquet skin reaction is often absent in cases of tuberculous meningitis. It is difficult to see why this should be so, considering the reliable results it gives with other forms of tubercle. Sugar is often present in the urine in the final stages of the disease.

MORBID ANATOMY.—The flattening of the convolutions and the sticky feel of the surface of the brain in tuberculous meningitis is very characteristic. Fine scattered tubercles can usually be seen in the pia mater, extending along the course of the vessels.

The Sylvian fissures are usually matted together, and in the situation and around the optic chiasma there is usually a considerable amount of greenish lymph.

Sometimes the exudation may be on the vertex of the cerebrum, sometimes on the under-surface of the cerebellum. A very common place to find fine miliary tubercles is on the dorsal surface of the cerebellum. The ependyma of the lateral ventricles often shows numerous tubercles on its surface. Dilatation of the ventricles and general softening of the *velum interpositum* and *foenic* is often present. It is rare for actual thrombosis of the middle cerebral artery to take place as the result of infiltration around this vessel, but sometimes obliteration does take place, leading to softening and to cerebral haemorrhage. It is not uncommon to find rounded masses of tuberculous substance, yellow in colour, and varying from the size of a pea to that of a hazelnut, scattered about the cerebral substance; these have generally during life given rise to no symptoms (Fig. 76).

Another form, less frequently met with than the above, is that of irregular masses of tubercles, often of wedge shape, apparently due to blocking of some small vessel and infiltration of the area with tuberculous matter.

The membranes of the spinal cord are almost always involved in tuberculous meningitis. The meningitis is not as a rule so marked as at the base of the brain, but scattered tubercles can always be seen over the pia arachnoid and along the posterior roots of the spinal cord.

DIAGNOSIS.—The early symptoms of tuberculous meningitis may give rise to difficulty in diagnosis, but as regards the later symptoms there is but little doubt, and any that remains is removed by the examination of the cerebro-spinal fluid obtained by lumbar puncture.

The irritability, loss of appetite, vomiting, headache, and lassitude, which

precede the onset of more definite symptoms, suggest the possibility of intracranial disease, such as tumour, abscess, thrombosis of cerebral sinuses, ear disease, and other forms of meningitis. These symptoms may also occur in the onset of general disease, such as typhoid and pneumonia, but even in the early stages of the disease the examination of the cerebro-spinal fluid will lead to a correct diagnosis.

PROGNOSIS.—There is but one prognosis in cases of tuberculous meningitis when once the diagnosis is confirmed by the presence of tubercle bacilli in the cerebro-spinal fluid. It is true that certain observers have recorded recovery from



FIG. 76.—HORIZONTAL SECTION OF BRAIN, SHOWING LARGE TUBERCULOUS MASSES ENBEDDED IN THE SUBSTANCE OF THE HEMISPHERES.

The child died of tuberculous meningitis.

tuberculous meningitis; there is, however, little doubt that in most of these cases there was an error in diagnosis. Martin investigated all reported cases of alleged recovery. In some twenty cases he found either that tubercle bacilli had been present in the cerebro-spinal fluid, or on post-mortem examination that old tubercles were present in the pia mater. These might be accepted as undoubted cases of recovery from tuberculous meningitis. Personally I have never seen a case of tuberculous meningitis recover. Most marked remissions in the severity of the symptoms occur, but the improvement is only temporary, never permanent.

Martin, in his investigations of reported cases of recovery at certain London hospitals, failed to find any case in which the diagnosis was not questionable.

TREATMENT.—Taking the view that tuberculous meningitis is always fatal, the treatment can be directed only to the relief of symptoms.

Lumbar puncture is of use in relieving the headache, and temporary improvement occurs after its performance. Bromide, chloral, and other drugs, relieve the headache and diminish the restlessness which is such a painful symptom in the child. The methods of treatment which were employed in those cases in which recovery took place were—iodide of potassium in drachm doses every four hours, leeches over the mastoid process, repeated lumbar puncture, and injection of a solution of iodine in iodide of potassium in the spinal canal.

Most of these methods I have tried, also decompression by removal of a large portion of the vault, injection of silver salts by lumbar puncture, inunction of mercury, and administration of tuberculin, all without any success.

Staphylococic and Streptococic Meningitis.—**Ætiology.**—This form of meningitis occurs in infants as the result of some infection of the umbilicus or of the skin. In older children it arises in connection with an otitis, with an empyema, an arthritis or erysipelas of streptococcal origin. Almost any form of streptococcal or staphylococcal infection may, in a child, terminate with a meningitis.

SYMPTOMS.—The symptoms of this form do not differ from other forms of meningitis. In some cases hardly any symptoms appear, and the condition is found unexpectedly on post-mortem examination.

DIAGNOSIS.—The diagnosis of this form of meningitis is not as a rule difficult, and the differential diagnosis from other forms of meningitis is made by the bacteriological examination of the cerebro-spinal fluid.

The greatest difficulty arises in distinguishing cases of cerebral or cerebellar abscess from those in which the infection is more general.

In abscess the symptoms are more localized, the headache, vomiting, and optic neuritis, more pronounced. The examination of the cerebro-spinal fluid may aid the diagnosis, for in meningitis the fluid is turbid and contains numerous cells and organisms, whereas in cerebral abscess the fluid is clear and only a few cells are present.

I have, however, seen cases of cerebral abscess in which the cerebro-spinal fluid resembled that of meningitis.

This form of meningitis is usually, if not always, fatal. There is no treatment which can be said to be of use, although the injection of an antistreptococcus serum into the cerebro-spinal canal might be tried.

Rheumatic Meningitis.—There is no doubt that meningitis occurs in the course of rheumatic fever and endocarditis. Whether such a meningitis is due to the *Diplococcus rheumaticus*, as described by Forryton and Paine, or to infection of the meninges by some other organism, it is difficult to say. Forryton and Paine believe that there is a rheumatic meningitis, and bring forward evidence in support of their contention which it is difficult to gainsay. Personally, I have never had under my care a case of rheumatic meningitis.

Influenzal Meningitis.—Influenzal meningitis is a rare affection, but the rarity of recorded cases in the past is probably due to the difficulty in recognition of the organism, and even at the present time it is by no means certain that the organisms

described in all cases of influenza meningitis is identical with that described by Pfeiffer.

Cohen in 1909 collected twenty-six cases which he is willing to accept as undoubted cases of influenza meningitis. Of these twenty-six cases, fourteen occurred in children under one year of age, all of whom died. Langer recorded the case of influenza meningitis in a boy aged nine years, who recovered. Eatten reported five cases of influenza meningitis in infants in 1910. All these occurred in the Children's Hospital, Great Ormond Street, during the years 1908, 1909, and 1910, and of these four died and one recovered.

SYMPTOMATOLOGY.—The clinical features of a case of influenza meningitis do not differ from those of other forms, the malaise, the fever, rigidity of the neck muscles, and the retraction of the head, being exactly like the symptoms seen in meningococcal meningitis.

The fluid obtained by lumbar puncture flows out under considerable pressure, is turbid, and on cytological examination the cell deposit is found to consist almost entirely of polymorphonuclear cells among which both extra- and intra-cellular organisms are found. Sometimes the meningitis seems to be part of an influenza septicæmia, or may arise from a joint infected with the influenza bacillus, as shown by Dudgeon and Adams.

MORBED ANATOMY.—The appearance of the brain and spinal cord do not differ from the other forms of infective meningitis, their surface being covered with a thick exudation.

DIAGNOSIS.—The essential difficulty in the diagnosis of influenza meningitis is the recognition of the



FIG. 37.—INFLUENZA MENINGITIS.

The base of the brain is seen to be covered with a thick exudation. The causative organism in this case was shown to be the influenza bacillus.

organism. Unless routine cultures are made on some blood-medium, the organism may be easily overlooked and the fluid reported as sterile. Cohen has described an organism which he says has been confused by former observers with the Pfeiffer bacillus, which it resembles most closely morphologically and in staining reaction, but which is pathogenic to guinea-pigs and rabbits, and against which it was possible to vaccinate an animal and obtain an antitoxic serum. Cases due to this organism might well be placed under a heading, "Meningitis due to an 'influenza-like' organism." Henry describes these cases under the heading "Meningitis due to Hemophilus Organisms."

PROGNOSIS.—Influenza meningitis is a most fatal disease so far as infants are concerned. In older children recovery has been recorded.

TREATMENT.—The only line of treatment which up to the present has been attended by success is that of the administration of hexamethylaminine, 20 grains being given every four hours to a baby under a year old. The rationale of this treatment is based upon the observation of Crowe: "That this drug appears in the cerebro-spinal fluid and exercises a decided inhibitory effect on the growth of organisms."

Meningitis due to Other Organisms.—Cases of meningitis due to infection with the *B. typhosa* and with *B. enteritidis* (Gaertner) occur in children.

This form of meningitis may occur in the course of an ordinary typhoid infection, or the infection may be primary in the meninges, and remain limited to this region.

Rocas and Carlos record eight cases of typhoid meningitis in children, all of whom recovered.

Meningitis may occur in association with mumps, with gonorrhoea, with *B. coli* infection, and actinomycosis, but the infection by such organisms must be very rare in childhood. With regard to mumps, although there is no doubt that in a small proportion of cases meningeal symptoms appear, yet there is no good evidence as to the nature of the causal organism, and such cases are better considered under the title of serous meningitis.

Syphilitic Meningitis.—Syphilitic meningitis and the condition known as "juvenile general paralysis of the insane" are so closely related that the conditions, so far as older children are concerned, will be described under the latter heading. There is, however, a condition of syphilitic meningitis in infants and young children which has all the features of a chronic meningitis, and gives a clinical picture which is quite distinct from that of juvenile general paralysis of the insane.

The condition is not common, and out of 100 cases of fatal meningitis, other than tuberculous, this form of meningitis occurred six times. It is probable that this hardly represents its frequency, for the cases run a long and chronic course, and would be discharged from the hospital as incurable before death occurred.

Ætiology.—The cause of this condition is congenital syphilis, and in nine out of fifteen cases recorded by Hanks the *treponema pallidum* was found. The presence of a positive Wassermann reaction in association with a high lymphocytic cell content in the cerebro-spinal fluid is essential to warrant the diagnosis of syphilitic meningitis.

Symptomatology.—During the first few months of life the infant may seem to be normal, but at such time as it should begin to hold up its head it is noticed that it fails to do so. At the same time it may be observed that the child does not take notice of its surroundings. As it grows, the head may appear larger than normal, but no great degree of hydrocephalus develops. The arms and legs may be rigid, and there may be comparatively little power of movement. Fits may from time to time occur.

A history of sudden onset of symptoms may be given, but on careful inquiry it is found that previous to such an onset, the child has been backward, and has shown other signs of cerebral defect. Such a symptomatology will not serve to distinguish a syphilitic meningitis from other forms of congenital cerebral defect; but the presence of manifestations of syphilis or of a syphilitic chorioiditis serves to distinguish syphilitic from such forms of cerebral defect. The examination of the cerebro-spinal fluid, both for the presence of a Wassermann reaction and for increased leucocytosis, helps to confirm the diagnosis.

PATHOLOGY.—The appearance of the membranes and of the brain is very characteristic.

The pia arachnoid over the vertex is opaque and thickened, often adherent to the cortex, from which it is separated with difficulty. The convolutions of the brain are shrunken, and the sulci wide. The surface of the convolutions have a parts the appearance of wash-leather. On section through the brain, the ventricles are found dilated, and there is a separation of the superficial layers of the cortex from the deeper layer by a loose areolar tissue, which contains a milky or clear fluid. This fluid drains away on section, and leaves a space between these layers.

DIAGNOSIS.—The diagnosis of a syphilitic meningitis is not difficult with the modern pathological method of examination. The child may or may not present the usual signs of congenital syphilis. The signs of meningitis are obvious, and

the cerebro-spinal fluid contains an excess of lymphocytes. Both the blood and the cerebro-spinal fluid give a positive Wassermann reaction.

PROGNOSIS.—When the diagnosis of syphilitic meningitis has been made, the prognosis as regards recovery is bad. The child may live for many months, but complete recovery does not take place.

TREATMENT.—Mercurial inunction and large doses of iodide may, in the early stages, be of some service. An intravenous injection of salvarsan should be tried. It is by no means easy to get a needle into the vein of a young child without first dissecting out the vein.



FIG. 78.—VERTICAL SECTION THROUGH THE BRAIN OF AN INFANT WITH SYPHILITIC MENINGITIS.

Note the wasted condition of the convolution, the dilatation of the ventricles, and the area of softening and cavity formation between the superficial and deeper layer of the cortex. This is well seen in the convolution to the right of the longitudinal fissure.

Serous Meningitis.—This name is given to a class of case in which there are symptoms of meningitis, and in which, on lumbar puncture, the cerebro-spinal fluid flows out under increased pressure. The fluid is clear, contains an excess of albumin, a small proportion of lymphocytes, but no bacteria. The fluid obtained from cases of tuberculous, syphilitic, and the late stage of meningococcal meningitis might be said to conform to this type; but such cases should not be included in this group, and the name should be reserved for those in which no organism can be found.

ETIOLOGY.—This form of meningitis commonly arises in association with other media, or some acute infection, and gives rise to alarming symptoms from which the child often makes a rapid and perfect recovery.

SYMPTOMATOLOGY.—The symptomatology can well be gathered from the following case: A child, aged five months, was taken suddenly ill on July 20. The throat was noticed to be sore on July 25, and on the following day the legs were rigid, and other signs of meningitis were present. The cerebro-spinal fluid examined twice during life showed that the fluid was clear, albuminous, had a slight leucocytosis, and was sterile. The child died on the twenty-first day after onset of the disease. No meningitis was present either on macroscopical or microscopical examination. There was a general streptococcal infection, and streptococci were also obtained from the cerebro-spinal fluid taken after death.

If this child had survived its general streptococcal invasion, there would have been no reason why it should not have completely recovered from its meningeal symptoms.

DIAGNOSIS is made by examination of the cerebro-spinal fluid. A clear fluid, which comes out under pressure, has an excess of albumin and excess of leucocytes, but no bacteria, is characteristic of serous meningitis.

PROGNOSIS.—The prognosis is relatively good. A considerable number of those cases in which symptoms develop in the course of otitis media, with or without operation, recover completely.

TREATMENT.—The symptoms are often rapidly relieved by the removal of the cerebro-spinal fluid by lumbar puncture.

Meningism.—A name applied to a group of cases which present symptoms of meningitis, but in which no pathological change can be demonstrated, either in the cerebro-spinal fluid or if death occurs in the meninges or cerebral tissue.

ETIOLOGY.—Meningism commonly arises in association with some acute febrile disease in children, and is due to the toxin which is present in this condition.

SYMPTOMATOLOGY.—The symptoms are those of meningitis: headache, vomiting, convulsions, squint, and rigidity of the trunk and limbs. There may be coma or a period of restlessness and irritability. The symptoms may last a few days, after which the child makes a complete and rapid recovery.

DIAGNOSIS.—The presence of the signs of meningitis and the absence of any change in the cerebro-spinal fluid are the features on which the diagnosis of meningism is based.

PROGNOSIS.—Meningism is an alarming condition, and a favourable prognosis can only be given when the diagnosis is certain. Recovery usually takes place, and is rapid and complete.

TREATMENT.—Apart from the treatment of the general symptoms, restlessness, convulsions, and hyperpyrexia, if such exists, but little needs to be done. Lumbar puncture is necessary for diagnosis, and is also of service for the relief of pressure.

REFERENCE.

HENRY, H.: *The Journal of Pathology and Bacteriology*, 1912, xvi, 174.

INTRACRANIAL ABSCESS.

INTRODUCTION AND ÆTIOLOGY.—Apart from ear disease, cerebral and cerebellar abscess in childhood is rare.

It may, however, occur from pneumococcal, streptococcal, and other infections, derived from the nose, and bones of the skull, from general infections, and may follow injury.

SYMPTOMATOLOGY.—The symptoms produced by an intracranial abscess are those of increased intracranial pressure, and do not differ from those caused by a tumour, except in their more acute onset. Headache, vomiting, and optic neuritis, are commonly present, the pulse may be slow, and the temperature persistently subnormal. As long as the abscess is within the brain, and there is neither meningitis nor sinus affection, the temperature usually remains subnormal; but if thrombosis or meningitis occurs, it is liable to extreme variations.

The localized symptoms produced by an abscess depend on its situation, and these symptoms are discussed under the "diagnosis of intracranial tumours."

Various stages have been described in intracranial abscess—a latent stage, an initial stage, a full stage, and a terminal stage. No great advantage is derived from such a division, either for clinical or descriptive purposes.

The early symptoms of headache, malaise, and irritability, pass gradually into those of drowsiness, with slowing of the pulse, and later into deeper coma, slow and "grouped" respirations, and convulsions.

Since the commoner situations for an abscess are the temporo-sphenoidal lobe and the lateral lobe of the cerebellum, it should be borne in mind that the former commonly give rise to pyramidal symptoms at an earlier stage than the latter, and alterations in the character of the reflexes on the one side—viz., the increase of knee-jerks, the absence or diminution of the abdominal reflex, and the presence of an extensor plantar response—would point to a temporo-sphenoidal abscess; whilst the presence of ataxia on one side and nystagmus to the same side would point to a cerebellar affection.

Owing to the pressure on the medulla produced by a cerebellar abscess, there is a great tendency for respiration to fail, whilst the cardiac action is good, and it is this sudden failure which is the cause of death in some cases. Artificial respiration is able to keep these patients alive for many hours after all normal respiration has ceased.

In some cases an intracranial abscess will run a most chronic course, the abscess becoming encapsulated with a very thick wall. Such cases present the symptoms of an intracranial tumour.

Multiple abscesses most commonly occur in general infection, and not in localized infections derived from the ear.

DIAGNOSIS.—The greatest difficulty in the diagnosis of an intracranial abscess arises in its distinction from meningitis.

Both may have a more or less acute onset, and both may have been preceded by ear discharge. Headache and vomiting occur in both. Optic neuritis is an earlier and commoner manifestation in abscess than in meningitis.

Convulsions occur in both conditions. A rise of temperature is commonly

present in meningitis. A persistent subnormal temperature is an important sign indicative of cerebral abscess.

Lumbar puncture and examination of the cerebro-spinal fluid will decide if the meninges are affected or not; but it must be remembered that in some cases of cerebral abscess the cerebro-spinal fluid will show an increased number of leucocytes, an increase of albumin, and possibly the presence of an organism; therefore, a too hasty judgment must not be made on the examination of the cerebro-spinal fluid alone.

Shortly, it may be said that the rapid onset of intracranial symptoms, the presence of optic neuritis, a subnormal temperature, a slow pulse, a normal or but little altered cerebro-spinal fluid, and localized cerebral or cerebellar signs, are in favour of an intracranial abscess.

PROGNOSIS.—If the diagnosis can be made early, the abscess localized and efficiently drained by operation, the prognosis is not unfavourable. Many cases of cerebral abscess, however, die from respiratory failure, some quite suddenly.

TREATMENT.—Operation and draining the abscess cavity is the only method of treatment.

CEREBRAL EPENDYMITIS ("ACUTE HYDROCEPHALUS").

INTRODUCTION.—Inflammation of the lining membranes of the ventricle is a relatively common condition when associated with inflammation of the membranes of the brain, but a rare condition when it occurs apart from meningitis. It might even be questioned whether it ever occurred, but the evidence accumulated during the past years by P. Merle makes its occurrence certain.

ÆTIOLOGY.—The causes of an ependymitis are the same as those of a meningitis—viz., tubercle, syphilis, and infection by the pneumococcus, the meningococcus, and many other organisms.

SYMPTOMATOLOGY.—The clinical symptoms resemble in the acute cases those which occur in meningitis, whilst in the more chronic cases they resemble those of tumours. In the acute cases there are the usual signs of increased pressure, hydrocephalus, headache, vomiting, and rigidity of the neck and limbs. Paralysis of the ocular muscles is frequent. Early and intense optic neuritis with hemorrhage is a striking feature, and in contrast to the late onset and somewhat slight neuritis seen in meningitis. Loss of vision and early atrophy of the discs is liable to occur.

The cerebro-spinal fluid obtained from lumbar puncture is quite clear, may contain some excess of albumin, and be under some increase of pressure, but contains no organism, and has a normal appearance. The cerebro-spinal fluid obtained from the puncture of the ventricle has, on the other hand, the characters indicative of inflammation, a lymphocytic or polymorphonuclear increase, and organisms may be present.

In the chronic cases the symptoms resemble those produced by an intracranial tumour. There is a gradual onset of weakness, sometimes limited to certain cranial nerves on one side of the body, at other times giving rise to a hemiplegia with rigidity or flaccidity of the limbs. Such symptoms suggest the probability of a localized growth. The cardinal symptoms of an intracranial tumour may be

present—viz., headache, vomiting, and optic neuritis, but the optic neuritis has not the intense character seen in the acute variety, and it is less common. An ependymitis localized to the fourth ventricle may give rise to bulbar symptoms, tachycardia, respiratory disturbances, and polyuria.



FIG. 79.—Cerebral Ependymitis: Horizontal Section of the Brain, showing Pus within the Ventricles and Inflammation of the Ependyma.

The organism in this case was the *Bacillus influenzae*.

PATHOLOGY.—The ependymal surface and the subependymal tissue may show active granulation, perivascular infiltration, and hemorrhages, or degenerative changes, with shedding of the epithelium. The choroid plexuses may be involved.

DIAGNOSIS.—The diagnosis is most difficult, and depends upon the cerebro-spinal fluid obtained from a lumbar puncture being normal, whereas that obtained from the ventricle is abnormal. In a child in whom the fontanelle is widely open

and the ventricle distended, there is but little difficulty in withdrawing the fluid from the ventricle; but it is an operation which is by no means free from risk, for the needle may easily puncture a vein in the wall of the ventricle, and give rise to an extensive and fatal hæmorrhage.

Apart from such a ventricular puncture, the diagnosis must depend upon the rapid onset of signs of increased intracranial pressure, associated with an intense optic neuritis.

PROGNOSIS.—The prognosis in a case of ependymitis depends to some extent on the nature of the infection. If the meningococcus is the cause of the disease, the injection of the antimeningococcal serum into the ventricles is said to have been followed by recovery. Personally, I have never seen a case recover in which there was proof that the ventricles were affected.

TREATMENT.—If the diagnosis can be made, there are only two lines of treatment available: the one by injection of the antimeningococcal serum into the ventricle in cases which can be shown to be of meningococcal origin, and, second, the opening of the skull and drainage of the lateral ventricles.

REFERENCE.

MULLER, P.: Thèse de Paris, 1910.

POLIOENCEPHALO-MYELITIS — POLIOMYELITIS — INFANTILE PARALYSIS — HEINE-MEDINISCHE KRAANKHEIT — EPIDEMIC PARALYSIS.

INTRODUCTION.—The clinical conception of few diseases has altered more in the last decade than that of acute poliomyelitis. Formerly regarded as an acute disease of the spinal cord, it is now recognized as one of the acute specific fevers, having an incubation period of from four to seven days, and liable to be communicated from person to person. The tendency of the disease is to attack the nervous system. Any portion of the system may be affected and give rise to a variety of clinical symptoms dependent on the situation of the lesion.

The names under which the disease has been recognized are numerous. Some are given above. None are altogether satisfactory. Undoubtedly "poliomyelitis" is that in the most general use, but such a name fails to recognize the important class of case in which portions of the nervous system other than the spinal cord are involved.

The name "polioencephalo-myelitis" gives some indication of the nature of the disease, and the wide extent in which it may affect the nervous system; but it is cumbersome, and the name "acute poliomyelitis and polioencephalitis" is the name under which the disease has been made notifiable, and that which is probably best to adopt.

ÆTIOLOGY.—The disease is one which tends to affect children between the ages of one to three years more than at any other period of life. Cases have been recorded in which it has been noticed in the first few weeks of life, and there is some evidence that it may even occur during intra-uterine life. After the age of three years the incidence of the disease rapidly falls, and the proportion of cases after the eighth year of life is small.

In the northern hemisphere it always occurs in its epidemic form during the

summer months of the year—*i.e.*, June, July, August, and September—the month of August being the month of greatest prevalence.

In Australia the months of prevalence are March and April. In South Africa no record of any epidemic has been recorded, but that it is and has been from time to time very prevalent is undoubted, and the cases which have come under my notice have occurred in or about the month of December.

Some sixty or more epidemics have been recorded in various parts of the globe. One of the earliest was that described by Medin, in Stockholm, in 1887, although there are scanty records of epidemics prior to this. Norway and Sweden have suffered severely, but Germany, Italy, France, America, and Australia, have all had epidemics. Two of the largest occurred in Norway and Sweden in 1905, and in New York and Massachusetts in 1907 and 1909. The disease is not confined to large towns, but occurs in country districts and small villages. It is always epidemic in London during the summer months, but it is impossible to obtain reliable figures as to its relative prevalence in different years. Certainly in the summer of the year 1904 it was widely prevalent in London, and in the year 1910 and 1911 there were several epidemics scattered about England and Scotland.

Association with other diseases is not striking. In the great majority of cases the children were and had been in good health at the time when they were taken ill.

The actual cause of the disease has been shown by Levaditi, Landsteiner, Popper, Flexner, and Lewis, almost simultaneously and independently to be due to a poison, possibly an ultra-microscopic organism, which cannot be cultivated on any of our present media. It will pass the finest filter, and will withstand glycerination for several months. In all these respects the poison closely resembles that of rabies.

Under ordinary circumstances the monkey and ape are alone susceptible to the poison, rabbits, guinea-pigs, and mice being immune, though Marks has shown that rabbits may transmit the disease.

The infection of the monkey may be brought about by injection of the virus into the brain, peritoneal cavity, the general circulation, or subcutaneous tissue, but however brought about the infection falls upon the brain and spinal cord.

The period of incubation on experimental evidence is from eight to twelve days, but on evidence derived from clinical observation probably about four days. There is a considerable variation in the experimental results depending on the method of injection, the dilution of the virus, and the method of treating the virus previous to injection. There is good evidence that the infection may be carried by a third person.

There is as yet no evidence that the infection is carried by animals, although the likeness of the poison to rabies suggests such a possibility. Animals have been noticed to be paralyzed at the same time as the affection has occurred in man, but there is no evidence that the condition which caused their paralysis was poliomyelitis. Although infection must be regarded as the causative factor in the disease, yet the exposure to cold and prolonged exertion, especially in relation to bathing, must be looked on as factors in determining the onset. The instances in which more than one member of a family are affected are not numerous when considered in relation to the number of cases occurring, but in forty instances of such family affections—in thirty-one families two members were affected, in five families three members were affected, in three families four members were affected, and in one instance seven members were affected.

Various factors, such as atmosphere, temperature, humidity, the presence of domestic animals, the nearness of water, the sanitary condition, the presence of venom, have all been investigated in the Massachusetts epidemic in 1909, but with a negative result.

SYMPTOMATOLOGY.—In dealing with the symptomatology of this disease it is convenient to describe the symptoms according to the portion of the nervous system involved, and for this purpose the division suggested by Wickman will be adopted. It must, however, be remembered that though for the sake of clinical description the divisions are thus made, yet many cases arise which exhibit the characteristic features of two, three, or even more divisions.

The divisions are—

- The spinal form.
- The ascending and descending form.
- The bulbar and pontine form.
- The cerebral form.
- The cerebellar or ataxic form.
- The neuritic form.
- The meningitic form.
- The abortive form.

The symptoms of each of these will be described separately, but there are certain general characteristics which may well be considered together.

GENERAL SYMPTOMS.—The disease usually begins with a sudden onset; there is commonly fever of a moderate degree, which lasts for two to three days. The child is often drowsy, complains of headache and pain in the limbs, which may be so acute as to give rise to the impression that the child has rheumatism. The pain is greatly accentuated by any movement, and even the approach of an individual to the bed will often make the child cry out in fear of any movement. In some few cases vomiting and diarrhoea may occur.

The onset of the disease is sometimes attended by convulsions, and such convulsions may occur in cases which at a later stage show no sign of cerebral involvement.

The neck may in some cases be stiff and even retracted, and any movement forward causes pain. In the drowsy condition the child may pass urine and faeces involuntarily, but even when no such drowsiness exists, loss of control over the sphincters or retention of urine may be present, and last for a few days.

Hyperæsthesia of the skin is said to be present in some cases, but whilst admitting that the child resents the least movement, it has always seemed to me doubtful if this should be described as hyperæsthesia. Rash is not uncommonly seen, there may be general erythema of the skin, and this may give rise to the suggestion of scarlet fever. I have seen a vesicular rash which resembled a rather diffuse herpes, and one which was certainly not varicella nor a sudamina. In some cases there is profuse sweating.

The occurrence of herpes in relation to poliomyelitis is well recognized, but it is curious how seldom a true herpes is seen in a case of poliomyelitis.

It is undoubtedly true that in most cases of poliomyelitis no alteration of sensation occurs; as a rare manifestation, however, it must be admitted that there may be complete loss of sensation in the distribution of or below the level of the

lesion. In most cases, after a few days of acute illness, the general health is restored, but the child is left with a flaccid paralysis of one or more limbs. On the other hand, a raised temperature may persist, and only resume a normal level after five to six weeks.

In some cases, and especially those in which the temperature persists, there is a tendency for the paralysis to extend: these cases have been designated by Barlow as the "jump" cases—i.e., after an interval of a few days or weeks a sudden increase of the paralysis occurs. They are undoubtedly closely related to the form of ascending or descending paralysis described by Wickman as resembling Landry's paralysis.

Another type of onset must be recognized in which a child is put to bed apparently in perfect health, and on the following morning a flaccid paralysis of one leg is noticed. There is no pain on passive movement, and there is apparently no constitutional disturbance.

Allen Starr states that, in 100 consecutive cases, 66 began with fever and 23 began without fever.

The Spinal Form.—The disease as it affects the spinal cord may give rise to a paralysis of almost any individual group of muscles.

There is no doubt that the muscles of the leg below the knee are the most commonly affected, and the anterior tibial group seem especially liable to be involved.

Both legs may be simultaneously affected, and there may be a complete flaccid paralysis of both legs below the hip, or certain groups of these muscles may be paralyzed, leaving others in a normal condition.

Such localized weakness of muscles gives rise to a variety of deformities dependent on the unantagonized contraction of the normal or partly paralyzed muscles.

Owing to the flaccid and wasted condition of the muscles, the limbs can often be placed into the most extraordinary positions in relation to the trunk. In these cases in which the upper lumbar segments of the spinal cord are involved there is weakness of the muscles of the pelvic girdle, so that the child is unable to extend the trunk on the thighs, or to flex the thighs on the abdomen.

In those cases in which the thoracic segments of the cord are involved, paralysis of the abdominal and intercostal muscles results. The involvement of the cord may be unilateral, and such a lesion gives rise to the localized weakness of the abdominal oblique muscles on one side. The protuberance of the intestine which results from the weakness of these muscles gives rise to the appearance of an abdominal tumour, and on coughing or on any exertion which puts the abdominal muscles into action this protuberance becomes more marked.

Again, if the intercostal muscles are involved, respiration is carried on by the diaphragm and accessory muscle, or in the event of one side of the cord alone being affected by the intercostal muscles of the opposite side.

The dorsal muscles of the back may also be affected, and such weakness, either unilateral or bilateral, gives rise to a curvature of the spine, the variety of the curve depending on the muscles and the extent to which they are paralyzed.

In cases in which the cervical region of the cord is involved paralysis of the muscles of one or both arms occurs. The muscles of the arms most commonly paralyzed are those about the shoulders, the muscles of the forearm and hand remaining relatively intact. It is most unusual to see the small muscles of the

hand paralyzed by poliomyelitis, whilst the shoulder muscles remain normal. When the small muscles of the hand are paralyzed, it is usually in association with a complete flaccid paralysis of the whole arm.

The muscles of the neck may be involved so that the child is unable to hold up, or even to move, the head. Such a condition as an isolated paralysis is rare, and these cases in which the cervical region is affected are commonly fatal, owing to the involvement of the respiratory muscles.

Paralysis of the diaphragm, either unilateral or bilateral, may occur; both are rare except in the acute and rapidly fatal cases.

Paralysis of the cervical sympathetic, with narrowing of the palpebral fissure and smallness of the pupils, is also a rare manifestation of poliomyelitis in the lower cervical and upper dorsal region of the cord.

During the acute stage of the disease all the deep reflexes of the affected



FIG. 50.—LATERAL CURVATURE OF THE SPINE PRODUCED BY POLIOMYELITIS AFFECTING THE DORSAL MUSCLES OF THE BACK.

In this case the arms and thoracic muscles were unaffected, whilst the legs were greatly affected.



FIG. 51.—POLIOMYELITIS AFFECTING THE SHOULDER AND UPPER ARM MUSCLES OF BOTH SIDES.

Note wasting of shoulder and upper arm muscles.

limb or limbs are abolished, and generally the superficial reflexes are also absent.

As recovery takes place the superficial reflexes tend to return, but the deep reflexes remain long absent, even after a considerable amount of voluntary power has returned in the muscle.

In some cases the plantar responses would seem to be extensor; such a phenomenon may depend on the fact that the flexor muscles of the toes are paralyzed, whilst the extensors remain intact, or the phenomenon may depend upon some involvement of the pyramidal tract in the cord.

The abdominal reflexes are commonly present, unless there is evidence of involvement of the segments of the cord supplying the abdominal muscles.

After the acute stage of the disease is past, which may last from a few days to a few weeks, a second stage is entered upon in which there is a gradual return of power in the paralyzed muscles, some of which may wholly recover, leaving others more or less completely paralyzed. It is exceptional for complete recovery to take place. The writer has seen a few such cases. When, however, such a recovery takes place, it always does so rapidly—i.e., within four to six weeks of the onset. During this second stage also the affected muscles begin to waste, contractures begin to occur, and the vasomotor phenomena—i.e., coldness and lividness of the extremities—make their appearance.

In young children the wasting of the muscles is covered up by the large amount of subcutaneous fat, and does not become apparent until the child begins to lose this excess of fat, when it is often brought by its parents for a wasting of the limb, which is said to be progressive.

It cannot be denied that as a rare event progressive muscular atrophy does occur in a limb which has been affected by poliomyelitis, but such atrophy does not occur until adult life is reached, and I know of no example of a progressive muscular atrophy supervening on an acute poliomyelitis in a child.

It is during the so-called "second stage" of the disease that recovery gradually takes place in the muscles which have been completely paralyzed. This improvement may extend over a very considerable period, and even two years after the onset of the disease improvement may take place, especially in those cases in which a continuous line of treatment has not been adopted. In the case of a child who has been carefully treated for that length of time but little improvement is to be expected.

The final stage of permanent paralysis is now reached, in which no further recovery takes place, but the limb develops with the growth of the child. In some cases arrest of growth occurs, and the paralyzed limb remains as an atrophied appendage to an otherwise well-developed body.

A few further points need special mention in connection with the spinal form of the paralysis.

Sensation.—It may be stated as a general rule that sensation in cases of poliomyelitis is never affected, but, on the other hand, it must be admitted that loss of sensation does rarely occur, either in the form seen in a transverse lesion of the cord or in that of a peripheral neuritis. In the former it usually remains complete to all forms of sensation; in the latter it usually clears up rapidly.

Electrical Reactivity.—It is curious how rapidly the electric excitability of the paralyzed muscles is lost to faradic stimulation. Even in a few days of the onset of the paralysis the muscles, if tested, will be found not to react to a powerful faradic current. The galvanic excitability will remain brisk for some time.

In the later stages of the disease the paralyzed muscles will not respond to a faradic stimulus, and may or may not respond to a galvanic. The response to the galvanic is often slow and prolonged, and the contraction obtained at the anodal and kathodal closing currents are equal and similar in character. Thus it may be said that the reaction of degeneration is present.

As recovery gradually takes place a return of the normal reaction may occur, but the return of an active contraction of a muscle to the faradic current may be delayed long after there is very fair voluntary power.

The presence of a good contraction to faradism in a paralyzed muscle may be taken as a sign that recovery will take place. The presence of a contraction to galvanism in a paralyzed muscle cannot be regarded as a sign on which a favourable prognosis may be given.

Ascending and Descending Form (wrongly called "*Landry's paralysis*").—In connection with the spinal form of the disease must be mentioned those cases of ascending or descending paralysis which have been described by some writers under the title of "*Landry's paralysis*."

Such cases start with the usual constitutional symptoms, weakness appears in the legs, generally in the muscles about the hip, and extends to the knees and toes.

The abdominal muscles then show weakness, and the paralysis gradually extends and affects the muscles of the arm and neck, the diaphragm and intercostal muscle now being paralyzed; respiration is carried on only by the accessory muscles, which likewise soon fail, and death supervenes without any loss of consciousness. The disease may run its whole course in twenty-four to forty-eight hours, but, on the other hand, it may last from seven to ten days. Sensation is preserved throughout, although the deep and superficial reflexes are abolished. In some cases the weakness has started in the arms, and has spread downwards and upwards from that point.

These cases do not always prove fatal, for after the disease has reached a certain level arrest takes place, and gradual recovery follows. In other cases an arrest of the disease occurs, and the symptoms remain stationary for some days, then a sudden extension of paralysis occurs, which may prove fatal. These cases have been called the "jump" cases, from the fact that the paralysis tends to increase by jumps. Similar clinical features have been noted in monkeys, in whom experimental poliomyelitis has been induced (Roemer).

The Bulbar and Pontine Form.—In this type of the disease one or more of the cranial nerves are affected either singly or in association with the spinal type of the disease. There is no doubt that the seventh cranial nerve nucleus, giving rise to weakness of the face, is more commonly affected than any of the other cranial nuclei. When such a weakness occurs in association with a typical paralysis in some other portion of the body, there is no reasonable doubt as to the causation, but when it occurs as an isolated paralysis it is difficult to differentiate from forms of facial paralysis due to affection of the peripheral nerve dependent on one or other disease.

The close proximity of the nucleus of the sixth nerve to that of the seventh renders it likely that both might be involved in the same lesion, and such an association of paralysis has been noted. On the other hand, there is good pathological evidence that the seventh cranial nerve nucleus may alone be affected.

It must be remembered that an affection of the sixth nerve nucleus gives rise to loss of conjugate movements of the eye to the affected side, and not to an isolated paralysis of the external rectus muscle.

Cases of paralysis of the external rectus muscle of one eye have been described as cases of polioencephalitis, although not denying the possibility of a lesion in that region of the medulla giving rise to such a paralysis, yet it is far more likely that such paralysis is due to pressure on the sixth nerve itself by effusion at the base of the brain, or, as has been shown by Cushing, by the inferior cerebellar artery as it crosses the sixth nerve. Such cases of isolated paralysis of the external

rectus should not be accepted as due to poliomyelitis without pathological proof or their occurrence in association with other signs of acute poliomyelitis.

The affection of the oculo-motor nuclei gives rise to a paralysis of the extrinsic and intrinsic muscles of the eye, which may be complete or partial—unilateral or bilateral. Ptosis, loss of power in the movements of the eye, dilatation of the pupil, and loss of reaction to light and accommodation, may be present. Loss of vision may occur concomitantly with the ocular paralysis, and is presumably due to the interruption of the visual fibres in the region of the external geniculate bodies. Out of 488 cases of all forms of poliomyelitis observed by Wickman, the ocular muscles were affected in 9. In 6 of these cases the paralysis was associated with paralysis in other parts of the body, whilst in 3 the ocular paralysis was the only manifestation.



FIG. 82.—POLIOMYELITIS GIVING RISE TO LEFT FACIAL PARALYSIS.

The child had, in addition to the above, paralysis of the neck and shoulder muscles on the right side, and her brother was simultaneously affected with the same disease.

Complete recovery of both vision and ocular paralysis may occur, or the ocular paralysis may clear up, leaving the patient completely blind.

Weakness of the motor portion of the fifth nerve has been observed in connection with other lesions. Such a weakness is shown by a paralysis of the masseter and temporal muscles, and by deviation of the jaw to the affected side on opening the mouth.

Facial paralysis of one side may be associated with hemiplegic weakness of the opposite side of the body, and a single lesion to cause such a condition must be situated in the lower portion of the pons.

Affection of the nuclei of the sixth, tenth, eleventh, and twelfth cranial nerves gives rise to paralysis of the palate, larynx, sterno-mastoid, tongue, and difficulty in phonation and deglutition.

It is not uncommon to see the affection of these muscles on one side in association with a poliomyelitis of the cervical region of the cord. An extensive affection of the bulb necessarily leads to a rapidly fatal result. Atrophy of one half of the tongue, paralysis of one side of the palate, and abductor paralysis of one vocal cord, may be the result of an attack of poliomyelitis. The occurrence of a sudden bilateral nerve deafness suggests the possibility of a poliomyelitis as a cause. Cases of this nature are seen in children, but I know of no pathological evidence to prove that they are due to such a lesion.

The Cerebral Form.—The affection of the cerebral hemispheres gives rise to a variety of symptoms dependent on the portion of the hemisphere affected.

The Frontal Region of the Hemisphere.—There are cases in which a perfectly healthy child is suddenly taken ill with some acute febrile illness, ushered in by fits and attended by loss of consciousness; on recovering consciousness the child screams, fails to recognize parent or nurse, becomes dirty in habits, destructive, and fails to speak intelligibly.

Such a history suggests the possibility of some acute infective condition, and the case is usually assigned to the group "meningitis." It seems reasonable to regard some of such cases as possibly due to a poliomyelitis of the frontal region of the hemisphere, but there is at the present time no definite pathological evidence to prove the assumption.

The Motor Region of the Hemisphere.—Hemiplegia is probably the commonest manifestation of the cerebral form of this disease. The onset may be rapid or it may be gradual, extending over several hours or even a few days. It may be associated with aphasia and deviation of the head and eyes to the side, loss of consciousness, and convulsions. The hemiplegia may be transient or may persist, and be followed by contraction and atrophy. Poliomyelitis must not, however, be regarded as a common cause of infantile hemiplegia. Out of 100 consecutive cases of hemiplegia in children collected for the purpose of investigating this point, in not more than 10 per cent. could the hemiplegia on clinical grounds alone be attributed to poliomyelitis. The hemiplegia is accompanied by the usual alteration in the reflexes. In some cases the symptoms may be bilateral and give rise to the clinical picture of a cerebral diplegia.

The Occipital Cortex of the Hemisphere.—The evidence that the occipital cortex may be affected is based solely on clinical and not on pathological grounds.

The symptom produced by such a lesion is blindness, which may be complete or hemianopic. The disease may be ushered in with convulsions and a period of unconsciousness, and when consciousness returns the patient is completely blind. The pupils, however, react to light, in whichever direction this is thrown on the retina. In such cases recovery may take place, so that in three to six weeks the vision is perfectly restored, or permanent blindness may result.

The Cerebellar or Ataxic Form.—The characteristic feature of this form of acute poliomyelitis is ataxia of a cerebellar type.

The onset may be attended by headache, fever, vomiting, loss of consciousness, and convulsions, such as occur in the other forms of the disease. After consciousness has been recovered, the ataxia is usually first noticed in the arms, owing to the difficulty which the child has in feeding itself. The ataxia may affect one side only, or may be bilateral. If the child attempts to grasp an object, wild inco-ordinate movements result, which only stop when the desired object is grasped. On any further movements being attempted the wild oscillations begin again, and are only arrested on the object being attained. Whilst at rest no movements are present.

If the child is sat up, he oscillates from side to side, the oscillation becoming wider and wider till he overbalances. The head, too, shakes in a to-and-fro direction. If such a child is stood up on his legs, he will be able to support his weight so long as the person holding the child maintains his balance; but if the child is left to stand by himself, he at once assumes a "wide base"; and if an attempt to walk is made, the most wild ataxia results. There is no actual loss of power in the limbs, all movements being capable of being performed, but in an ataxic manner.

The intelligence is not impaired. The articulation is often affected, so that the child speaks in a slow, jerky manner with a monotonous voice. Nystagmus is sometimes present.

It must be recognized that the ataxia is not always due to a lesion in the cerebellum itself. It is the executive function of the cerebellum which is at fault, and an interruption of paths leading from the cerebellum either in the mid-brain

or in the medulla may cause the ataxia just as well as a lesion in the cerebellum itself.

In some cases ataxia is seen in association with the manifestation of poliomyelitis of the cord, or with the affection of other cranial nerves. The following case illustrates such an association: A boy had sudden paralysis of the left side of the face, with ataxia of the left arm and leg. At the same time his sister had paralysis of the left side of the face and a flaccid paralysis of all the muscles of the neck. In the boy the ataxia rapidly cleared up, but the facial weakness slowly recovered. In the girl the weakness of the neck muscles was more or less permanent. Numerous instances of ataxia in association with poliomyelitis are on record, both in the same individual and also in other members of the same family.

These cases run a variable course. Some rapidly recover, so that in a few days or weeks the patient is perfectly well; others only make a complete recovery after several months or years. Some cases never recover; in these cases it is probable that there has been, in addition to the damage to the cerebellar tracts, some damage to the motor path. It has been shown experimentally that the ataxia due to a lesion of the cerebellum rapidly clears up, whereas if the motor path is damaged at the same time, recovery does not take place.

The Neuritic Form.—It is doubtful whether at the present time it is possible to assert that the poison of poliomyelitis falls upon the peripheral nervous system only.

If the presence and persistence of pain are to be taken as evidence of the "neuritic type" of the disease, then it may be asserted that there is no justification for the title, since both these symptoms are well-recognized accompaniments of the affection of the spinal cord. On the other hand, if there is evidence of a weakness and wasting of the peripheral muscles, which affects all the extremities more or less asymmetrically, and at the same time there is definite alteration of sensation in the peripheral portion of the limb, with tenderness of the muscles, then it would be justifiable to regard such a case as one of neuritis. If such a case occurred with a sudden onset, or in association with typical cases of poliomyelitis in other members of the same family or household, it would be justifiable to assume that the cause was due to the same poison. Wickman quotes a case which would seem to be of this nature, and gives an account of others, but the evidence on this point is far from convincing. Cases of peripheral neuritis in children for which, after the most careful investigation, no cause could be found, have come under the notice of the writer, and such cases might be attributed to the poison of poliomyelitis, but the justification for such a conclusion is not great.

The type has been included here, but the writer fully recognizes that at the present time there is neither sufficient clinical nor pathological evidence for its full acceptance of the condition as a manifestation of poliomyelitis.

The Meningitic Form.—It is not uncommon for cases of acute poliomyelitis to exhibit symptoms of meningeal irritation. The child complains of headache, is drowsy and irritable, and there may be rigidity of the neck. There are other cases which exhibit more marked signs of meningitis than the above. The child may be completely comatose, with rigidity or retraction of the neck, and convulsions may be present. Such symptoms may be the only sign of acute polioencephalitis, and none of the ordinary paralytic phenomena may be present or develop. These cases might come under the heading of "serous meningitis," and are easily mis-

taken for cases of tuberculous meningitis; for on lumbar puncture the cerebro-spinal fluid flows out under increased pressure, has an excess of albumin, and an increase in the number of lymphocytic cells.

These cases may be rapidly fatal; on the other hand, a rapid and complete recovery may take place, and Wickman quotes a case in which complete recovery took place in twelve days.

It is important to distinguish these cases from those of tuberculous meningitis, and this can usually be done by examination of the cerebro-spinal fluid, for it contains fewer lymphocytes, has less albumin, and no tubercle bacilli.

The Abortive Form.—It is necessary to recognize an "abortive form" of the disease, in which there may be fever, headache, pain in the limbs, and a general weakness, but no paralysis. Such symptoms may be accompanied by sore throat and redness of the fauces, or in some cases diarrhoea may be present.

It is, of course, impossible to recognize such cases as due to the poison of poliomyelitis, unless they occur in association with typical instances of the disease; for there is at the present time no serum reaction or blood-test by which they can be recognized.

Such cases would undoubtedly be regarded as cases of indolent.

Herpes zoster occurs sometimes, though rarely in association with acute poliomyelitis. Wickman only had one case in the Stockholm epidemic of 1893.

Head has pointed out the close similarity of the lesion in the posterior root ganglion which produces herpes to that of poliomyelitis. Garrod called attention to the frequent occurrence of herpes zoster at the same time as poliomyelitis was present in Maryport in 1910.

PATHOLOGY—Macroscopic Examination.—During the acute stages of the disease the examination of the surface of the brain and spinal cord of a case of acute poliomyelitis, but little abnormal may be noticed; the membranes may be but little affected, and the surface of the brain and spinal cord may appear normal. On the other hand, the surface of the brain and the membranes may appear congested.

On section through the affected region of the brain, foci of intense congestion and redness are apparent. These may be confined to a single area, or may be widely distributed. A similar appearance may affect the basal ganglia, the pons, or medulla, and may pick out a single nucleus.

In the spinal cord the redness and congestion of the grey matter is the most striking appearance; or if the disease has been of somewhat longer standing, softening may have taken place, so that the grey matter of the anterior horns is almost diffused. These changes may be limited to the region of one horn, or may be widely scattered about the cord.

In the later stages of the disease a sclerotic condition of the cortex may be present, or a condition of porencephaly in which the surface of the brain is sclerosed and a cavity filled with fluid lies between the surface of the cortex and the lateral ventricle.

In the spinal cord, on section, one side may appear smaller than the other, and this is especially noticeable in the grey matter. The difference in size of the cord on the two sides is most striking in the antero-lateral tracts, the posterior column being usually about the same size.

On *Microscopic Examination*, the most characteristic change is a perivascular

exudation around the sheath of the vessels. There is marked hyperemia, and infiltration of the tissue both by leucocytes and blood-cells; the latter are always present in the severe cases.

The exact nature of the cells which surround the vessels is a matter on which there is considerable difference of opinion. The large majority may be regarded as small lymphocytes; plasma cells and large mononuclear cells are present only in small numbers. The cellular infiltration is, according to some observers, attributed to a great proliferation of the neuroglia, and a large number of the cells in the perivascular lymphatics are regarded as glial cells.



FIG. 83.—SECTION OF THE SPINAL CORD FROM A CASE OF ACUTE POLYMYELITIS OF GRAY MATTER'S DEGENERATION, SHOWING ENGORGEMENT AND PERIVASCULAR LYMPHOCELLULAR INFILTRATION, CHIEFLY THE VESSELS OF THE GRAY MATTER OF THE ANTERIOR HORN, BUT AFFECTING ALSO THE WHITE MATTER AND, TO A SLIGHT EXTENT, THE MENINGES.

The changes in the nerve cells are slight, except in the immediate region of the lesion, and it is striking to see a cell with normal staining of the nucleus, nucleolus, and the chromophilic substance surrounded by lymphocytic cells.

Degenerate cells are, of course, present, and all degrees of changes can be found, from a normal cell to one which is completely destroyed.

The changes in the spinal cord may well be taken as representative of the changes which occur in other portions of the nervous system, and for the convenience of description they will be described in three stages—

The first stage of acute inflammation.

The second stage of subsidence of the inflammation, leaving areas of softening.

The third stage of cicatrization.

If a section of the spinal cord be examined during the first few days after the onset of the disease, the perivascular exudation will be found to be present, especially around the vessel of the anterior median fissure, and about its branches as these pass into the grey matter. The pia mater on the ventral surface of the cord may also be found to contain an excess of cellular element. Although the anterior median vessel is as a rule most severely affected, yet the inflammation is not limited to this vessel, and the vessels of the pia arachnoid, as they pass into the spinal cord, may show the same change.

The grey matter of the anterior horns may in some cases be packed with blood-cells and lymphocytes, so that no nerve cells can be seen; in other cases small areas of the grey matter may be destroyed; whilst in others the nerve cells can be well seen, with a few haemorrhages and cells lying in close relation. Although this condition is not absolutely limited to the region of the anterior horns, yet the whole stress of the disease falls upon that area of the cord, and it is rare for any considerable destruction to be met with posterior to the cells of Clarke's column (Fig. 83).

In the second stage degenerative changes take place in the spinal cord consequent on the vascular lesion. These changes are very well shown by staining the cord by the Marchi method, which brings into prominence all the degenerate tissue by staining it black. The grey matter of the anterior horns is seen to be crowded with black granules, which represent the broken-down myelin; the black granules often surround the vessels. The anterior roots, both in their intramedullary and extramedullary course, show marked degeneration, and the degeneration can be traced into the peripheral nerves (Fig. 84).

The antero-lateral tracts of the spinal cord also show a large number of degenerate fibres, and the fibres can be traced up the spinal cord. The direct cerebellar tract may also show some degenerate fibres, but only in those cases in which Clarke's column has been affected. The pyramidal tract usually appears normal, but a few degenerate fibres may be present.

The destruction of the nerve elements, both in the grey matter and in the white matter, can be shown also by other methods of staining.

In the third stage shrinking of the cord takes place, owing to the contraction



FIG. 84.—SECTION OF THE SPINAL CORD FROM A CASE OF POLIOMYELITIS OF THREE MONTHS' STANDING, SHOWING SOFTENING AND DESTRUCTION OF THE WEDGE OF THE GREY MATTER OF THE ANTERIOR HORNS.

The section has been stained by the Marchi method, and the fatty products of degeneration are stained black. The child had complete faccid paralysis of the lower extremities.

of the new-formed fibrous tissue. The products of degeneration become absorbed and the Marchi method is no longer suitable for showing the change. There is in some parts of the spinal cord a complete absence of all cells in the grey matter, or one or two normal cells may be seen and a few atrophied ones. The fine mesh-work of the anterior horns is replaced by connective tissue (Fig. 85).

If the cord be stained by the Weigert-Pal method, the diminution of the number of medullated fibres in the antero-lateral tracts is well seen.

The posterior portion of the cord appears perfectly normal, as do also the posterior roots.

With regard to the muscles, the condition found varies with the stage of the disease at which they are examined. In the later stages of the disease bundles of perfectly normal muscle fibres are seen in close juxtaposition to bundles of completely degenerated or bundles of very atrophied fibres. In some muscles all the

fibres are replaced by fibrous tissue, the muscle consisting only of fat and fibrous tissue. In the earlier stage of the disease fatty degeneration can be seen in the fibres of the muscles affected.



FIG. 85.—SECTION OF THE SPINAL CORD OF A CHILD WHO, TWO YEARS BEFORE DEATH, HAD SUFFERED FROM POLIOENCEPHALITIS, LEAVING BEHIND HIM FLACCID PARALYSIS OF ONE LEG.

The grey matter of the cord on the affected side is greatly atrophied, and contains but few nerve cells. Stained by Weigert-Pal method.

DIAGNOSIS.—The diagnosis of a case of poliomyelitis in its later stage is as a rule a simple matter. The flaccid paralysis, the wasting of the muscles, the coldness and blueness of the extremity, the loss of reflexes, with the preservation of normal sensation, is a clinical picture not easily confused with that of other diseases.

In the earlier stages of the disease the diagnosis is by no means so simple.

During the acute stage the severe pain often masks the paralysis, and the condition is mistaken for acute rheumatism. The absence of any swelling around the joints and the freedom of movements in the joints should put one on the guard against such an error. The pain in the limbs bears a striking resemblance to the condition seen in cases of scurvy, and when the condition occurs in children during the first two years of life it is often most difficult to decide as to the diagnosis on first inspection. The onset of the disease may be attended by convulsions, and give rise to the fear of meningitis. Convulsions may be indicative of the meningeal or encephalic type of the disease, but may occur in cases in which the disease is limited to the spinal cord. The examination of the cerebro-spinal fluid is of the greatest importance in the differential diagnosis of this condition from meningitis.

A birth injury to the spine often gives rise to a condition which closely resembles that produced by a poliomyelitis. Such injury is usually attended by active contraction of certain muscles, with an inward rotation of the head of the affected

An acute epiphysitis of syphilitic origin may give rise to a flaccid paralysis of a limb, often of sudden onset. Such paralysis commonly occurs in infants from six weeks to three months old. The enlargement of the epiphysis can usually be felt, and a skiagram will show the irregular margin of the bone. The possibility of such a condition has only to be borne in mind, and the diagnosis is easily made.

A psoas abscess may give rise to symptoms which suggest a poliomyelitis—pain, loss of power in one leg, and loss of knee-jerks. The history of an acute onset makes the similarity of the clinical picture still more striking, and if the abscess is small and cannot be felt, the diagnosis is extremely difficult. A few days' careful observation will, however, generally make the diagnosis clear.

In those cases in which there is affection of the bulb, the weakness of the palate and the feebleness of respiration, with absent knee-jerks, suggest the possibility of a diphtheritic paralysis. The fact that it is the diaphragm which is usually affected in diphtheritic paralysis, whilst the intercostals are usually affected in poliomyelitis, is a point that should be borne in mind.

In the cerebral form of the disease the diagnosis is difficult, and it is largely by the process of exclusion that the diagnosis is arrived at rather than by a positive finding.

The acute onset of cerebral symptoms in a child suggest the possibility of meningitis. Meningitis should be excluded by a cytological and bacteriological examination of the cerebro-spinal fluid. The possibility of a vascular condition also suggests itself—either a hæmorrhage, an embolus, or thrombosis of arteries or sinuses, and a cause for these should be sought. The possibility of an abscess must also be borne in mind. When a child who has previously been quite healthy is taken acutely ill with cerebral symptoms which give rise to a local paralysis, the possibility of a polio-encephalitis must be considered. If these symptoms are widespread and tend rapidly to clear up, it is a point in favour of a polio-encephalitis.

In those cases in which the stroke of the disease has fallen on the cerebellum, and ataxia is a marked feature, the possibility of a cerebellar tumour and abscess must be borne in mind. The stationary and retrogressive nature of the symptoms is strongly in favour of a polio-encephalitis; but it must be remembered that tuberculous tumours in children not infrequently become quiescent and the symptoms tend to clear up. In those cases in which the ataxia persists the differential diagnosis from a Friedreich's ataxia requires consideration.

Shortly, it may be said that the acute onset of cerebral symptoms during the summer months in a child in whom meningitis can be excluded, strongly suggests that the condition is due to a polio-encephalitis. When, however, such symptoms occur in association with a flaccid paralysis in one or more limbs of the body, or about the same time another member of the same household is similarly affected, the diagnosis may be made with certainty.

PROGNOSIS.—The prognosis with regard to life in this disease depends upon the seat and extent of the lesion. If the bulb is affected, death, either immediate or in consequence of broncho-pneumonia, is most liable to occur. On the other hand, the disease itself is not very fatal to life. In the epidemics which have been observed carefully the percentage mortality has been about 11 or 12 per cent. In one epidemic the mortality amounted to 18 per cent., and in another to 6 per cent.

With regard to complete recovery, the prognosis is bad. Some few cases do undoubtedly recover completely, but the percentage is small, probably about

15 per cent. Many are left permanently crippled, with weakness of one or both legs and arms, with hemiplegia, with mental defect, squint, loss of sight, and permanent ataxia.

Great care in giving a prognosis in any individual case should be exercised, for under suitable conditions it is often striking how much improvement will take place in a case which otherwise seems to be almost hopeless.

The greatest amount of improvement usually takes place in the first six months after the onset of the disease, but further improvement may take place for another two years. After that period very little improvement is likely to occur in any case which has been carefully looked after.

TREATMENT.—A case of acute poliomyelitis should be isolated, and the contacts given a quarantine of fourteen days. The period for which the patient remains infective is doubtful; but a period of at least three weeks' isolation should be insisted on, and he should be free from all nasal discharge. A solution of 0.2 per cent. of potassium permanganate or of chlorine water should be used for the cleansing of the nasal and oral cavities of both patient and contacts.

The acute stage of the disease calls for but little active treatment. So far as is known, when once the infection has taken place nothing can arrest the course of the disease; on the other hand, it seems certain that any exertion or over-exertion is liable to greatly accentuate the disease. Rest is therefore the first essential, and the child should be placed in whichever position is most restful, the legs being carefully supported. A water-bed will often give great relief to the pains. Acetyl salicylic acid (aspirin) or salicin in 3 to 5 grain doses will often help to allay the pain. Hot applications to the spine may be used, but in some of the severest cases the pain is only relieved by morphia.

Hexamethylenamine should be given in 5 to 10 grain doses every four hours, for it has been shown experimentally that this drug passes into the cerebro-spinal fluid, and delays, if it does not always inhibit, the experimental infection of animals. It can, with perfect safety be given in the above doses to quite young children.

Whether it is advisable to give those in contact with a case of poliomyelitis hexamethylenamine in order to diminish the risk of infection is a point on which definite evidence is not yet forthcoming.

The subdural injection of epinephrin has been shown by Clark to have a marked effect in limiting the spread of the disease in animals, and might well be used in severe and ascending forms of poliomyelitis. Experimentally 1.5 c.c. of a 1 in 1000 solution of epinephrin was used.

When the disease is of wide extent, and the muscles of respiration and deglutition are affected, great care must be exercised in feeding the child, and if there is any evidence of failure of the glottis to close whilst the child is being fed, it is advisable to feed the child with a tube passed into the stomach.

After the acute stage, which usually lasts from five to ten days, has passed, recovery begins to take place. The important indications with regard to treatment are the prevention of contractions and deformities, the maintenance of the temperature of the limbs, and the stimulation of the muscles, so as to keep them in good condition whilst recovery of the damaged neurons is taking place.

Contraction of muscles which produce deformity is best prevented by massage, passive and resistive movements, and by the application of a light splint, which will keep the limb in a normal position whilst the child is in bed, and also serve to support the weak limb when up. A light "celluloid" splint carefully moulded

to the leg is most suitable, and should be worn both during the day and night. These splints were first made by Calot, of Berck-sur-Mer, and were introduced into this country by Guérain. They used them extensively in the treatment of tuberculous disease of bones and joints. In some cases deformity is almost certain to arise whatever precautions are taken, such deformity being due to the unopposed contraction of certain muscles. The application of electricity in its various forms is of considerable value in selected cases. It is, however, important to use that form of current to which the paralyzed muscles respond. The application of a faradic current to paralyzed muscles which do not respond to this current is in many cases not only a useless proceeding, but actually harmful, for the healthy muscles in the immediate neighbourhood actively contract and unduly stretch the paralyzed muscles.

The question as to the length of time for which the treatment by massage and electricity should be carried out is one which can only be decided in each individual case. Improvement may take place for a period of two years after the onset of the disease. If during this time treatment has been carefully carried out, it is improbable that any further improvement will occur. On the other hand, a case which has not been carefully treated during the first two years will often improve considerably when massage and electricity is begun.

Warmth is most essential to the good recovery of the limbs affected, and the greatest care should be exercised in keeping the limbs warm. This is in part done by loose woollen stockings and overalls, by friction of the skin, and by the application of heat, a hot-water bottle, or an electrotherm.

The late stage of poliomyelitis demands a variety of treatment dependent on the nature of the case.

Mechanical Support.—A considerable number of cases are greatly helped by some mechanical support, and a well-adjusted splint or instrument gives a child a wide scope of movement. In the earlier stages of the disease a well-fitted celluloid splint is probably the most convenient form, for it is light, it can be worn both night and day, and it keeps the affected muscles in a relaxed position, and thereby tends to hasten recovery and prevent deformity (Fig. 86).



FIG. 86.—CHILD WEARING CELLULOID SPLINTS IN WALKING APPARATUS.

The child had flaccid paralysis of both legs below the hips due to poliomyelitis.

In the later stages of the disease a metal splint with a knee-lock will for many cases be more convenient, for most celluloid splints fix the knee in the extended position.

Division of Tendons.—The shortening or lengthening of tendons is often a most useful measure in conjunction with other lines of treatment.

Transplantation of Tendons is a method which again is useful in selected cases.

Reaction of a Joint, so as to give a fixed instead of a flail joint, is of considerable value in selected cases.

Nerve Reaction and Reunion.—The principle of this operation depends upon the observation that when the proximal end of a degenerated nerve is connected with the distal end of a nerve in connection with a healthy nerve centre, the peripheral portion of the nerve regenerates, and hence impulses are able to pass from the centre to the muscles. This successful application of such an operation depends on several factors:

First, on the possibility of obtaining a healthy nerve in proximity to the degenerated nerve; secondly, on the paralyzed muscle retaining muscle fibres capable of regeneration when normally innervated; and, thirdly, that the partial section of the healthy nerve will give rise to no loss of power of serious importance.

The number of cases in which such treatment is applicable is small, and the results which have been obtained in a series of cases are by no means encouraging.

The operation should be performed before the sixth month after the onset of the disease, but no improvement is to be expected in the condition of the muscles for at least six months after the operation.

Whatever line of treatment is adopted, it must be borne in mind that warmth, massage, and active and passive movements, are essential to the maintenance of the nutrition of the limbs, and these methods of treatment should be kept up for at least two years after the onset of the disease.

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INTRACRANIAL TUMOUR.

INTRODUCTION.—Intracranial tumours in children give rise to the same cardinal symptoms commonly met with in the adult—viz., headache, vomiting, and optic neuritis, with a gradual increasing weakness of various parts of the body dependent on the position of the tumour.

Although such a statement is generally true, yet the features are often modified by factors peculiar to children.

Headache may not be a symptom of which the child complains, since, partly owing to the deeply situated position of the tumour, and partly to the yielding of the skull and separation of the sutures, so great pressure is exercised on the dura mater. When the tumour is situated in the pons, the child often makes no complaint of headache. Vomiting is by no means a constant symptom, and in some

cases the disease will run its whole course without vomiting. The occurrence of vomiting without nausea is a sign to which no great importance should be attached.

Optic neuritis is almost a constant feature when the tumour is situated in the cerebellum, but when the tumour is situated in the pons, optic neuritis is often late in developing, and the writer has watched a case of glioma of the pons for six months, and no neuritis developed up to the time of death. With tumours in the basal ganglia, optic neuritis is usually present at some stage of the disease.

Situation of the Tumour.—Tumours of the cerebral hemisphere (excluding the basal ganglia) are rare in children. Out of forty-six cases examined post mortem at the Children's Hospital, Great Ormond Street, in only four cases were the tumours situated in the cerebral hemispheres. Two were situated in the frontal region, and came under the care of the ophthalmic surgeon for proptosis of the eye, one was situated in the Rolandic region, and one invaded the corpus callosum.

No instance of an occipital or temporo-sphenoidal tumour occurred other than multiple tuberculous nodules which are not included in the present series of cases. In ten cases the tumours were situated in or near the basal ganglia, the corpus quadrigemina and optic thalamus being a common situation.

In ten cases the tumour was situated in the pons.

In twenty cases the tumour was situated either within one of the lobes of the cerebellum or pressed upon and displaced the cerebellum. In the remaining two cases the tumour was situated in one in the medulla, and in the other there was a diffuse sarcoma all over the base of the brain and cerebellum.

NATURE OF THE TUMOUR.—The nature of the tumour is variable. Almost one-third of the tumours are tuberculous in nature; the remainder show the structure of a new growth, some being glioma, others glio-sarcoma, others show a considerable amount of fibrous tissue.

Of tumours situated in the pons nearly two-thirds are gliomatous, the remainder being tuberculous; whilst of tumours situated in the cerebellum nearly two-thirds are tuberculous, whilst the remainder are new growths.

Age.—Tumours in children below the age of twelve months are rare. The frontal tumour above mentioned, which produced proptosis of the eye, occurred in a child six months old, and a tumour of the basal ganglia occurred in a child of four months. This latter gave rise to hydrocephalus, and the case was diagnosed as such during life, the local signs—viz., the hemiplegia—which the child exhibited being thought to be due to the hydrocephalus. Five of the forty-six cases above mentioned were under the age of two years. Intracranial tumour is therefore a comparatively rare disease in children under two years of age.

SYMPTOMATOLOGY.—The localizing symptoms due to intracranial tumours will be considered under the following headings:

1. Tumours of the cerebral hemisphere.
2. Tumours of the basal ganglia, crus, optic thalamus, corpus quadrigemina.
3. Pontine tumours.
4. Medullary tumours.
5. Cerebellar tumours.

Tumours of the Cerebral Hemisphere.—*Tumours of the Frontal Lobe.*—It has already been stated that tumours in this region are rare, and little need be said

with regard to their localization. Frontal tumours manifest themselves by proptosis in the side of the lesion, with failure of sight, and probably loss of smell on the same side.

Tumours of the Rolandic Region.—Tumours in the region of the precentral gyrus give rise to hemiplegia of gradual onset. The weakness may start in the face, arm, or leg, according to the position of the tumour, and may be accompanied by aphasia when situated on the left side in a right-handed child. The weakness may be accompanied by fits limited to the part affected, or the convulsion may be general.

Subsequent to a fit, the weakness is often increased. Fits may occur whether the tumour is situated in the cortex or in the substance of the cerebral hemisphere.



FIG. 87.—PHOTOGRAPH OF A TUMOUR INVOLVING THE BASE OF THE BRAIN OF A CHILD IN WHOM THE INITIAL SYMPTOMS, FAILURE OF VISION, OCCURRED THREE MONTHS BEFORE THE DEVELOPMENT OF ANY OTHER SIGN OF THE DISEASE. THEN THE USUAL SYMPTOMS OF INTRACRANIAL PRESSURE DEVELOPED, WITH LOSS OF DEEP REFLEXES.

The child died seven months after the initial symptoms. The pituitary body was unaffected.

If the lesion extends behind the fissure of Rolando, hemianæsthesia may be produced, and a deeply-situated lesion in the region of the posterior part of the internal capsule will produce hemiplegia, hemianæsthesia, and hemianopsia.

The usual physical signs of such a lesion will be present—viz., increased deep reflexes, ankle clonus, extensor plantar response, and absent abdominal reflex on the side opposite the lesion.

Tumours of the Pituitary are said to occur in children, and give rise to the same characteristic symptoms—viz., bitemporal hemianopsia, failure of sight, and optic atrophy.

In association with affection of the pituitary there may be an abnormal development of fat such as occurs with other affections of this gland, and give rise to Fröhlich's syndrome—viz., adiposity, a delayed development of the genital organs, an absence of hair, and a backward mental condition (see Diseases of the Ductless

Glands, p. 571). A radiograph of the skull will show the distension of the sella turcica, and furnish valuable diagnostic evidence.

Tumours of the Pinea Gland occur in childhood. Of fifty-nine cases collected by Bailey and Jelliffe, fourteen occurred before the thirtieth year of life. Such tumours may give rise to no symptoms, being found accidentally on post-mortem examination. The symptom most frequently described in association with tumours of the pineal gland is a constant tendency to fall asleep. The child can be roused, but rapidly again falls into a condition of natural sleep. This symptom has been attributed to the hydrocephalus rather than to the involvement of the pineal body.

A tumour in this situation may by pressure on the corpora quadrigemina give rise to ocular paralysis, especially to defective upward movements of the eyes. Glycosuria, adiposity, early sexual maturity, and cachexia, have all been described in association with pineal tumours (see Diseases of the Ductless Glands, p. 571).

Tumours of the Temporo-Sphenoidal and occipital lobes are so rare that the symptoms which they produce will not be considered, and can easily be deduced from a consideration of the functions of the parts involved.

Tumours of the Basal Ganglia, Optic Thalamus, and Corpora Quadrigemina, form an important group. One of the most characteristic features of this group is ocular paralysis. This may manifest itself by an external strabismus from weakness of the internal rectus due to involvement of the nucleus or fibres of the third nerve. The third nerve on one side may be completely paralyzed, or there may be defective upward and downward movement of both eyes, whilst the lateral movement remains good. Accompanying this defect there may be more or less ptosis. The pupils may be dilated and fail to react to light and accommodation, or they may fail to react to light and react on accommodation (Argyll-Robertson pupil).

In the early stages the symptoms may be limited to the ocular symptoms, but as the tumour grows other symptoms become manifest from pressure on neighbouring structures.

The involvement of the fibres of the optic radiation in the region of the external genicular body gives rise to hemianopia or to blindness. As the tumour extends deeper, the red nucleus receiving the fibres from the superior cerebellar peduncle of the opposite side may become involved, and give rise to weakness and a rhythmic tremor of the limbs on the side opposite to the lesion (see diagram, p. 786).

As the tumour increases, pressure is exercised on the pyramidal tract; hemiplegia or diplegia is produced, with rigidity or flaccidity of the paralyzed side ;



FIG. 88.—PHOTOGRAPH OF A CHILD, SHOWING BILATERAL PTOSIS AND EXTERNAL STRABISMUS DUE TO A LESION IN THE REGION OF THE CORPORA QUADRIGEMINA, INVOLVING THE NUCLEUS OF THE THIRD NERVE.

The position of the tumour is seen in Fig. 90.

and the iter between the third and fourth ventricles becomes blocked, and hydrocephalus develops.

The tendency to react excessively to stimuli both unpleasant and pleasant, a symptom which in adults has been described in association with lesions which

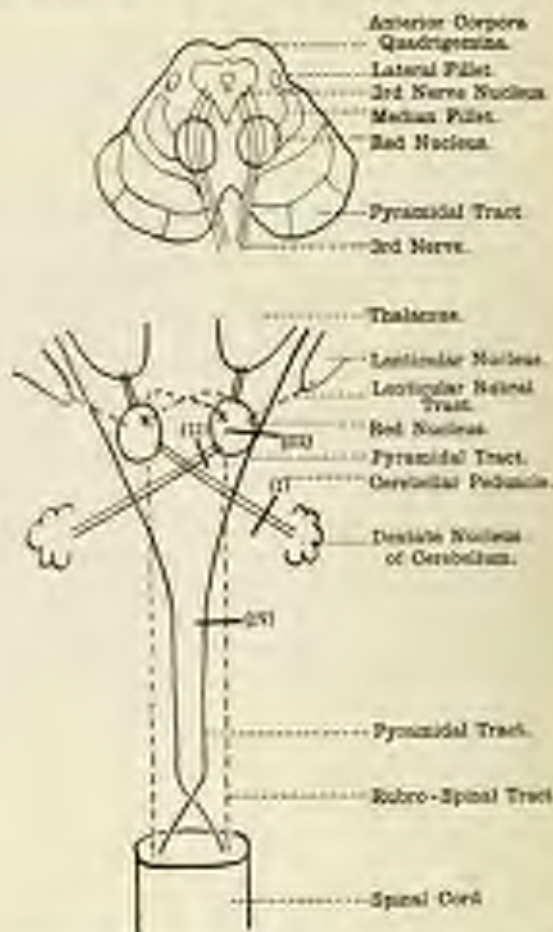


FIG. 88.—DIAGRAM TO SHOW THE CONNECTION OF THE RED NUCLEUS WITH THE CEREBRUM AND WITH THE RUBRO-SPINAL TRACT.

A lesion at (I) gives rise to a tremor homolateral to the lesion; at (II) to a tremor contralateral to the lesion; at (III) to a rhythmic tremor contralateral to the lesion; and at (IV) to a tremor homolateral to the lesion.

interrupted the cortico-thalamic paths, cannot be demonstrated in young children (see *Syndromes Thalamiques*, p. 525).

The course of disease in the case of a tumour situated in this region is often prolonged, lasting many months, the final stage of the disease being that of rigidity of the body and limbs in the extended position.

Tumours of the Pons are more common in children than in adults. The earlier manifestations of such a lesion are squint, owing to weakness of the external rectus,

a hemiparesis of one side and a facial paralysis of the opposite side—i.e., a "crossed paralysis." The paralysis of the face is of the peripheral type due to involvement of the lower motor neuron, and the child has difficulty in closing the eye and in raising the frontalis muscle. In some cases in which the tumour is situated far forward, the weakness of the face may be on the same side as the hemiplegia, but in that case the facial weakness has the character of a supranuclear lesion—viz., the frontalis and orbicularis palpebrarum are relatively little affected.

Both the motor and sensory portions of the fifth nerve are liable to be involved. The weakness of the motor portion is demonstrated by the weakness of the temporal and masseter muscles on the affected side, and by the deviation of the jaw to the paralyzed side on attempted opening of the mouth. The affection of the sensory portion of the fifth is shown by anaesthesia and analgesia over the side of the



FIG. 90.—VERTICAL SECTION THROUGH BRAIN, SHOWING TUMOR IN REGION OF THE CORPORA QUADRIGEMINA.

(Compare with Fig. 92, tumour of the pons.)

face corresponding to the distribution of the three divisions of the fifth nerve, and by the absence of the corneal reflex. The diminution or absence of the corneal reflex is an early sign, and can often be demonstrated before any analgesia is detected.

Tumours of the pons are often bilateral, and may give rise to the signs of cerebral diplegia, with great rigidity and opisthotonos, these symptoms being due either to the direct involvement of the pyramidal fibres in the pons, or to the hydrocephalus which, in some cases, follows the blocking of the iter. It is unusual for anaesthesia of the trunk to be present, in spite of the fact that the sensory fibres must be pressed upon by a tumour of the pons of any considerable size. Ataxia may occur in tumours of the pons, owing to the involvement of the cerebellar peduncles.

Tumours of the pons almost always give rise to cranial nerve palsies, but

exceptional cases are met with in which, in spite of the growth surrounding and pressing on the cranial nerves, no paralysis is produced.

Tumours of the Medulla are so closely related to cerebellar and pontine tumours that there is no clear line of separation. Most cases of pontine tumour, and many cases of cerebellar tumours, in their later stages develop bulbar symptoms, either owing to the growth invading the medulla or to pressure on the medulla.

The symptoms presented by tumours of the medulla are—(1) Loss of lateral movements of the eyes, owing to the involvement of the sixth nerve nucleus, which serves as a lower centre for conjugate deviation; (2) facial paralysis; (3) nerve

deafness; (4) paralysis of the palate and constrictors of the pharynx; (5) paralysis of the vocal cords, of the sternomastoid and upper portion of the trapezius muscle; (6) paralysis and wasting of the muscles of the tongue; (7) forced movements of rotation. This movement is probably dependent on the involvement of cerebellar and vestibular fibres in the region of Deitens' nucleus and the inferior cerebellar peduncles.

Tumours of the medulla may also give rise to symptoms of hemiplegia and diplegia with loss of sensation of the face on one side, and loss of sensation on the other side of the body; but such symptoms are rare and difficult to demonstrate in any young child. Again, the



FIG. 62.—PHOTOGRAPH OF A BOY, SHOWING SCISSOR PRODUCTION BY WEAKNESS OF THE RIGHT EXTENSOR FLEXOR, AND RIGHT FACIAL PARALYSIS DUE TO A TUMOUR OF THE PONS.

The position of the tumour is seen in Fig. 59.

affection of the nuclei in the medulla is liable to cause death before the pressure on to motor and sensory paths is sufficient to cause any disturbance of function.

Tumours of the Cerebellum form the most important group of intracranial tumours in children, not only because of their greater relative frequency than in adults (45 per cent. being situated in the cerebellum), but because they are more accessible to surgical treatment, and also sometimes spontaneously cease to grow or exhibit symptoms of their presence.

Tumours may be situated in the lateral or in the mid lobe of the cerebellum, or may be situated outside the organ, and give rise to cerebellar symptoms owing to pressure on one lobe of that organ. These form two important groups—viz. intracerebellar and extracerebellar tumours and the symptoms due to them will be described separately.



FIG. 92.—VERTICAL SECTION THROUGH BRAIN, SHOWING NEW GROWTH
(STARTING THE PONS).

The tumor produced at first spasm and paresthesia of the right side of the face and left side of the body. Optic neuritis was never present. (Compare Fig. 90).



FIG. 93.—LONGITUDINAL SECTION OF BRAIN, SHOWING A TUMOROUS MASS SPREADING IN THE
MEDULLA, AND INVOLVING THE PONS AND THE CEREBELLUM.

Forced movements of rotation in the direction of an outgoing corticospinal tract were in the early stages of the disease the most prominent symptom.

Intra-cerebellar Tumours.—Tumours of the lateral lobe of the cerebellum give rise to the following symptoms:

1. Ataxia on the side of the lesion.
2. Loss of tone (hypotonia) on the side of the lesion.
3. Attitude of head and trunk.
4. Rotation and forced movements.
5. Weakness on side of lesion.
6. Ocular paralysis.
7. Alteration in shape of head.

These symptoms shortly stated will now be considered in detail:

Ataxia.—This is the most striking symptom of affection of the cerebellum. It is usually more marked in the lower than in the upper extremities, and is manifested by the wide base on which the patient stands, the reeling gait, and the difficulty which the patient has in co-ordinating the leg on the affected side. The inco-ordination of the leg can be easily demonstrated in a patient who is unable to walk by asking the patient to place the heel of the one leg on to the knee of the opposite side.

In the muscles of the trunk and neck the ataxia manifests itself by the difficulty the patient has in maintaining the sitting posture. The inco-ordination of the upper extremities is brought out by asking the child to feed itself, to touch an object or its own nose. In some cases these movements can be relatively well performed, and yet the patient is unable to carry out in rapid succession movements of antagonistic muscles. This symptom has been termed by Babinski "*dyadimochinesis*." The usual method of testing the patient for this sign is to ask him to rotate the hands rapidly, using the muscles of pronation and supination. On the affected side the movement will be slow and inco-ordinate, on the normal side rapid and co-ordinate. Numerous methods can be adopted for demonstration of the ataxia. Cerebellar ataxia tends to affect the legs rather than the arms, the trunk and proximal muscles rather than those distally situated.

Hypotonia—loss of tone—is a symptom which is by no means constant in cases of cerebellar tumours, owing to the fact that the general increase of intracranial pressure brings other factors into play. Hypotonia is demonstrated by the loss of tone in the muscles, and the fact that the limbs can be placed into abnormal positions owing to the flaccid condition of the muscles. Hypotonia manifests itself on the side of the lesion.

Attitude.—After experimental ablation of one lobe of the cerebellum in animals, a peculiar attitude of the head is observed. The ear on the side of the lesion is approximated to the shoulder of the same side, and the side of the face corresponding to the cerebellar lesion is turned upwards, and the chin is diverted to the same side. The trunk is curved with the concavity to the side of the lesion.

In children suffering from cerebellar lesions this peculiar attitude is sometimes observed: The head is held to one side, so that the ear approximates to the shoulder of the same side, and the face is turned to the opposite side.

The position of the head does not, however, always correspond to the side in which the lesion is situated, and in a series of cases investigated with this object in view it was found that in three the position of the head corresponded to the side of the tumour, whilst in three it did not correspond. The curvature of the

spine is but rarely seen in children. It should be noted, also, that the cerebellar attitude has been observed in cases in which no lesion has been found in the cerebellum—e.g., hydrocephalus.

Rotation and Forced Movements.—Closely related to attitude are two curious phenomena sometimes seen in cerebellar lesions—viz., rotation in the long axis of the body, and tendency to deviate to one side when walking. Experimentally, a lesion of the right lobe of the cerebellum gives rise to cirous movements in an anti-clockwise direction, and to rotation in the long axis as an out-going corkscrew.

The same phenomena will sometimes be seen in children lying in bed, in that they always tend to turn to one side, and if turned on to the other side often vomit and return to the same side. In walking the child always tends to fall to one side. Theoretically this should be on the side opposite to that on which the lesion is situated; in practice it is often found, however, that the child tends to fall to the side of the lesion.

In investigation of this symptom it is well to get the child into the kneeling position, and ask it to crawl. It will be noticed that the buttocks fall to one side or the other. Sometimes the slightest touch on one side will make the buttock fall to the opposite side, whereas a touch on the opposite side will have no effect. By the application of the in- or out-going corkscrew test the side of the lesion will be indicated.

Weakness.—Weakness on the side of the lesion is sometimes seen, but is rather a rare manifestation. It may in some cases account for the fact that the patient tends to fall to the side on which the tumour is situated.

Ocular Paralysis.—Various forms of ocular paralysis are seen in connection with cerebellar tumours. The most common is to find weakness of the movements of conjugate deviation to the side of the lesion, and it is to this side that nystagmus is most marked when there is not actual paralysis. Weakness of the external rectus on the side of the lesion is not infrequent.

A rare form of paralysis, and one seen usually after operation on the cerebellum, is what is known as *skew deviation*, the eye on the side of the lesion being displaced



FIG. 94.—PHOTOGRAPH OF A CHILD, ILLUSTRATING THE POSITION OF THE HEAD ASSUMED IN SOME CASES OF CEREBELLAR DISEASE.

The position seen in the photograph would indicate a lesion of the left lateral lobe of the cerebellum.

upwards and outwards, and the eye on the opposite side being displaced downwards and outwards.

Optic neuritis occurs early in the case of cerebellar tumours; it is commonly bilateral, and no special significance can be attached to a unilateral optic neuritis, nor to its intensity on one side as compared with the other.

Alteration in the shape of the head may occur in some cases where the tumour is of slow growth and the skull not thick. Actual prominence of the occipital region on one side may be of some diagnostic value, but the cases are few in which this symptom is present. A radiograph of the skull is seldom of value in the diagnosis of cases of cerebellar tumours.

Reflexes.—In uncomplicated cases of cerebellar tumours it may be stated that the deep and superficial reflexes are normal. The knee-jerks and ankle-jerks are present, and the plantar and abdominal reflexes give a normal response.

It is perfectly true that in some cases of cerebellar tumours the knee-jerks

become decreased or abolished; but it is the exception to find them so diminished or absent except in late cases. It is not infrequent in cerebellar disease to find the knee-jerks at one time brisk, and at another absent, and this alteration may occur within a few hours. The superficial reflexes are not altered in an uncomplicated case of cerebellar disease.

Various explanations of the loss of knee-jerks in cerebellar tumours may be given. It is supposed by some that the hypotonia of the muscles causes the loss of knee-jerks. The increased intracranial pressure is given as a cause. It is a well-known fact that any intracranial tumour may give rise to degeneration of the posterior roots and posterior columns of the spinal cord, and this



FIG. 95. — EXTRACEREBELLAR TUMOUR; SOMETIMES KNOWN AS "PONTINE ANGLE" OR "ACUSTIC NERVE TUMOUR."

Note the tumour is situated outside the tissue of the cerebellum, but presses upon the lateral lobe of the cerebellum, and compresses the pons.

is at least one possible explanation why the knee-jerks should be absent in some cases of cerebellar tumours. It does not explain, however, the variability of the knee-jerks.

Extracerebellar Tumours. owing to their situation, give rise to a set of symptoms which is very characteristic. Briefly it may be said that they cause cerebellar signs on the one side of the body, and cerebral signs on the opposite side of the body.

The cerebellar signs are the same as those just described above—viz., ataxia, hypotonia, and nystagmus, on the side of the lesion.

Owing to the situation of the tumour in the pontine angle at the exit of the seventh and eighth nerves, these nerves become affected, and give rise to facial paralysis and nerve deafness on the side of the lesion. The sixth nerve is also often affected owing to pressure, and sometimes the fifth may be affected, giving rise to

anesthesia of the side of the face and weakness of the muscles supplied by the fifth nerve.

In addition to these signs there is weakness of the opposite side of the body, with increased deep reflexes, ankle clonus, extensor plantar response, and absent abdominal reflex, these signs indicating involvement of the pyramidal tract by pressure of the tumour on the side of the pons and medulla.

Certain subjective sensations with regard to rotation of self or objects are sometimes described by adults, but are never spoken of by children.

Tumours of the Mid-Lobe of the Cerebellum give rise to few, and sometimes to no, localizing symptoms. The general symptoms—headache, vomiting, and optic neuritis—are often intense; but there may be no ataxia, no ocular paralysis, and no altered reflexes. There is often a marked tendency to fall backwards, but this symptom cannot be considered as diagnostic of tumours of the mid-lobe, since it



FIG. 96.—CYSTIC TUMOUR OF THE MID-LOBE OF THE CEREBELLUM.

The cyst is filled with a yellow viscid fluid, and adhesion in connection with a glioma or glio-sarcoma. Note the compressed condition of the pons below the cyst.

occurs with tumours in other situations. When the tumour has grown sufficiently large, it may involve the dentate nucleus and cerebellum on one side, and thus give rise to symptoms pointing to affection of the lateral lobe.

Tumours in this situation more frequently, however, give rise to hydrocephalus, and it is often from the presence of the hydrocephalus that the symptoms arise rather than from the cerebellar lesion itself.

Pseudo-Tumours.—Certain cases have been described in which all the symptoms of tumours are present—viz., headache, vomiting, and optic neuritis—sometimes accompanied by localized weakness or ataxia, such as to suggest a localized tumour, and yet on operation or on post-mortem examination no tumour is found.

A case of this nature came under the writer's observation, and, from certain symptoms, was diagnosed as a tumour of the right lobe of the cerebellum. Operation was performed, but no tumour was found. The boy rather suddenly died, and on examination, although there was much flattening of the cerebellar con-

lution, indicating considerable pressure, yet on examination neither hydrocephalus nor signs of tumour could be found; nor on microscopical examination was there anything to suggest a general infiltration of the cerebral substance with glioma. Noone, however, says that this condition is due to an infiltrating glioma with very few cells; possibly the microscopical examination in this case was not extensive enough to find the glioneous tissue.

Small localized intracranial tumours may be found on post-mortem examination, which have given rise to no symptoms during life. Such tumours are commonly tuberculous, but may be of the nature of cholesteatomata, and situated at the base of the brain.

PATHOLOGY.—Almost 50 per cent. of the tumours which occur in children under twelve years of age are tuberculous. (These figures do not include cases in which tuberculous nodules are found on post-mortem examination, and which have given rise to no symptoms.) Of the remaining 50 per cent., the variety of growth is almost as great as it is in adults, and it is often most difficult to say to what class a growth belongs—a sarcoma, a *glia-sarcoma*, a glioma, or an endothelioma. Some of these growths are cystic, and contain a fluid or a jelly-like material. The nature of the growth varies to an extraordinary extent, according to the position from which the portion examined is taken. Some growths are infiltrating and invade the cerebral tissue; others are circumscribed and encapsulated.

DIAGNOSIS.—The diagnosis of an intracranial tumour in the later stages of the disease is not difficult; on the other hand, in the early stages it is often most difficult. The history of headache, attacks of vomiting, and gradual increase of symptoms, suggest the probability of an intracranial neoplasm; the presence of optic neuritis strengthens that probability. It is not in all cases, however, that these cardinal symptoms are present. Headache and vomiting may be absent, and the gradual onset of localized weakness, stasis, or an ocular paralysis, may be the first indications of disease. Again, periodic attacks of uncontrollable vomiting may be the early manifestation, and these attacks suggest the possibility of "cyclic vomiting," and in such a case the most careful physical examination may reveal no sign of affection of the nervous system. The position of the headache is important: frontal headache may be due to a variety of causes, not dependent on actual intracranial disease; occipital headache is commonly connected with intracranial disease.

Meningitis will give all the cardinal signs of an intracranial tumour, and in some cases the symptoms may be of such a gradual onset as to suggest a growth rather than a lesion of inflammatory origin.

The diagnosis between meningitis and an intracranial tumour should be easily made by the examination of the cerebro-spinal fluid. It is well to bear in mind that fatal results have been recorded after lumbar puncture in the case of cerebellar tumours. In performing this puncture on a child in whom a cerebellar tumour is suspected, it is well to keep the child in the lateral position, to stop the flow of fluid as soon as enough fluid has been obtained for diagnostic purposes, and to have a hypodermic syringe with strychnine ready in case the patient shows any sign of respiratory failure. Personally, in a long series of lumbar punctures, I have only once seen any bad effect from this operation, and that was in the case of a cerebellar cyst. There was for a short time cyanosis and respiratory distress, which was rapidly relieved by strychnine.

Cerebral and cerebellar abscess often give rise to considerable difficulty in

differential diagnosis from tumour. The presence of some focus of infection may suggest the possibility of abscess, and the rapidity of onset may also favour such a view. In some cases the symptoms of cerebral abscess are latent, and it may be weeks, or even months, after an ear infection that the symptoms of intracranial disease arise. The character of the temperature chart will not aid the diagnosis, except in so far as a subnormal temperature would be in favour of an abscess.

Sinus thrombosis will give rise to all the signs of intracranial tumour, but as a rule the symptoms are of much more rapid onset, and the condition of coma into which the child passes is very marked, as compared with the drowsy condition which may be present in cases of cerebral tumours.

"*Spas-epoplexie*" is another condition which may give rise to some difficulty in diagnosis. In this condition symptoms pointing to intracranial disease may arise weeks, or even months, after an injury to the head, which at the time may have appeared quite trivial. Such symptoms are due to vascular disease, but their gradual onset in association with the other signs of intracranial disease makes the diagnosis from intracranial tumour one of great difficulty.

The diagnosis of the situation of an intracranial tumour depends upon the correct observation and interpretation of the various physical signs which have been described in dealing with the symptomatology. It may be well to repeat, however, certain general facts. Tumours of the cerebral cortex and of the cerebral hemisphere are of the greatest rarity in children, and a diagnosis of such should only be made after most careful examination and consideration of the facts observed. Tumours of the frontal lobes are practically unknown in children. Tumours of the basal ganglia, pons, and cerebellum, are of common occurrence, and usually give rise to signs from which their situations can be rightly indicated.

The nature of the tumour can only be guessed. Occasionally lumbar puncture may show the presence of tubercle bacilli in the cerebro-spinal fluid when the tumour is situated in close relation to the surfaces, coming in contact with the cerebro-spinal fluid, or under the same conditions abnormal cells may be present which are derived from a new growth. Apart from these rare circumstances, the difficult diagnosis between tubercle and new growths can only be made from a careful consideration of the general features of the case and the family history.

PROGNOSIS.—The prognosis in any case of intracranial tumour is of necessity bad. Tuberculous tumours sometimes cease to grow, and the acute symptoms subside; but the nervous system has generally been damaged, the eyesight may be defective, hydrocephalus may be present and give rise to periodic attacks of headache and vomiting. Sudden and unexpected death often occurs in such cases.

The prognosis as regards the duration of life is most variable. Some cases, especially of infiltrative glioma of the pons, will run a long course of six months or more after obvious signs of the tumour first present themselves, and during the whole of the time the child may remain fairly bright and well, but with an ever-increasing weakness of the limbs. It is an extraordinary fact that many of these children, though suffering from a squint of a paralytic nature, seldom complain of diplopia. This is not due to defective vision in the one eye, for if the other eye be closed they recognise and follow objects correctly. It would seem that the false image is most easily suppressed in childhood.

Tumours of the cerebellum rarely run such a long course as tumours of the pons or basal ganglia.

TREATMENT.—Removal of the tumour by operation is, owing to the situation of the tumour, rarely possible; and even in those cases in which it would seem possible, the results obtained are not such as to encourage anyone to take a hopeful view as to the ultimate success of the operation. Tumours of the cerebellum are those most accessible, and in any given case it is well to consider the advisability of an exploratory operation in order to find out the nature of the tumour. If the tumour be cystic, a very considerable amount of relief may be obtained by the evacuation of the cyst, although it may be impossible to remove the whole tumour. In the case of a tuberculous tumour which is sharply circumscribed, it may be possible to remove it, but not infrequently a tuberculous meningitis develops and is rapidly fatal. The treatment of tuberculous tumour by injection of tuberculin is a line of treatment which, so far as I know, has not been attended by success.



FIG. 37.—DIFFUSE SARCOMA OF THE CENTRAL NERVOUS SYSTEM: PHOTOGRAPH OF THE POSTERIOR ASPECT OF THE CEREBELLUM, SHOWING THE GROWTH SPREAD OVER THE SURFACE OF THE VERMIS.

The growth was not limited to this situation, but involved both brain and spinal cord.

The relief of optic neuritis in children by decompression is an operation which has less application than in the adult. Many cases, especially those in which the tumour is in the pons, run their whole course without the development of optic neuritis. Again, tumours situated at the base of the brain often give rise to loss of vision owing to the pressure on the optic tract.

Cases of cerebellar disease in which optic neuritis is intense are those most likely to be favourably influenced by decompression, and are also those in which it may be possible to remove the tumour.

The relief of headache is often best obtained by the administration of small doses of calomel. Children with intracranial tumours seem to suffer much less than adults from severe headache. Vomiting can often be controlled by simple dieting, though severe and persistent vomiting is a comparatively rare symptom.

Phenazone is undoubtedly a useful remedy both for the headache and for vomiting, and should be given in $1\frac{1}{2}$ to 3 grain doses. Eucodal and chloral are often of great service. The administration of iodide and mercury has been recom-

mended under the assumption that the growth may sometimes be syphilitic. There is no doubt that both these drugs, either alone or in combination, may give rise to relief of symptoms and temporary improvement. A localized gumma in the brain has been described.

Diffuse Sarcomatosis of the Nervous System.—This is a somewhat rare affection of the nervous system in childhood, and one in which the diagnosis is not easily made.

SYMPTOMS.—If the disease attacks the brain in the first instance, the symptoms are those which occur in association with a cerebral tumour or with a chronic form of meningitis. If the disease first attacks the spinal cord, spinal caries, spinal meningitis, and spinal tumours, are first thought of. At a somewhat later period the signs of pressure on the cord may become more definite and localized, and the presence of a localized growth may be diagnosed. If submitted to operation, a diffuse growth of the pia arachnoid will be found which it is impossible to remove.

Since the growth may affect almost any part of the nervous system, it is easily understood that there may be great variations in the characters of the symptoms. Pain is frequently felt, and may be one of the most distressing symptoms. Localized paralysis, except of the cranial nerves, is rare. General wasting of the limbs takes place, and in the final stages the child passes into a marasmic condition.

MORBED ANATOMY.—On opening the cranial cavity and spinal canal, a diffuse growth is seen covering the spinal cord and surface of the cerebellum or cerebrum, in distribution like a meningitis. On section through the growth and subjacent nervous tissue, the growth is seen to be limited to the membranes on the surface, and rarely invades the substance of the brain or spinal cord. The growth behaves in every respect like a meningitis of infective origin, and often affects the posterior root ganglion. The growths are of a sarcomatous nature—small round cells in a rather loose meshwork (Fig. 57).

DIAGNOSIS.—Diagnosis is only possible by obtaining the cells of the growth in the cerebro-spinal fluid withdrawn by lumbar puncture, and the absence of cells will not exclude the presence of a growth.

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VASCULAR LESIONS OF THE CENTRAL NERVOUS SYSTEM.

INTRODUCTION.—Under this heading are included such conditions as are due to hemorrhage from, thrombosis and embolism of, the cerebral vessels.

Hæmorrhage on the surface or into the substance of the brain is commonly due to trauma occurring at birth, or more rarely at some later period of life. It may be delayed for some days or weeks after the injury—as called “Spät-apoplexie” (late apoplexy).

Hæmorrhage either into or on the surface of the brain may, however, occur

as the result of hooping-cough, purpura, broncho-pneumonia, blood-diseases, and general infective conditions, such as endocarditis and *B. coli* cystitis. It may occur as the result of sinus thrombosis (see p. 805).

Hæmorrhage due to birth injury is in part dealt with under Infantile Hemiplegia, but it frequently gives rise to general rather than localized signs. There is little doubt that many cases of intracranial hæmorrhage occurring at birth clear up completely.

The hæmorrhages which occur with hooping-cough may be meningeal, or may be of a punctiform character, scattered throughout the substance of the brain.



FIG. 98.—BRAIN OF INFANT, SHOWING LARGE HÆMORRHAGE INTO THE SUBSTANCE AND ON THE SURFACE OF THE LEFT TEMPORO-SPHENOIDAL LOBE, DUE TO INJURY BY FORCEPS AT THE TIME OF BIRTH.

They may give rise to localized symptoms, or the child may gradually pass from a drowsy condition into one of coma without localized weakness.

A condition which it is important to recognize is late apoplexy, in which symptoms do not develop for some time after the injury. The following case, recorded by Miller, illustrates the leading features: A child was struck on the head with a stone. She complained of headache and was a little drowsy, but attended school on two days in the following week. A little ptosis of the eye

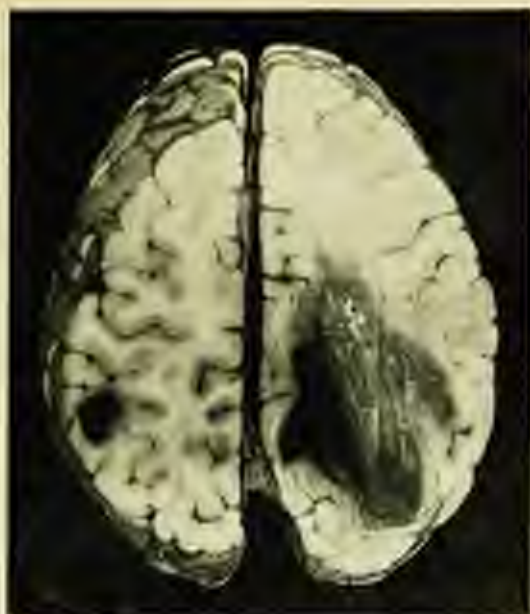


FIG. 99.—CORONAL HEMORRHAGIC EXTENSION IN A CASE OF DUCTECTOMY OF THE URINARY TRACT WITH BACILLARY COLIC.

The child, who had been treated for the urinary trouble three months, and was apparently cured, suddenly had a series of fits, developed hemiplegia, and died in less than twenty-four hours. (Dr. Garrod's case.)



FIG. 100.—LATE APoplexy: BRAIN OF A CHILD AGED TWELVE, SHOWING AN AREA OF SOFTENING ON THE INNER SIDE OF THE RIGHT TEMPORO-SPHENOIDAL LOBE, HEMORRHAGE FROM WHICH CAUSED DEATH OF THE CHILD SIX WEEKS AFTER THE ONSET OF THE ILLNESS.

(From photograph kindly lent by Dr. A. H. Miller, "Lancet".)

was noticed on the fourth day after the accident, and by the seventeenth day there was complete paralysis of the right third nerve. At the end of another fourteen days the child was much better, though she still had the ocular paralysis. She continued in this condition till six weeks after the injury, when she suddenly cried out, became unconscious, and died on the following day without regaining consciousness. A cavity containing blood-clot was found on the under-surface of the right temporo-sphenoidal lobe, and it was from this softened area that the hemorrhage spread over the surface of the brain.

The cause of each "late apoplexy" is probably due to rupture of a vessel taking place in an area of necrotic and softened brain tissue.



FIG. 101.—HEMORRHAGE INTO THE MEDULLA AND FOURTH VENTRICLE CAUSED BY A FALL.

The case presented symptoms suggesting a meningocerebral aneurysm. On lumbar puncture, blood-stained fluid only was obtained, and this was thought to be due to puncture of a vessel. Note the hemorrhage in the ventricle and in the substance of the medulla.

SYMPTOMATOLOGY.—The symptoms produced by hemorrhage are commonly of sudden onset, but it has been shown that this is not invariably the case, and that the symptoms may not arise for some days, or even weeks, after the injury.

The symptoms produced by hemorrhage vary according to the situation of the lesion, and in some cases may closely simulate those of other diseases—*e.g.*, meningitis.

No detailed description of the symptoms produced by hemorrhage will be given, for they vary according to the situation of the lesion.

Intraventricular Hemorrhage is a comparatively rare condition in childhood. It occurs in birth injuries, and is not invariably fatal; for, in a case in which there was evidence of such a hemorrhage, the cerebro-spinal fluid obtained by lumbar

puncture, which was at first deeply coloured with blood, eventually became quite clear, and the child made a good recovery. Intraventricular hæmorrhage may be produced by rupture of a vessel in puncture of the lateral ventricle for the relief of intraventricular pressure. Hæmorrhages may also occur in cases of tuberculous meningitis in which cerebral softening has occurred, and a vessel subsequently given way.

Thrombosis and Embolism of the Cerebral Vessels.—Thrombosis and embolism of the cerebral vessels are much less common in children than in adults. These conditions may follow any of the infective diseases—diphtheria, typhoid, scarlet fever, pneumonia or measles, or rheumatic endocarditis.

It is often difficult to distinguish between a primary thrombosis of a vessel and one which is secondary to an embolus. The sudden onset of symptoms is



FIG. 102.—HEMORRHAGE INTO LATERAL, THIRD, AND FOURTH VENTRICLES, AND ALONG THE VENTRAL SURFACE OF THE CEREBELLUM, CAUSED BY PUNCTURE OF THE LATERAL VENTRICLE UNDERTAKEN FOR THE RELIEF OF PRESSURE.

embolism and more gradual symptoms in thrombosis may in some cases serve to distinguish the two conditions, but in other cases no such distinction can be drawn. Convulsions occur so frequently in children that no diagnostic value can be attached to their occurrence. The vessel commonly affected is undoubtedly the middle cerebral artery, giving rise to hemiplegia, homianæsthesia, homianopsia, and in some cases to aphasia.

An interesting group of cases in which hemiplegia is associated with complete blindness of the eye of the opposite side has been described by Guthrie and Flatters. Such cases are due to a blocking of the ophthalmic artery, together with the middle cerebral vessel. Such a lesion can only occur when the ophthalmic artery has an abnormal origin from the middle cerebral artery. It is possible for other vessels to become thrombosed, such as the cerebellar artery, and give rise to acute cerebellar

symptoms. Such cases are very rare in comparison to thrombosis of the cerebral vessels.

After the immediate effects of the lesion have passed off, there remains a more or less permanent weakness, which usually manifests itself by hemiplegia. Such a condition may be followed by fits which have in their earlier stages the character of a Jacksonian epilepsy. Later the attacks become more general, with complete loss of consciousness, and the child may become a chronic epileptic.



FIG. 163.—Brain from a child, aged Ten Months, who suffered from Congenital Morbus Cerebrus; Cerebral Thrombosis, possibly due to an Embolus, softened, and gave rise to Right Hemiplegia.

PATHOLOGY.—Little need be said with regard to the pathology of this condition. Softening occurs in the distribution of the vessels affected. The products of necrosis gradually become absorbed, and a cystic condition remains. The process is essentially the same as that which occurs in the adult.

DIAGNOSIS.—The diagnosis of the cause of intracranial symptoms in childhood is sometimes most difficult. The sudden onset of symptoms will point to a vascular lesion, the rapid onset to an inflammatory lesion, and a gradual onset to a neoplasm or granuloma. It has been shown that a vascular lesion may have a gradual onset, and it is true that both with an inflammatory lesion and a neoplasm the onset of symptoms may be sudden. In the case of a child who is more or less unconscious, who presents the signs of a hemiplegia, and has had convulsions,

vascular lesions, polio-encephalo-myelitis, meningitis, at once suggest themselves as possible causes. Meningitis can in most cases be excluded by the examination of the cerebro-spinal fluid, but there remain the two great groups in which the greatest difficulty arises. Polio-encephalitis should not, in my opinion, be diagnosed unless the onset is acute, attended by fever and constitutional symptoms, and occurs when the child is otherwise in good health. The occurrence of the onset of the illness during the summer months, and especially at a place where other cases of polio-myelitis are taking place, strengthens the probability of a polio-encephalo-myelitis. The cerebral symptoms which occur in the course of, or during convalescence from, acute illness are commonly due to thrombosis of vessels or sinuses.

PROGNOSIS.—In the acute stages no certain prognosis can be given either with regard to life or complete recovery. After the acute stage is over and consciousness is recovered, something may be judged from the degree of the paralysis. In some few cases recovery is complete; in most cases a permanent weakness, more or less complete, remains.

TREATMENT.—During the acute stage but little can be done. The fits can and should be controlled by the administration of bromide and chloral and if necessary by chloroform. After the acute stage has passed, local treatment to the limb or limbs paralyzed should be undertaken. The placing of the limbs in suitable splints to prevent deformity is an important part of the treatment, together with massage and exercises.

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SINUS THROMBOSIS.

INTRODUCTION.—Thrombosis may take place in any of the cerebral sinuses, but most frequently occurs in superior longitudinal and lateral sinuses. The inferior longitudinal sinus, the cavernous sinus, and the veins of Galen, are rarely thrombosed.

Ætiology.—The commonest cause of thrombosis of the longitudinal sinus is an illness which gives rise to great wasting and feebleness, such as enteritis, prolonged suppuration, endocarditis, cachexia, and hooping-cough. Thrombosis of the lateral sinus commonly occurs in association with middle-ear and mastoid diseases. Thrombosis of the other sinuses is generally associated with conditions of sepsis and profound asthenia.

Some cases follow shortly after operation undertaken for conditions which cannot be regarded as septic. Thus, in three out of twenty cases of cerebral sinus thrombosis occurring at the Children's Hospital, one occurred in a child of four years, operated on for the radical cure of hernia; one in a child aged nine weeks, after operation for pyloric stenosis; and one in a child aged two and a half years, after removal of a foot of the large intestine. In all these cases thrombosis of the longitudinal sinus occurred.

Of the remaining seventeen cases, fourteen were cases in which some infective condition was present—mastoid disease, appendicitis, empyema, tubercle, and pyopericarditis. The three other cases occurred in children aged three weeks, eight months, and fourteen months, respectively; all were in a wasted condition and presented symptoms which suggested meningitis. In twelve of the cases the superior or inferior longitudinal sinus was thrombosed, in seven the lateral sinus, and in one the cavernous. The thrombosis of the veins of Galen occurred in association with thrombosis of other sinuses. It is clear that the important factor in determining thrombosis is the presence of some infective condition.

SYMPTOMATOLOGY.—The symptoms to which thrombosis of the cerebral sinuses gives rise are similar to those of meningitis. The child, who may be at first noisy and irritable, gradually becomes lethargic, and finally comatose. The temperature commonly runs a most irregular course, often rising to 105° to 106° F., then dropping to the normal level, and again rising to a high degree. Marked head retraction is sometimes seen, but in many cases this symptom is absent. Convulsions frequently occur, and are followed by paralysis and rigidity of the limbs. Squint and optic neuritis are often present. Edema of the face and eyelids, and protrusion of the eyes, are symptoms dependent upon the situation of the thrombosis. If the cavernous sinus is thrombosed, there is always proptosis, edema of the eyelids and root of the nose, and paralysis of the ocular muscles.

The examination of the cerebro-spinal fluid is of considerable diagnostic value. In cases of uncomplicated thrombosis the fluid is clear, flows out under pressure, contains no organism, a faint trace of albumin, and the dextrose reaction is present. Blood may be present, but in most cases it is not present.

In cases in which the lateral sinus is thrombosed the symptoms are more definite. There is swelling in the region of the mastoid, and in the neck the hard and tender thrombosed jugular vessel can be felt. Before actual thrombosis has occurred, the tenderness of the mastoid, the irregular temperature, and the fulness in the neck, would suggest that inflammation had taken place around the vein, and methods should be adopted to prevent the spread of infection and formation of a thrombosis. Even when a thrombus has formed, the ligature of the jugular below the thrombosis will prevent a general infection.

PATHOLOGY.—The appearance of the surface of the brain in cases of thrombosis of the superior longitudinal sinus is most striking. The large veins on the surface of the cortex stand out as hard dark combs. In some cases the surface of both hemispheres may be covered with blood, but more frequently some one part of the surface—either the frontal or occipital region—is covered by a hæmorrhage. If the inner surface of the dura mater be examined, the large and distended veins as they enter the longitudinal sinus are seen, and the clot in these is found continuous with the clot in the sinus. The clot in the sinus is sometimes so adherent to the wall that it is with difficulty detached; in very acute cases, however, it can be detached without any difficulty. The clot in almost every instance can be shown to be septic.

In those cases in which there has been thrombosis of the veins of Galen there is considerable softening of the cerebral substance; whilst on section of the brain numerous dark points, due to thrombosis of the smaller vessels and hæmorrhage, are seen. In some cases hæmorrhage has taken place within the ventricles.

DIAGNOSIS.—The diagnosis of thrombosis of the cerebral sinuses is often a difficult matter. In the case of the lateral and cavernous sinuses the difficulty is not so great as with the other sinuses, for they give rise to a definite set of symptoms; in regard to the former, early diagnosis is of the greatest importance, for it is by the removal of the septic matter around the lateral sinus that extension of the thrombosis can be prevented.

In cases of thrombosis of the longitudinal sinus occurring with *meningitis*, the symptoms are so little marked that the diagnosis is often not made during life, the



FIG. 164.—THROMBOSIS OF SUPERIOR LONGITUDINAL SINUS.

The veins leading into the sinus are thrombosed, and there is extravasation of blood over the surface of the cortex.

infant passing from a condition of collapse into one of uncorroborated, and the thrombosis of the sinus is only found at post-mortem examination.

In most cases the real difficulty arises in the diagnosis of sinus thrombosis from meningitis and intracranial abscess. Lumbar puncture and the examination of the cerebro-spinal fluid will in many cases clear up the diagnosis, but there will remain a group of cases in which there is little or no alteration in the character of the cerebro-spinal fluid, and such cases may be either due to sinus thrombosis or abscess. Optic neuritis, unilateral or bilateral convulsions or paralysis, irregular temperature, may be present in both conditions, and it will only be from the most careful consideration of the mode and character of onset and the possible source of infection that a correct diagnosis will be arrived at.

Thrombosis of the cerebral sinuses is a more common condition in early childhood than intracranial abscess.

PROGNOSIS.—Except in so far as the lateral sinus is concerned, it may be said that the disease is invariably fatal. It is possible that some cases of local thrombosis may clear up, but convincing evidence of such is difficult to obtain. Cases of lateral sinus thrombosis, if only diagnosed early and suitably treated, should do well.

TREATMENT.—The operative treatment of thrombosis of the lateral sinus has been mentioned, but as regards detail of operation reference must be made to a surgical work. When once thrombosis of the sinuses has taken place, it is doubtful if its progress can be prevented. In those cases, however, in which there is evidence of venous engorgement and failure of the heart (conditions under which thrombosis is known to occur), it is probable that it can be prevented by suitable measures. For the sake of example, in a case of severe whooping-cough with dilated right heart in which there is cyanosis and cerebral symptoms, such as spasmodic jerking of the face or limbs, venesection should be performed or leeches applied, and at the same time stimulants should be given.

The cases which occur after operation are most difficult to avoid. Most have occurred after imperative operations undertaken for some septic condition, but some few have occurred in cases in which there was no such imperative demand.

CEREBRAL DIPLEGIA—CONGENITAL SPASTIC PARAPLEGIA— LITTLE'S DISEASE.

Under this title are included a great group of cases in which there is evidence of a bilateral affection of the cerebral hemispheres. The term is essentially a clinical one, for it brings into a single group cases which have an entirely distinct pathology.

There are some cases due to the arrest of cerebral development during fetal life (prenatal); there are others due to changes taking place at birth, either from too rapid or too prolonged delivery (natal); there are others in which the vital resistance of the neurons is defective, either from intrinsic or extrinsic causes (post-natal); and in some of these cases there is progressive cerebral degeneration.

ÆTIOLOGY.—The causes which give rise to these defects cannot be well defined.

In the first group, the health of the mother during pregnancy would certainly seem to be an important factor, but it cannot account for the majority of cases of defective development; and on careful inquiry in such cases, the mother's health during pregnancy cannot be shown to have been in any way impaired. Syphilis, consanguinity, inheritance, undoubtedly play a part.

In the second group, injury to the brain at birth by forceps or by pressure is generally thought to be the great factor in the production of cerebral diplegia, cerebral hæmorrhage and thrombosis of the cortical veins being the direct cause. How far this factor plays a part in the production of the disease is questionable, for the cases are not investigated pathologically till long after such an occurrence, and all trace of hæmorrhage may have disappeared. It is a notable fact that, in many cases in which the injury is attributed to damage at birth, the birth has been easy, delivery rapid, and the child small.

The third group of cases, in which the disease develops after birth, are due to a

degenerative process dependent on some toxic condition or defective vital endurance. As an instance of the latter, the hereditary and family spastic paraplegia may be given; of the former, those cases of diplegia which develop after meningitis or as the result of congenital syphilis.

SYNTHROMATOLOGY.—For the purpose of clinical description, cases of cerebral diplegia will be divided into groups, according to the most striking features which they present. It must not, however, be assumed that all cases can be allocated to any one group, for numerous instances occur which present the symptoms of two or more groups.

The following groups will be described :

1. The spastic group, with generalized rigidity.
2. The "athetoid" group.
3. The choreic group ("post-hemiplegic chorea").
4. "Perverse movements" group.
5. The "stereic" group.

1. The Spastic Group.—This class of case is characterized by generalized rigidity. This feature may be noticed soon after birth, but it comes into greatest prominence at the time of life when the child first attempts to walk. When the child is placed on its feet, the legs become crossed, owing to the active contraction of the adductor muscles of the leg, and on attempted progression the pelvis is tilted laterally and the knees pressed close together. The muscular power in such cases is often surprisingly good. The knees may be in a flexed or extended position, and *pes equinus* or *pes cavus* may be present. The arms may likewise be rigid, extended, and strongly pronated, but as a rule the arms are less affected than the legs. The rigidity may affect the trunk and neck, and the condition known as a "spastic face" may be present.

It is usual to find in this group that the knee-jerks are exaggerated, ankle clonus is present, and the plantar response is extensor in type; but in some cases the rigidity of the legs makes it most difficult to obtain either the knee or ankle jerks.

Considering that there is good evidence of affection of the pyramidal tract, it would have been expected that the abdominal reflexes should be absent. Such



FIG. 935.—CEREBRAL DIPLEGIA. CHILD ATTEMPTING TO WALK, AND SHOWING THE TYPICAL CROSSED-LEGGED PROGRESSION.

(From a photograph kindly lent by Dr. James Collie.)

however, is not by any means always the case, and many children with spastic diplegia show active abdominal reflexes.

2. The "Athetoid" Group.—This group is characterized by slow, worm-like movements which occur either with or without voluntary movements. The "athetoid" movement commonly affects the arms rather than the legs, but may be present in both. The same characters are seen in the movements of the face and tongue, and give rise to grimacing and an alteration in articulation. The "athetoid" movements are not usually present at birth, but come on as the child begins to develop, and often increase in the later years of childhood.



FIG. 166.—CEREBRAL DIPLEGIA: PHOTOGRAPH OF A CHILD SUFFERING FROM A PROGRESSIVE FORM OF CEREBRAL DIPLEGIA, GIVING RISE TO RIGID EXTENSION OF THE LIMBS AND RETRACTION OF THE HEAD.

(From a photograph kindly lent by Dr. James Collier.)

In a pure case of athetosis there may be no weakness of muscles, no spasticity, and no alteration of reflexes, such as suggest affection of the pyramidal tracts. The knee-jerks are present, there is no ankle clonus, and the plantars are flexor in character. In many of these cases the intelligence is perfectly good, although the movements of the limbs and the defective articulation give rise to the impression of mental defect.

3. The Chorea Group.—The movements in this group are similar to those seen in rheumatic chorea, which were well described by Sturges as "exaggerated idiosyncrasms." In some cases these movements may be so violent that it is impossible to keep the child in an ordinary bed, and special arrangements have to be made to protect him from injury. In some cases there may be sharp, short contractions of certain muscles like a myoclonus. There

is as a rule a good deal of alteration in the mental condition. Convulsions are not uncommon, and the choreic movements have a tendency to increase from time to time. It is sometimes very difficult to separate cases presenting the choreic movement from those presenting the athetoid on the one hand, and the "perverse" movement on the other.

4. "Perverse Movement" Group.—Under this heading are included a group of movements described by Collier as "maladroitness." The movements are not typically athetoid or choreic. The hand, after attempting to grasp an object, remains fixed in that position, and is with difficulty relaxed. There is often a definite intention tremor which resembles that seen in disseminated sclerosis. This perverse movement may give rise to an awkward gait, but not one that is either choreic or athetoid.

There are certain symptoms which may occur in association with any of the groups above mentioned. *Mental defect* may be met with in any form, but is certainly more common in the spastic paraplegia and in the choreic group than in the others. Every degree of mental defect may be found, from complete idiosy to feeble-mindedness or moral perversion. *Speech defects* are common. There may be complete absence of, imperfect or delayed development of, speech. *Blindness*, either cortical or due to optic atrophy, may be present, and this may be accompanied by nystagmus. Various forms of squint may be met with, an internal strabismus being the most common. *Dribbling of saliva* from the mouth and difficulty in swallowing solids may be present. Liquids are generally easily swallowed. The attitude which the limbs and trunk assume depends upon the muscles affected. Various attitudes have been described. The two most common



FIG. 107.—BRAIN OF DEFECTIVE CHILD, SHOWING MICROCELL AND AN ABNORMAL ARRANGEMENT OF THE CONVOLUTIONS.

It will be noted that the microgyria is especially marked in the occipital lobes. In such cases the children are usually blind.

are—first, that in which the limbs and trunk are flexed; and, second, that in which there is rigid extension of the limbs and retraction of the head.

5. The "Atonic" Group.—There is a type of cerebral diplegia associated with great loss of tone (hypotonia) of the muscles, so that the limbs can be placed in most abnormal positions in relation to the trunk—positions similar to those which have been described in *myotonia congenita*. This condition is commonly seen in association with marked mental defect and an atrophic condition of muscles.

Whether such a condition is due solely to a lack of development of the cerebrum, or is in reality a manifestation of lack of development of other portions of the nervous system and muscles, is uncertain. The symptoms closely resemble those seen in defects of muscular development, but in some of the cases, at any rate, the normal reaction of the muscles to electrical stimulation is such as to suggest that there is no gross defect in the muscles themselves, and the condition is due to a cerebral defect.

Pathology.—As the clinical manifestations of cerebral diplegia show enormous variations, so do the cerebral conditions which give rise to those symptoms. It is easy to show that meningeal and cerebral hemorrhages occur at birth (see Fig. 98).



FIG. 108.—CEREBRAL DYSPLASIA: SCALP OF A CHILD WITH CEREBRAL DYSPLASIA SHOWING A GENERAL ATROPHY OF THE CEREBRAL CORTEX.

The brain substance was usually firm and tough.



FIG. 109.—CEREBRAL DYSPLASIA: HORIZONTAL SECTION OF BRAIN SEEN IN FIG. 108, SHOWING THE ATROPHY OF THE CEREBRAL CORTEX AND COMPENSATORY DILATION OF THE VENTRICLES.

It is, however, most difficult to prove in the later cases that the pathological change is due to hemorrhage; in fact, the condition of the cerebral hemisphere in many cases renders this impossible.

The following are the conditions usually found:

1. Atrophic Sclerosis.—The condition most frequently found in cases of cerebral diplegia is a widespread atrophic sclerosis of the cortex. The convolutions are shrunken, and the sulci between them are wide and deep. This condition may affect the whole of the cerebral cortex, or be more or less limited to some one part. On section through the brain, the wasted condition of the convolutions is still more evident, and in stained sections the absence of medullated fibres is striking. The basal ganglia suffer from the same lack of development as may also the brain-stem and spinal cord. The pyramidal tracts are undeveloped or are only partly medullated.

2. Arrested Development of the cortex accounts for some cases of diplegia. Figs. 110 and 111 represent such a condition. It is unilateral in its distribution, but the symptoms which the child presented were bilateral. In this case the development of the cortex was arrested in the fifth to sixth month of foetal life.



FIG. 111.—CEREBRAL DIPLEGIA: LATERAL VIEW OF THE ABOVE HEMISPHERE, SHOWING THE PRIMITIVE CONDITION OF THE CONVOLUTIONS.



FIG. 110.—CEREBRAL DIPLEGIA: BRAIN OF AN INFANT AGED SEVEN MONTHS, SHOWING ARREST OF DEVELOPMENT OF THE LEFT HEMISPHERE ABOUT THE SIXTH MONTH OF FOETAL LIFE.

Fig. 112 represents a condition of defective development in which some parts of the cortex are normally developed, while others are entirely absent.

3. Occlusion of Cerebral Vessels in intra-uterine life is a cause of hemiplegia and diplegia.

Fig. 113 shows a condition of the hemisphere due to obliteration of the middle cerebral vessels in intra-uterine life.

4. The Various Forms of Meningitis which may give rise

to diplegia are dealt with under the heading *Meningitis*. Syphilis as a cause of diplegia is dealt with in the chapter on *Syphilis of the Nervous System*.

DIAGNOSIS.—The diagnosis of cerebral diplegia is not as a rule difficult. The spastic condition of the limbs and the crossed-legged progression give a clinical picture which is distinctive. There are, however, cases in which the symptoms



FIG. 112.—CEREBRAL DYSPLASIA: BRAIN OF INFANT, SHOWING GROSS FORM OF CEREBRAL DEFECT MET WITH IN THIS CONDITION.

Note the deficiency of the cerebral cortex, and the exposure of the basal ganglia, the lateral ventricles, and the choroid plexuses. The defect was bilateral, though not quite symmetrical.



FIG. 113.—INFANTILE HEMIPLEGIA: BRAIN OF AN INFANT, AGED FIVE MONTHS, WHO SUFFERED FROM RIGHT HEMIPLEGIA FROM THE TIME OF BIRTH.

The whole of the cortex supplied by the middle cerebral vessel was in a sponge-like condition. The middle cerebral vessel was represented by a hard cord, but it was impossible to say whether the condition had occurred during intra- or extra-uterine life.

are by no means so marked, and a slight spasticity of the legs, with increased knee-jerks, ankle clonus, and extensor response, may suggest a spinal rather than a cerebral lesion. The absence of pain, of anaesthesia, and the presence of increased arm-jerks and an active facial reflex, would point to a cerebral rather than a spinal lesion. Tumours of the pons may give rise to symptoms resembling those of diplegia; but the occurrence of paralysis of some one or more of the cranial nerves generally serves to give the correct diagnosis, apart from headache and vomiting, which usually accompany intracranial tumours.

The cases with choreic and perverse movements are, when first seen, difficult to distinguish from ordinary rheumatic chorea, and it is only by noting the persistence of the movements associated with the mental defect that a correct diagnosis can be made.

The movements of some cases resemble those seen in disseminated sclerosis in the adult; but since disseminated sclerosis is a disease almost unknown in child life, the differential diagnosis need not be considered.

PROGNOSIS.—There is one class of case in which the symptoms are slowly and steadily progressive, and in these nothing can be done to arrest the progress. There is another and larger class in which there is no tendency for the disease to progress, and in these cases much can be done to relieve the symptoms, so that improvement steadily takes place. The cases with athetosis and perverse movements seldom improve; they remain in exactly the same condition for years, and it is wonderful how much they can perform, in spite of their physical disabilities, so long as the mental condition is normal.

It is probable that many of the cases of cerebral diplegia in infants die either from inanition or from some intercurrent disease. The writer has observed infants, in whom during life there was no sign of visceral disease, steadily waste in spite of the greatest care with regard to diet and surroundings, and on post-mortem examination cerebral defect alone was found.

TREATMENT.—The treatment of a case of cerebral diplegia is important. Those cases which are most amenable to treatment are the slightly spastic cases. Massage, passive and active movements, hot baths, and extension, will do much to correct deformity and reduce the spasms.

After as much as possible has been accomplished by these methods, tenotomy, tendon lengthening, and stretching, should be resorted to; but it is important not to omit the other methods of treatment mentioned above after the tenotomies have been performed.

Section of the posterior roots has been advocated and tried in these cases by Förster. In selected cases the operation has given good results, but they need to be most carefully selected. The cases most suitable for this operation are those in which there is considerable spasticity, and in which the power of movement and mental condition are good. The roots which the operator usually selects to divide are the second, third, and fifth lumbar, and the second sacral, on both sides. It is, however, not an easy matter to separate off the roots, and none of the methods of identifying them are altogether reliable. The mortality as direct result of the operation is considerable.

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INFANTILE HEMIPLEGIA.

Infantile hemiplegia stands in close relation to many cases of cerebral diplegia, and the same factors are causative in both conditions. There is, however, one cause of cerebral diplegia which is not a cause of infantile hemiplegia, and that is progressive cerebral degeneration.

Under the term "infantile hemiplegia" may be included all cases of hemiplegia in infants due to a cerebral lesion. It is not intended, however, in this section to deal with those cases of hemiplegia in infants which are due to a vascular lesion occurring after the time of birth, nor with cases which may be due to a poli-encephalo-myelitis. These have been dealt with under the heading of Vascular Lesions and Poliomyelitis. Hemiplegia due to meningitis and to intracranial tumours will also be found under their respective sections. Having thus narrowed down the scope of this section, the causes which produce infantile hemiplegia may be shortly stated.

Ætiology.—Defects in the cerebral development of one hemisphere are a cause of infantile hemiplegia. Such defects may be an agenesis of the cortex, an arrest of development, a porencephaly. Instances of such have already been given under the section Cerebral Diplegia.

Natal and Prenatal Hemorrhages may be caused by prolonged labour, too rapid delivery, and pressure of forceps. These hemorrhages are commonly situated on the surface of the brain, but may involve the hemispheres, and in some cases the hemorrhages may be intraventricular.

Intra-uterine Thrombosis and Embolism of the cerebral vessels is a rare cause of infantile hemiplegia.

SYMPTOMATOLOGY.—The hemiplegias of the infant show the same clinical variations as the diplegias—viz., a spastic, an athetotic, a choreic, and "perverse movement" type. The spastic variety is the common one; the athetotic, choreic, and "perverse movement" types are rarely seen during the first years of life, and only become manifest as the pyramidal tracts become developed. In some cases the hemiplegia is of the flaccid variety in the earliest stage.

During infancy the paralysis manifests itself by the inability to move the paralyzed side, whilst the movements on the other side are free. As the child grows, it is usually late in learning to walk, and, when it does so, drags the leg and holds the arm in a flexed and adducted position to the side affected. In some cases the arm is thrown about in a disorderly manner during the attempt to walk.

The face may show some weakness, and there may be dribbling from the side of the mouth. The development of speech may be delayed, and remain in complete abeyance if the hemiplegia is on the right side. Mental defects are not infrequent. The limbs on the hemiplegic side may sometimes be smaller than those on the sound side.

The knee-jerks and ankle-jerks on the paralyzed side are usually increased, ankle clonus may be present, and the plantar will give an extensor response. The abdominal reflex is commonly present on the hemiplegic side, a condition contrary to that which might be expected.

In some cases, and more especially those in which athetotic movements are

present, the deep reflexes may be unaltered, and the plantar may give a flexor response. Such reflexes would indicate that the lesion producing the hemiplegia lies outside the pyramidal tract. Children with infantile hemiplegia are very liable to become epileptic, and in the first instance such attacks may be Jacksonian in type.

PATHOLOGY.—The pathology has already been discussed under the heading Diplegia.

DIAGNOSIS.—Little difficulty presents itself in the diagnosis of infantile hemiplegia after the first few weeks of life. A spinal hemorrhage in the cervical region may give rise to a hemiplegia, but in such a case the paralysis of the arms is not of a spastic type, and other symptoms, such as crossed analgesia and paralysis of the sympathetic on the side of the hemiplegia, giving rise to pupil and eye symptoms, may be present, and render the diagnosis clear.

PROGNOSIS.—The prognosis in most cases of infantile hemiplegia is unfavourable so far as complete recovery is concerned. Many cases, however, develop a considerable amount of power in the leg, but the arm commonly remains much paralyzed.

In those cases in which epilepsy develops the outlook is most unfavourable; the child gradually passes into a degenerate condition with mental impairment.

TREATMENT.—Massage and passive movements are of the greatest use in preventing the rigidity and promoting movement in the paralyzed limbs. Tenotomies may be of service in those cases in which contraction of muscles has taken place, but the judicious use of suitable splints will in many cases avoid the necessity for such operations. Section of the posterior roots has been advocated, and some successes have been recorded, but the writer has had no personal experience in cases of hemiplegia, and the operation could only be of service when there was considerable spasticity.

HYDROCEPHALUS.

Hydrocephalus may be due to an accumulation of fluid within the ventricles (internal hydrocephalus), or to fluid between the membranes and the brain (external hydrocephalus). External hydrocephalus is a rare condition, and only seen in conjunction with defective development of the brain.

ETIOLOGY.—The causes of internal hydrocephalus are numerous, and, though it is convenient for the purposes of description to regard hydrocephalus as a disease, it is in truth only a symptom of many diseases. Syphilis, congenital defects in development, infective conditions producing meningitis or ependymitis, and tumours, are the chief causative factors. Enlargement of the lateral ventricles takes place in various degenerative conditions of the brain. Such conditions should not be designated "hydrocephalus."

Congenital Hydrocephalus is due to an abnormal secretion of the cerebro-spinal fluid by the choroid plexuses, or to an intra-uterine meningitis or ependymitis. If the process has begun some time before birth, the resulting hydrocephalus is probably so great that delivery is impossible without destruction of the head. In other cases the head may be obviously hydrocephalic at birth; in others, again, it may appear normal during the first few days, weeks, or even months, of life, and then begin to enlarge.

Acquired Hydrocephalus may be due—

1. To an acute or chronic affection of the ependyma and the choroid plexus.
2. To mechanical causes which prevent the normal flow of the cerebro-spinal fluid from the ventricles into the subarachnoid space. These causes may be—Meningitis, especially when situated in the region of the fourth ventricle; tumors of the base of the brain, of the pineal gland and brain stem; and inflammatory conditions of the ependyma, which give rise to blocking of the passages between the various cisterns of the brain.

SYMPTOMS.—The enlargement of the head is the characteristic symptom of hydrocephalus. In infants and young children the sutures of the cranium separate, and the bone becomes absorbed, so that "windows" of membrane are formed in the middle of the cranial bones, and a condition of craniotabes is produced.



FIG. 114.—HYDROCEPHALUS AS THE RESULT OF MENINGOCOCCAL MENINGITIS.

Note the dilatation of the lateral and third ventricles. In this case the block occurred in the region of the fourth ventricle.

In many of these cases the child seems to suffer no headache, does not vomit, and has no paralysis; this absence of signs of intracranial pressure is due to the fact that the sutures easily separate. As the head increases in size, the forehead becomes more prominent, the eyes become turned downwards, and the sclerotic is exposed above the cornea. This downward displacement of the eye is sometimes so marked that the pupil is eventually covered by the lower lid, and in order to see the child has to pull down the lower lid. This is well shown in Fig. 115.

Percussion of the hydrocephalic head will often give a note having a tympanic quality.

The eye symptoms are numerous and varied. Optic atrophy may occur, and complete blindness. On the other hand, vision may be perfectly good. Optic

neritis rarely occurs. Nystagmus is frequently present, and may be lateral, vertical, or rotatory. Squint from ocular paralysis often is present.

Any of the other cranial nerves may be paralyzed either on one or both sides. The presence of, and the progressive nature of, such symptoms may give rise to the suggestion that the child is suffering from a cerebral tumour, when in reality only hydrocephalus is present. In addition to the paralysis of the cranial nerves, there may be symptoms due to involvement of the pyramidal and cerebellar tracts.

The child may exhibit a spastic condition of arms and legs, with increased knee-jerks, ankle clonus, and slow extensor response of the plantars; or the child may show a considerable amount of ataxia of the arms and legs, with a hypotonic condition of the limbs.

Chronic Cases.—In those cases in which there is chronic hydrocephalus, the most marked feature is usually periodic attacks of severe headache and vomiting. Such attacks may occur at long intervals, and may last for from twenty-four to forty-eight hours, during which time the patient is prostrate. In the intervals the patient may be in every way normal, or may show some mental defect and some slight paralytic signs. The cerebro-spinal fluid from a case of hydrocephalus may vary greatly, both in regard to its appearance and its cytological and chemical characters. The fluid may be perfectly normal even when removed in large amounts.

In other cases the fluid may appear of a yellow colour, and may clot spontaneously immediately after withdrawal. Such a fluid always contains a large amount of albumin, and has the characters of an inflammatory exudation.

In other cases the fluid may appear clear, but contain an excess of albumin, and on cytological examination may show an increased number of leucocytes, either lymphocytic or polymorphic in character.

In some cases a large amount of fluid can easily be obtained; in other cases only a few drachms will flow. When only a small amount is obtainable, it is suggestive that the hydrocephalus is due to a block of the foramina around the fourth ventricle, or to a block in the iter between the third and fourth ventricles, or to a block in the third ventricle itself. In those cases in which there is free communication between the ventricles and the other reservoirs of cerebro-spinal fluid a large amount of fluid is usually obtainable.



FIG. 115.—CHRONIC HYDROCEPHALUS. PHOTOGRAPH OF A CHILD WITH SO MUCH DOWNWARD DISPLACEMENT OF THE FOREHEAD THAT VISION WAS ONLY POSSIBLE BY PULLING DOWN THE LOWER LIP; THIS ACTION THE CHILD HAD LEARNED TO PERFORM.

(Photograph kindly supplied by Mr. Tompkin.)

PATHOLOGY.—Hydrocephalus is sometimes purely due to mechanical obstruction, sometimes to inflammatory conditions of the ependyma, sometimes to a perverted action in the secretion of the choroid plexus and the failure of absorption. A discussion on the relative importance of these various factors does not come within the scope of this book.

The condition found on post-mortem examination is a dilatation of the lateral ventricles with thinning of the cortex. In some cases, when the head has grown to an enormous size, the cortex may be thinned to less than one-eighth of an inch. The corpus callosum is so thin that it is represented only by a delicate membrane,



FIG. 116.—HYDROCEPHALUS STARTING THREE MONTHS AFTER BIRTH, WITH HYPERPLASIA OF THE CHOROID PLEXUSES.

The condition was associated with other defects in the development of the ventral vertebrae. The choroid plexus weighed 6.5 grammes; the normal plexus weighs 9.75 grammes.

which almost always ruptures on removal of the brain from the cranium (unless this is done under water), and the velum interpositum no longer exists. The third ventricle and the iter between the third and fourth ventricle may be dilated, and the fourth ventricle and the cerebellar ventricle may also be distended; but the distension of these depends upon the cause of the hydrocephalus.

The choroid plexuses are almost always firm, often matted together, sometimes cystic, and no longer have their normal soft, seaweed-like appearance when floated out in water. Sometimes they are greatly enlarged, it may be to six or seven times their normal size (Fig. 116).

The opacities are sometimes thickened and of yellow and granular appearance, but in cases of simple dilatation does not appear abnormal.

DIAGNOSIS.—The diagnosis of hydrocephalus is generally self-evident. The cause of the hydrocephalus is a matter much more difficult to determine. The examination of the cerebro-spinal fluid withdrawn by lumbar puncture will sometimes assist the diagnosis.

In some cases hydrocephalus may be present without any marked enlargement of the head, and in such cases the diagnosis must be based on the periodic attacks of headache and vomiting in association with physical signs dependent on the increase of pressure.

The apparently large head of the rickety child, the bossed head of a congenital syphilitic, the large head of a cretin, of an achondroplasia, or of a cranio-cleido-dysostosis, should not be confused with the globular head of a true hydrocephalus.

PROGNOSIS.—The general outlook for a case of hydrocephalus is bad; on the other hand, a few cases have done well under a variety of forms of treatment.

Much will depend on the cause of the hydrocephalus and the age at which it occurs.

TREATMENT.—Lumbar puncture is necessary for diagnostic purposes as to the cause of hydrocephalus, and it is in some cases the best and most rational method of treatment.

In some cases the fluid in the ventricles may be drawn off by lumbar puncture, and after a certain number of tapplings the fluid may no longer tend to accumulate. At first the fluid should be drained off every three days, later on once a week, and then at longer intervals. The quantity removed will depend partly on the condition of the child, and partly on the pressure under which the fluid exists in the cerebro-spinal system. The maximum amount that has been removed at one time under the writer's care is 20 ounces; but in most cases not more than $1\frac{1}{2}$ ounces should be removed on the first occasion, and only this amount if the child shows no sign of respiratory distress.

The inunction of mercury may be of service. Iodide of potassium should be given in doses of 5 to 10 grains every four hours. Many children stand this quite well; others will not tolerate the drug by the mouth, and in such cases iodopin (H x-xx.) should be given hypodermically once a day. The injection of substances into the cerebro-spinal canal may be tried; some of them apparently do no harm, among which may be mentioned normal horse serum and 0.5 per cent. solution of procainol. The injection of iodine is dangerous.

It has been stated that the fluid removed from a pleural effusion and injected under the skin has led to the absorption of the effusion. Such a method might be tried in those cases of hydrocephalus which could be shown to be due to inflammatory conditions.

Operations of various kinds have been performed in cases of hydrocephalus, and several methods have been suggested for draining the intraventricular fluid into the subarachnoid space. Many of these operations have been fatal, and are unrecorded; some few have been successful, and are recorded. Stiles has ligatured the carotids in some cases.

Some of these operations have been successful so far as the preservation of life is concerned, but the mental impairment which has remained is very considerable. Quite recently, however, a case has been recorded by Bruce and Cottenill

in which complete recovery followed the opening of the fourth ventricle. In this case the mental dulness, the physical weakness, and the defect in vision, were so considerable that recovery was considered unlikely.

It is, of course, useless to operate in the region of the fourth ventricle if the block be higher up, and it is a matter of the greatest difficulty to decide at what point the block which causes the hydrocephalus may be situated.

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HEMATOMYELIA, HYDROMYELIA, SYRINGOMYELIA, AND CENTRAL GLIOMATOSIS.

Hæmatomyelia—hemorrhage into the substance of the spinal cord—is a condition which has been shown by Spencer to be not infrequently present in association with hemorrhages in other situations in new-born infants. It is probably due to traction on the child at the time of birth. Hæmatomyelia commonly occurs in the cervical region of the cord.

If the child survives, the spinal lesion may give rise to the typical Brown-Séquard syndrome—viz., loss of power on the one side, and loss of sensation to painful stimuli on the opposite side of the body or to a complete paraplegia. If the lesion is in the lower cervical segments, paralysis of the sympathetic, as evidenced by ptosis and failure of the pupils to dilate to shade on the side of the paralysis, may be present.

Some of the "birth palsies" may be accounted for by injury to the spinal cord either alone or in association with an injury to the roots of the brachial plexus. The diagnosis between such is impossible, and can only be revealed by post-mortem examination, as in the case recorded by Boyer.

SYMPTOMATOLOGY.—The following case, recorded by Beevor, illustrates the effect of spinal hæmorrhage:

The child was the fourth of four children; the birth, a breech presentation, was difficult, and labour lasted twelve hours. Immediately after birth the legs were noticed to be paralyzed, and the right arm was also paralyzed. The child, on examination when six weeks old, was normal so far as the cranial nerves were concerned. The left arm could be moved normally, but the right was rigidly extended and adducted to the chest wall. The legs were flexed. The knee-jerk were absent. There was paralysis of the abdominal and thoracic muscles. There was loss of sensation to the level of the second dorsal segment. The child lived till the age of fifteen weeks, and on post-mortem examination a cavity, representing an old hæmorrhage into the spinal cord, was found in the cervical and thoracic region. The paralysis of the right arm was probably due to injury of the right brachial plexus, the position being produced by the overaction of the innervated but unparalyzed muscles.

Herbert Spencer found that, out of forty-four cases of visceral hæmorrhage in still-born children in which the spinal cord was examined, hæmorrhage occurred in thirty cases. Of these, in twenty-one it was found outside the theca; in two between the dura and arachnoid; in six in the arachnoid; in three beneath the pia

water; in four in the anterior cornua; in one in Goll's column; and in only one into the whole thickness of the cord. As Beever points out, it must be a rare thing for a child with such an extensive lesion of the cord as here presented to live.

Hydromyelia.—Hydromyelia, or dilatation of the central canal of the spinal cord, occurs most frequently in association with a spinal bifida or occipital meningocele. It occurs also in association with some cases of hydrocephalus of a congenital origin. It is occasionally found post mortem in cases in which it has not been suspected during life.

Hydromyelia cannot be said to give rise to any definite group of symptoms. It would seem probable that dissociation of sensation might occur, but the cases in which the condition has been present have been either too young to be investigated or the condition has not been suspected during life.



FIG. 117.—HYDROMYELIA: SECTION OF THE CENTRAL REGION OF THE CORD OF A CHILD, SHOWING DILATATION OF THE CENTRAL CANAL.

This was associated with defective development of the cerebellum. No local manifestations occurred in association with the spinal lesion.

Syringomyelia.—It is very doubtful if a true syringomyelia occurs during child life. No case in which there was progressive muscular atrophy associated with thermo-anesthesia in a child has come under the observation of the writer, neither has he found the pathological record of any such case.

Central Gliomatosis is a condition closely allied to syringomyelia, and is due to a growth within the spinal cord. The growth would seem to start from the glia tissue in the region of the central canal, and extend from this position, always having a tendency to spread in the tissue round the central canal of the cord rather than into the white matter of the cord. Such a growth gives rise to symptoms similar to those seen in syringomyelia, and cannot clinically be distinguished from that disease.

Hæmorrhage into the Membranes of the Spinal Cord is a rare condition, and in its severe form gives rise to symptoms resembling a transverse lesion of the spinal cord. An infant who suffered from such a lesion had complete flaccid paralysis

of the lower limbs, with paralysis of the sphincter, abdominal and thoracic muscles. This hæmorrhage into the membrane was associated with hæmorrhages into the spinal cord (see *Hæmatomyelia*). Albright has recorded such a hæmorrhage into the spinal cord of a child eleven months old.

Hæmorrhage into the membrane of the spinal cord may be diagnosed by means of lumbar puncture, the fluid in the first stage being blood-stained, at a later stage being a peculiar yellow colour, due to xanthochrome (From), and not giving the ordinary blood-tests.

PROGNOSIS.—The prognosis in the above conditions depends upon the nature of the lesion. In a syringomyelia and central gliomatosis the symptoms are slowly progressive. In hydromyelia there are either no symptoms, or the symptoms are stationary, whereas in hæmatomyelia there is a tendency for the symptoms to diminish.

The conditions are, however, so rare that no prognosis should be given until the diagnosis has been long and carefully considered.

TREATMENT.—Massage and passive movements may be used for the paralyzed limbs but the result of such treatment will depend upon the nature and extent of the lesion.

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PARALYSIS OF THE CRANIAL NERVES: CONGENITAL, ACQUIRED, RECURRENT.

The paralyses of the cranial nerves are best dealt with under three separate headings: (1) Congenital, (2) acquired, (3) recurrent.

1. Congenital Paralysis of the Cranial Nerves.

Congenital paralysis of one or more of the cranial nerves may be said to be a rare affection. Instances of paralysis of the third, fifth, sixth, seventh, ninth, and twelfth cranial nerves have come under the observation of the writer.

Congenital Paralysis of the Third Nerve is commonly bilateral, and gives rise to a complete paralysis of the external muscles of the eyes supplied by that nerve, and to a partial or complete ptosis. It may be associated with paralysis of the sixth nerve, so that there is a complete ophthalmoplegia externa. The pupils usually react to light, and vision is good.

The condition may occur in more than one member of a family. An instance of such was that of a Jewish boy, aged six, who suffered from this affection, while his elder brother was similarly affected. The younger brother died suddenly whilst eating a péc, a mass of meat being found impacted in the glottis. The right subclavian artery in this boy had a peculiar origin from the thoracic aorta, and passed behind the œsophagus to reach its normal situation. The sudden failure of deglutition has been noted in other cases in which this abnormality has been

present, and such cases have been described under the name of "dysphagia lusca" (see p. 146).

Bradburne has described congenital ophthalmoplegia in five generations of a family.

Congenital and Familial Nystagmus is a rare condition. Nettleship has collected twenty-five pedigrees. Some of these would appear to be due to diseases or defects of the retina, chorioid or optic nerve, but others cannot be attributed to such causes.

Congenital Paralysis of the Sixth Nerve manifests itself by paralysis of the external rectus muscles of the eye, and results in the patient being unable to move the eyes in an outward direction. Such a paralysis may give rise to an internal strabismus.

In some cases the patient is unable to move the eyes in the lateral direction, whereas the movements upward, downward, and on convergence, are good. A child with this affection has, in order to see an object at the side, to turn the whole head. The failure of the associated lateral movements of the eye whilst the movements for convergence remain good, on theoretical grounds, suggests that the lesion is situated in the nuclei of the sixth nerve, and not in the nerve itself. The pathological proof for this, however, is not forthcoming.

Congenital Paralysis of the Fifth Nerve occurs in association with that of other cranial nerves, and manifests itself by weakness of the masseter and temporal muscles.

Congenital Paralysis of the Seventh Nerve (Facial Paralysis).—This occurs in association with other cranial nerve defects, and may be bilateral. Under this heading are not included those cases of facial paralysis which are due to some injury to the facial nerve at the time of birth, whether by forceps or other trauma. There is a true facial paralysis of congenital origin, due in all probability to a defect in the development of the seventh nerve nucleus (Babouzeuse and Heron).

Bilateral weakness of the muscles of the face is seen in certain forms of myopathy, especially in the types described by Landouzy and Dujéine. Such a facial weakness cannot be regarded as due to a nuclear defect, and is almost certainly a muscular defect.

Congenital Paralysis of the Twelfth Nerve.—Both unilateral and bilateral affections of the nucleus of the twelfth nerve occur, giving rise to atrophy and loss of power of one or both sides of the tongue. The affection of the tongue is sometimes seen in association with a facial paralysis. The furrowed appearance of the tongue is very striking.

A peculiar congenital affection of the motor portion of the fifth, ninth, tenth, and eleventh nerves is illustrated by the following case: A child born at full time of healthy parents had, since the time of birth, never been able to shut its mouth or suck, could not cry, and had weakness of the muscles of the neck. The child was well nourished, cried in a feeble manner like a diphtheritic paralysis; the jaw fell open, and if closed fell open again; there was almost complete loss of power on both sides of the face; the ocular movements were good. The movements of the tongue were relatively good, and, when the child was fed, it was apparently by the movements of the tongue that milk passed into the pharynx. The child did not swallow. Milk poured into the mouth flowed down the oesophagus.

The child otherwise appeared perfectly healthy, and the reflexes were normal. The child died, and, after a most careful microscopical examination of the nervous system, no lesion could be found in any of the cranial nuclei affected to account for the symptoms.

2. *Acquired Cranial Nerve Palsy.*

Acquired Paralysis of the Third Nerve may be due to a variety of lesions which interrupt the fibres of this nerve from their origin in the region of the corpora quadrigemina to the termination in the muscles they supply in the eye, and it is only by a careful consideration of the conditions under which the paralysis arises, and the effect on other structures in the neighbourhood, that the cause and seat of the lesion can be diagnosed.

Tumours, vascular lesions, birth injuries, meningitis, and tumours of the orbit, may produce paralysis of the third nerve.

Since the nuclei and the third nerves at their exit from the brain lie in close proximity to one another, it is likely that a single lesion causing a bilateral affection of the third nerve will be situated either in the region of the corpora quadrigemina or in the interpeduncular space; but a bilateral lesion of the third nerves in any part of their course must be considered as a possible cause of symmetrical affection of the two eyes. When certain muscles supplied by the third nerve are paralyzed whilst others are unaffected, it is probable that the lesion lies in the region of the third nerve nucleus. If, for example, the upward and downward movements of the eyes are paralyzed whilst the lateral movements remain good, the lesion is almost certainly situated in the anterior portion of the nucleus of the third nerve.

A slowly progressive ophthalmoplegia in childhood always suggests the probability of tumour. The rapid onset of a unilateral paralysis suggests meningitis.

A partial paralysis of the third nerve, with rhythmic movements of the pupil and of the levator palpebre, occurs.

[The dilatation of the pupil is accompanied by drooping of the eyelid, retraction of the pupil by elevation of the eyelid. The rhythmic action may occur at regular intervals, the period between the contractions being forty to fifty seconds. Cases of this and allied conditions had been described by Greaves, Cramer, and Ormond.

Acquired Paralysis of the Sixth Nerve.—The sixth nerve, owing to its long course, is most readily paralyzed by pressure. Paralysis of this nerve always suggests the possibility of meningitis, but there is no doubt that this nerve may be affected by simple oedema or serous meningitis, conditions which often occur in association with mastoid disease. Although an alarming symptom in this disease, it often is of transient duration, and passes off after a few days or weeks.

In lesions of the pons it is an early and marked symptom. Paralysis of the sixth nerve is a common symptom in any case of increased intracranial pressure, whether from tumour, meningitis, or other cause. It is a symptom to which so great localizing value should be attached.

Acquired Paralysis of the Motor Portion of the Fifth Nerve.—Paralysis of the motor portion of the fifth nerve gives rise to weakness of the temporal and masseter muscles, and to deviation of the jaw to the paralyzed side. It is most commonly seen in association with gross lesion of the pons. It occurs also in association with lesion of the other cranial nerves, but as an isolated symptom is of very rare occurrence.

Acquired Paralysis of the Seventh Cranial Nerve.—No cranial nerve is so frequently affected as that supplying the muscles of the face. This is chiefly due to its long course through a bony canal. The exposed position and its close relationship to the middle ear are also factors which play an important part in the causation.

Facial paralysis which is present at birth is often due to pressure of the forceps, and in most cases rapidly passes off, unless there has been actual rupture of the nerve. In infants the common cause of facial paralysis is an affection of the middle ear. It may also result from damage to the facial nerve during operation. In some children it occurs without any evidence of middle-ear disease, and is presumably due to pressure on the nerve caused by the inflammation of the periosteum of the bony canal in which the nerve lies. These cases are often attributed to rheumatism or cold, but are not infrequently associated with septic teeth.

There is the group of cases already mentioned associated with poliomyelitis (see p. 772). Tumours situated at the base of the brain at the ponto-cerebellar angle are very liable to give rise to facial paralysis. In the various forms of meningitis facial paralysis is liable to occur.

Acquired Paralysis of the Ninth, Tenth, Eleventh, and Twelfth Cranial Nerves.—A unilateral paralysis of the palate, of the vocal cord, and of the tongue, on the same side is a rare manifestation in a child. It occurs in tuberculous disease of the base at the base of the skull, and in the thickening of the membranes in this region.

Tuberculous masses in the region of the medulla may give rise to such symptoms, but the symptoms are then usually bilateral.

A tumour growing from the base of the skull may involve these nerves, and such a growth may be felt on examination of the post-pharyngeal region.

3. *Recurrent Paralysis.*

The only cranial nerve which appears to be liable to recurrent paralysis is the third. The onset of the paralysis may be attended with headache and marked constitutional symptoms, and in the first attack a serious view as to the symptoms of the paralysis might be taken. The paralysis of the muscles involved may pass off completely in a few days or few weeks. After an interval of months or years another and similar attack may occur. The duration of the second attack may be longer. These attacks have been designated "ophthalmic migraine." The cause of the condition is uncertain. Some of the cases have been shown to be due to the presence of a fibroma in the neighbourhood of the nerve. The condition is rare in childhood.

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DIPHTHERITIC PARALYSIS.

ETIOLOGY.—Diphtheria is not infrequently followed by paralysis. The clinical features of such a paralysis form a very definite symptom-complex which is not produced by any other toxin.

There are many toxins which will cause feebleness of the cardiac action, loss of knee-jerks, and general weakness; but there is no known toxin which will cause the symptom-complex known as "diphtheritic paralysis."

ETIOLOGY.—There is but one cause of diphtheritic paralysis, and that is the presence of the Klebs-Loeffler bacillus. The organisms need not be present in the throat, but its presence in the lips, nose, ears, eye, vagina, or on the surface of a wound, may be the origin of the toxin which gives rise to the paralysis.

It is not necessarily the severest cases of diphtheria which are followed by paralysis. Sometimes it follows the most mild cases. The toxin affects strong and feeble children alike. There is, however, a greater danger of cardiac failure following on a severe case of diphtheria than on a mild one (*cf.* Chapter XIX., p. 1009).

SYMPTOMATOLOGY.—The first symptom to appear is usually that of weakness of the palate. This may manifest itself by the nasal voice, and by the regurgitation of liquids taken by the mouth through the nose. This symptom usually occurs about ten to fourteen days after the onset of the diphtheria, but in some cases it may be delayed till between the sixth and eighth week after the sore throat.

The next symptom that occurs is the weakness of one or more of the ocular muscles. Those most frequently affected are the external recti and the muscles of accommodation. Ptosis not infrequently occurs.

During the early stages of the disease the knee-jerks may be present, and in some cases even active; but in the later stages they are almost always absent. As the paralysis advances, the weakness of the limbs and trunk muscles become manifest. There is usually no movement which the child cannot perform, but the power is most feeble. The diaphragm becomes paralyzed, and respiration is carried on by the intercostal and accessory muscles of respiration. During each inspiration the abdomen, instead of becoming prominent, tends to sink in. The action of the heart becomes feeble and irregular, the first sound being inaudible at the apex. Vomiting may occur, and it is a symptom to which most careful attention should be paid. It may be indicative of some affection of the vagus. When vomiting occurs as an early sign in diphtheritic paralysis, the greatest care should be exercised in the feeding of the child.

The symptoms as above depicted are those seen when the child has been under careful supervision, and has been kept at rest. When, however, a child has been allowed to get up, the symptoms which most often attract attention are gait and unsteadiness in walking. The unsteadiness in walking may be due to actual weakness, but in some cases there may be the most marked ataxia, and the child staggers about and uses the hands in a manner suggestive of cerebellar disease. The physical examination of a child with diphtheritic paralysis will show paralysis of one or more ocular muscles, paralysis of accommodation, paralysis of the palate and pharynx, weakness of the muscles of the neck, paralysis of the diaphragm, feebleness of the cardiac action, loss of knee and ankle jerks, with as a rule preservation

of the superficial reflexes. Sensation is usually unimpaired, but occasionally loss of sensation may be detected in the peripheral portion of the limbs. The limbs are not as a rule tender or painful on movement. Cases, however, occur in which there is the most severe pain on any movement of the legs. The amount of muscular wasting which occurs is usually slight, but in some cases it may be considerable, and may leave a permanent weakness and atrophy of muscles.

The condition of the heart should always be most carefully examined, for it is by the recognition of the early indications of disordered action that much may be done to prevent and guard against a sudden failure. Apart from intermission and irregularity of the action, especial attention should be paid to the character of the sounds, and when the sounds become even in character and the diastole becomes shortened so that the space between any two heart sounds is almost equal, then the greatest care should be exercised with regard to preventing any strain on the cardiac action.

The course of diphtheritic paralysis is subject to considerable variation. In some cases the paralysis remains limited to the pharyngeal and ocular muscles, and, as these symptoms usually pass off in four to five weeks, recovery may be complete in six to eight weeks after the initial attack of diphtheria. The paralysis of the diaphragm does not usually last long. In other cases, however, the paralytic symptoms are of long duration. In most cases it is the weakness of the heart from which it takes longest to recover, and gives rise to the greatest anxiety.

The return of knee-jerks is a good sign, but the knee-jerks may remain absent for months after recovery has been in other respects complete. In some cases of diphtheritic paralysis recovery is complete in two months, whilst in others recovery may take six to eight months, and even then the cardiac condition may need careful supervision. There are rare cases in which the initial lesion has been so severe that permanent paralysis of the ocular muscles, palate, and of the limbs, has resulted. Such cases are, however, most exceptional.

PATHOLOGY.—The nervous system of a case dying of diphtheritic paralysis presents nothing abnormal to the naked eye. The muscles of the heart wall may be flabby and show some fatty changes. On microscopical examination, however, marked changes can be shown to be present, not only in the anterior horn cells of the spinal cord and cranial nuclei, but also in the peripheral nerves. In those cases which die at an early stage of the disease the chromatolytic changes will be most marked in the cells, and but little alteration will be found in the peripheral portion of the motor neuron; whereas in those cases which die during the later stages of the disease, the changes will be most marked in the peripheral portion of the neuron, whilst the chromatolytic changes in the cell body may to a great extent have disappeared. The disease does not, however, limit itself to the motor neurons, for in some cases marked degeneration of the posterior roots is present. The skeletal muscles often show the most marked fatty degeneration, and the same change can be seen in the fibres of the diaphragm.

The muscles of the ventricles of the heart always show marked fatty degeneration of the fibres.

The changes found in the nervous system and in the muscles are of a degenerative nature, and interstitial changes are but rarely found.

DIAGNOSIS.—There is as a rule but little difficulty in the diagnosis of diphtheritic paralysis. The nasal voice, the regurgitation of liquid through the nose, the

ocular weakness, the paralysis of the diaphragm, the general asthenia, and the absence of knee-jerks, give a symptom-complex which is almost unmistakable.

Tumours of the pons and medulla may give rise to ocular paralysis and nasal voice, but such cases are accompanied by other symptoms suggesting pressure on the fibres of the pyramidal tract.

Progressive spinal atrophy of infants may give rise to profound asthenia, loss of knee-jerks, and some respiratory weakness; but in such cases the respiratory failure is due to weakness of the intercostals, whereas in diphtheritic paralysis it is primarily due to weakness of the diaphragm.

Certain cases of *acute poliomyelitis* may also give rise to symptoms suggesting a diphtheritic paralysis; but in such cases the onset is more acute and the paralysis more complete, though as a rule not so universal. Certain cases of *defective cerebral and cerebellar development* may give rise to symptoms very similar to diphtheritic paralysis, with flaccid paralysis, loss of knee-jerks, and difficulty in deglutition. This difficulty in diagnosis should only arise in rare instances, for mental symptoms form no part of the symptomatology of diphtheritic paralysis.

Myasthenia gravis in a child might well be mistaken for diphtheritic paralysis, but the diagnosis myasthenia should only be made after the most careful investigation, for myasthenia gravis in a child is a most rare affection.

PROGNOSIS.—The prognosis in cases of diphtheritic paralysis is good so long as the nature of the condition is recognized and suitable treatment adopted. The cases in which a fatal result may be expected are those in which vomiting occurs. Such cases often do not appear to be seriously ill, and yet are extremely liable to die suddenly from failure of cardiac action.

TREATMENT.—The treatment of a case of diphtheritic paralysis is almost entirely a matter of careful nursing. The child should be kept in a lying position, and not allowed to be raised into the sitting position under any pretext. During the period in which there is paralysis of the palate all liquid food should be thickened by the addition of starch in some form. The child should be fed with small quantities every three hours, and the greatest care must be exercised in feeding the child, so as to avoid choking. Should the effort of deglutition be difficult or seem to exhaust the child, it is well to resort to feeding by the nasal tube at once, and there is no difficulty in getting the child perfectly used to this form of feeding. The regulation of the bowels is important, and such purgatives should be used as do not give rise to violent peristaltic action, small doses of castor oil being usually effective in this respect. Should vomiting occur, it is important to regulate the feeding by reducing the quantity given at any one time, and to increase the frequency of feeding. It is often advisable to give the food in a pre-digested condition—i.e., to peptonize the milk and convert the starch.

With regard to drugs, I believe that the hypodermic injection of $\frac{1}{16}$ of a grain of strychnine and $\frac{1}{32}$ of a grain of atropine, given every four hours, is of the greatest value in preventing heart and respiratory failure. In some cases in which there appeared no indication for its administration, the writer has had cause to regret the omission after the sudden death of the child.

Alcoholic stimulants are often of value, but should be given at such times as there is evidence of failure of circulation rather than as a routine at stated intervals.

With regard to electrical treatment, I believe that it is not only useless, but actually harmful. To endeavour to stimulate the weakly-acting diaphragm with

an electrical current gives rise to a considerable amount of emotional disturbance in a child, and has, in my experience, rather tended to hasten than prevent the fatal result.

When the child is convalescent, electricity and massage will do no harm.

The administration of antidiphtheritic serum has been advocated in cases of diphtheritic paralysis; but this line of treatment seems unnecessary, and may, if the appearance of the serum rash coincides with the intensity of the paralytic symptoms, be a factor in determining a fatal issue.

Careful nursing is the one important factor in the treatment of a severe case of diphtheritic paralysis.

BIRTH INJURY TO THE BRACHIAL PLEXUS.

INTRODUCTION.—Injury to the nerves of the brachial plexus at birth occurs both in normal and abnormal presentations, but there is no doubt that traction exerted on the arm is the common cause of brachial plexus paralysis. The injury may involve the upper or lower portion of the brachial plexus, or sometimes both portions may be involved. When the upper portion is involved, a paralysis of the deltoid, supra- and infra-spinati, biceps, and supinator longus, results. Such a paralysis is often designated an "Erb's paralysis," since he first described it. When the lower portion of the plexus is involved, the muscles of the hand and flexors of the wrist are principally affected, and such cases are often designated "Klumpke's paralysis," since she first described this form.

SYMPTOMATOLOGY.—Loss of power is noticed in the affected arm immediately or soon after birth. The limb usually hangs flaccid by the side of the child, and movements are only made with the forearm and fingers. After the first few days the arm is rotated inward and the palm of this hand turns so as to face outward, and if the condition persists the contraction of the pectoral muscle renders this position more or less permanent. The arm is usually extended, and cannot be readily flexed at the elbow. In cases in which the lower portion of the plexus is involved, it is the hand and forearm muscles which are affected, whilst the upper arm remains normal.

Almost every degree of paralysis is seen—from cases in which there is only weakness of the deltoid to those in which there is a complete paralysis of all the muscles of the arms and shoulders.

It is a striking fact that in most cases sensation is little if at all involved. In lesions affecting the eighth cervical and first dorsal root there may be evidence of paralysis of the sympathetic on the side of the lesion—i.e., slight ptosis, a small pupil, and failure of the pupil to dilate when shaded or on the instillation of cocaine.

PATHEOLOGY.—The condition is due to traction on, to rupture of, or to hæmorrhage into, the nerve trunks of the brachial plexus. The rupture usually occurs after the junction of the fifth and sixth roots of the brachial plexus to form the upper cord. In some cases all the cords of the brachial plexus may be involved in fibrous tissue.

The injury may be so severe that damage to the spinal cord may occur at the same time. Such a condition could only be diagnosed if the injury to the cord were such as to give rise to affection of the pyramidal tract on the same side, and in such an event it would be impossible to say whether the paralysis of the arm trunks was due to the lesion in the cord or to the injury of the brachial plexus.

That both injury to the cord and to the plexus may coexist has been proved pathologically by Boyer.

DIAGNOSIS.—The diagnosis presents but little difficulty. Dislocation of shoulders might give rise to a weakness of the arm, but this cause would be readily recognized. A paralysis of the arm from a hemiplegic lesion would be associated with weakness of the face and legs on the same side. A monoplegia of cerebral origin might give rise to some difficulty in diagnosis, but such a condition is very rare. Neither the absence of wasting, alteration of electrical reaction, or rigidity, points of importance in older children, are of much service in infants. If the condition is seen some weeks after birth, an epiphysitis must be thought of; but epiphysitis commonly gives rise to a flaccid paralysis, and the enlargement of the epiphysis will be evident, if not immediately, at any rate a few days after the paralysis is first noticed. It is impossible in an infant to differentiate a lesion in the spinal cord alone for one in which the brachial plexus and the spinal cord are simultaneously affected.

PROGNOSIS.—The prognosis in most cases is favourable. In those which recover rapidly it is probable that traction has produced a physiological interruption, and after a few days or weeks complete recovery of function occurs.

In some few cases recovery takes place after three to six months. In these it is probable that there has been some rupture of fibres, and regeneration has slowly taken place.

In other cases only partial recovery takes place. In such cases matting around the brachial plexus is generally found.

In other cases no recovery takes place, and in these extensive injury of the plexus and spinal cord are found.

TREATMENT.—The slighter cases may be left to themselves to recover, which they do in a few days or weeks.

In the more severe cases massage, passive movements, and electrical treatment probably assist the recovery.

In the severest cases the operation of separating the bundles of the plexus from the adhesions which surround them is necessary. It must be remembered, however, that such an operation can be of no service in those cases in which the spinal cord is injured.

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VON RECKLINGHAUSEN'S DISEASE—DIFFUSE OR GENERALIZED NEUROFIBROMATOSIS.

INTRODUCTION.—This disease was first described by von Recklinghausen in 1882 and is characterized by the presence of (1) multiple tumours of the skin (molluscum fibrosum); (2) subcutaneous tumours situated on the nerves, either plexiform or multiple (fibrosarcomatosis); (3) pigmentation of the skin.

The tumours may be situated on any of the peripheral nerves, the cranial nerves, the nerve roots of the spinal cord, or on the sympathetic nerves.

Ætiology.—There is good evidence that the condition is sometimes hereditary, and that it is often a familial disease. Ten such cases have been collected and recorded by Alexis Thomson. It is probable that in many cases the disease dates from intra-uterine life. The tumours of the skin, pigmentation spots, and tumours of the nerves, have all been observed at birth or in early childhood. These may either remain stationary for many years or may at any time, and especially at puberty, take on active growth. The disease affects males and females alike. Of the seventy-seven cases collected by Alexis Thomson, twenty-eight presented symptoms at birth or early in life.

There is no evidence to show that the disease is ever the result of any external influence, although traumatism and pressure have been regarded as exciting causes.

Neurofibromatosis must be regarded as a developmental disease, and as a form of elephantiasis of the connective tissue of the peripheral nervous system.

Symptomatology.—Tumours of various sizes may be felt in the subcutaneous tissue along the course of the cutaneous nerves. These may vary in size from a pin's head to a large mass when situated in the brachial, lumbar, or sacral plexuses. These tumours are usually quite insensitive, and may be manipulated without causing any pain; sometimes, however, one or more may be sensitive.

Although these tumours are situated on the nerves, they rarely give rise to anaesthesia or to actual paralysis of muscles. When, however, the tumours are situated on the spinal roots or on the cranial nerves, they more frequently give rise to symptoms.

The involvement of the sympathetic may sometimes cause symptoms of sympathetic paralysis.

The *plexiform* variety may give rise to a considerable tumour and to a condition of *elephantiasis of the skin*. Such tumours are often present at birth, and may remain stationary or take on active growth.

Pigmentation of the skin is present in two forms: the first a *pusilliform* variety like dark brown freckles, and the second in which large areas of skin are involved.

Multiple pedunculated tumours project from the surface of the skin. Sometimes the whole surface of the body is covered with these. In some of these convoluted cords in the form of a plexus can be felt.

Some of the children affected with this disease are mentally defective.

Pathology.—The nerves are not only the seat of multiple tumours, but are diffusely and unequally thickened; it may be in their whole course from their origin in the brain or spinal cord to their termination in the muscles or skin.

The growths are usually elliptical in shape; they are encapsulated by the external sheath of the nerve trunk.

The substance of the tumours is usually firm, greyish white in appearance, and but little vascular.

The nerve trunks may be thickened so that they attain two or three times their normal dimensions.

The growth takes place in the endoneurial connective tissue between the individual nerve fibres, and affects the various bundles in a given nerve trunk in a most irregular manner, certain bundles being affected at one level and exempt at another.

It is probable that in most cases the nerve fibres pass through the tumours and are unaffected thereby, but in exceptional cases degeneration may occur.

The growth is made up of delicate fibrillated network separating the original fibres of the endoneurium; the fibrils of the network are branching processes of spindle-shaped cells with oval nuclei. The characters of the growths are remarkably constant in different cases.

DIAGNOSIS.—The diagnosis presents no difficulty in a typical case where there is pigmentation of the skin with multiple tumours on the nerves. A difficulty might arise in a case in which the tumour appeared in the neck, for this might be mistaken for a tuberculous gland.

PROGNOSIS.—The disease may remain stationary for an indefinite period. It may, however, take on an active stage, and, when it occurs in the cranial nerve, gives rise to symptoms of an intracranial tumour, and thus prove fatal.

TREATMENT.—There is no treatment. It is important to avoid operation, as that can only be attended by the disastrous results either of producing paralysis in the distribution of the nerve or nerves involved or of starting active growth of a sarcomatous nature in the tumours.

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POLYNEURITIS, OR MULTIPLE NEURITIS (TOXIC NEURITIS).

INTRODUCTION.—The occurrence of a true multiple neuritis in childhood is a comparatively rare disease. The causes which give rise to this affection in the adult are not common in childhood. Thus alcohol, lead, arsenic, and prolonged septic infection are not frequent in children, and yet it must be admitted that any one of the above poisons may cause multiple neuritis in childhood. The form of paralysis which follows diphtheria has been dealt with in a separate section. Whether the toxin of poliomyelitis can produce a true neuritis is doubtful; that it can produce symptoms which most closely resemble a neuritis is certain, and such cases have been described under the heading "poliomyelitis." New growths in the peripheral nerves occur, one form of which has been described in the chapter dealing with neurofibromatosis. A peculiar group of cases with recurrent multiple neuritis has been dealt with under a separate heading, and another group of cases with progressive muscular atrophy is the chapter dealing with that disease.

ETIOLOGY.—Of the poisons above mentioned arsenic is probably the most common cause of multiple neuritis in childhood, owing to the administration of large doses of this drug in chlores without sufficient supervision.

The method in which lead gets into the system is often most difficult to ascertain, but cases have arisen in which the child has sucked painted toys, lead objects, and from the use of lead plasters.

Alcohol may, in association with prolonged suppuration, give rise to symptoms of multiple neuritis, but alcohol alone must be a most rare cause of neuritis in children.

The cases of neuritis which follow the acute specific fevers—measles, whooping-cough, scarlet fever, and influenza—are most difficult to classify, and the factor which is causative in such cases is most difficult to identify.

SYMPTOMATOLOGY.—Weakness of the legs, and especially of the extensors of the feet, is generally the earliest manifestation of a peripheral neuritis. There is wasting of the muscles of the legs below the knee, and the child walks with the typical "foot-drop" gait. The hands, too, may exhibit "wrist-drop."

The proximal muscles of the arms and legs are in most cases relatively powerful. The knee-jerks are usually absent, and their absence may be the first indication of the poisonous effect of arsenic on the nervous system, and in a case of chorea treated by large doses of arsenic it is a strong indication to stop the administration of the drug.

The muscles may be tender on palpation, but this is by no means a constant feature, and cases of lead and arsenic neuritis often run their whole course without any tenderness or alteration of sensation. Vasomotor changes are not infrequently present in the skin.

DIAGNOSIS.—The diagnosis of a case of multiple neuritis is not difficult, but it is a most difficult matter to assign the cause of the neuritis. In lead neuritis a blue line on the gums will be of service in aiding diagnosis. In arsenic there may be a history of its administration, or it may be detected in the hair. Certain forms of myopathy have foot-drop as an early manifestation, and these must not be confused with a neuritis.

PROGNOSIS.—The outlook in a case of true multiple neuritis, if the toxic agent can be removed, is good. Most cases under suitable conditions make a complete recovery. There are, however, cases in which the continued administration of arsenic has given rise to a permanent paralysis.

TREATMENT.—Treatment is simple—elimination of the poison, rest, massage, and the application of electricity in that form which gives rise to contraction of the affected muscles. Splints are essential to prevent the occurrence of deformity, and their right application is an important factor in promoting and accelerating recovery.

RECURRING POLYNEURITIS.

The recurrence of polyneuritis is a very rare condition in children. Some of the cases which have been described under this title probably belong to the group of family periodic paralysis, and the case of a boy, aged fourteen, reported by Benstein, probably belongs to that group, although electrical changes were said to have been present in various muscles. Only one such case has come under the observation of the writer—a boy, aged eight, who had gradual onset of weakness of the legs with foot-drop. Although the weakness was most marked in the legs, it was not limited to them. There was some swelling of the feet; he had the greatest difficulty in getting up from the ground, and during one period of his recovery "limbed up his legs" like a myopathy. All the deep reflexes were absent. Sensation was very slightly if at all impaired, and there was no tenderness of the nerve trunks. The muscles gave a very diminished reaction to both galvanism and faradism, and a slow response to the former. No cause for the paralysis could be found.

There was no reason to suspect alcohol, and the very absence of tenderness was against this form of poison. There was no evidence of either lead or arsenic. The boy recovered from the attack in about four months; the knee-jerks returned, and the muscles reacted normally.

Four months later he had another attack of exactly similar nature—weakness and swelling of the legs, with loss of knee-jerks—without disturbance of the general health. For this attack no cause could be found, and he again made a good recovery.

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PROGRESSIVE "SPINAL" MUSCULAR ATROPHY OF INFANTS AND YOUNG CHILDREN (WERDNIG-HOFFMANN TYPE).

INTRODUCTION.—Progressive muscular atrophy occurs in infants and children from a variety of conditions dependent on diseases of joints, diseases of the muscles, affections of the peripheral nerve, spinal and cerebral lesions. Some of these conditions give rise to local weakness and atrophy, others to more general paralysis and wasting.

The special form of muscular atrophy here considered was originally described by Werdnig in 1891, and Hoffmann in 1893, under the title "Progressive Spinal Muscular Atrophy in Early Childhood." The word "spinal" indicated that the disease was due primarily to a spinal and not a muscular lesion. The authors also emphasized the familial character of the disease. Since this disease was first recognized some 16 cases have been recorded which have been confirmed by pathological examination—2 by Werdnig, 3 by Hoffmann, 1 by Brace and Thomson, 1 by Beevor, 2 by von Ritter, 2 by Bruns, 2 by Batten and Holmes, 1 by Parsons and Stanley. Other cases without a pathological examination are recorded by Serestre, Wimmer, Bruns, Senator, and Lange. Some of these, and especially those described by the last-named author, should not be accepted as instances of the disease without a pathological examination.

ETIOLOGY.—As to the direct cause of the disease nothing is known, but there is some evidence from the pathological examination of some cases to suggest that it may be due to a toxin acting during intra- or extra-uterine life. On the other hand, the occurrence of the disease in families, although not absolutely excluding the toxic theory, yet would seem to point to the condition being due to what has been designated by Gowers as abiotrophy, "a degeneration dependent on a defective vital endowment."

Family Character.—Werdnig gives an instance in which 2 brothers in a family of 6 were affected; Hoffmann 3 families, in the first of which 6 out of 15 were affected—4 boys and 2 girls; a second family of 9 in which 3 were affected—2 boys and 1 girl; and a third family of 12 in which 8 were affected, and an unaffected female member of this family had 6 children, 3 of whom were affected. Beevor describes a family of 8 in which 4 were affected.

Age.—The disease seems to affect males and females alike. The age at which the disease first manifests itself varies. In Werdnig's and Hoffmann's cases the first symptoms were noticed when the child was eight to ten months old. In other cases the disease would seem to have been present at birth, or a few weeks after birth (Beover, Serestre, and Ratten's cases).

Symptomatology.—The clinical features of this disease may be described as follows: An apparently healthy and intelligent child who has made normal progress during the first few weeks or months of life begins, without any sudden onset, manifestations of acute disease, or known causes, to lose power. If the child has already stood, it loses that power; it then loses the power of sitting up, and finally even the power of sitting up when placed in that position. The loss of power is first noticed in the muscles of the hip and then in those of the back, the shoulders, thighs, upper arm, forearm, and leg muscles, being successively affected in the order of parts as above mentioned. The paralysis of the proximal as compared to the distal muscles is a very striking feature of the disease, for the child will move the fingers and toes whilst the arms and legs are completely paralyzed.

The intercostal and abdominal muscles become affected with the other trunk muscles, whilst the power of the diaphragm remains intact.

Fibrillary twitching of the muscles may be present in some cases; but the amount of subcutaneous fat often covers up the wasting of the muscles and makes it difficult to observe such fibrillary twitching.

The neck muscles become weak, and the child is unable to hold up the head. Bulbar symptoms are said sometimes to supervene, and contraction of the limbs may be present.

The affected muscles will not as a rule react to the faradic or galvanic stimulations, and the reaction of degeneration is said sometimes to be present. The child will often bear without any manifestation of pain a faradic current so strong that it will produce a tetanus in the normal muscles, but react even comparatively weak galvanic currents. All the deep reflexes are abolished, and generally the superficial reflexes are also absent. There is as a rule no pain or tenderness. There is no alteration of sensation, and the child usually responds to all cutaneous stimuli.

The mental condition of the child continues unimpaired throughout the whole course of the disease, and the child takes notice, talks, laughs, and swallows its food in a perfectly normal way.

The happy and contented aspect of these children is a striking feature when



FIG. 115.—PROGRESSIVE SPINAL MUSCULAR ATROPHY (WERNIG-HOFFMANN); PHOTOGRAPH OF A CHILD TWO YEARS OLD BEING HELD UP BY A NURSE.

The head falls forward, and the arms "come away" from the trunk. The child has no power to kick the legs, move the arms, or raise the head. (Ratten and Böhm.)

seen in association with the complete flaccid paralysis of the trunk and limbs. Although the muscles are in a state of flaccid paralysis, yet there is not that marked condition of hypotonia—i.e., loss of tone—seen in some of the myopathic conditions, and the legs and arms of the child cannot be placed in the abnormal positions which are so well seen in the form of myopathy known under the name "myotonia congenita." The sphincters are normal.

PATHOLOGY.—The pathology of the disease is based on the examination of some sixteen cases by Wendig, Hoffmann, Bruce and Thomson, Beevor, Armand, Delille and Berdet, Bruns, Holmes, Stanley, Parsons, and Batten.

To the naked eye the only change visible is the atrophy of the anterior roots of the spinal cord and the paleness and smallness of the muscles of the limbs and trunk.

On microscopical examination there is atrophy of the cells of the anterior horns, and atrophy of the fibres of the lower motor neuron passing therefrom. In some of the cases which have died in an early stage of the disease the changes in the anterior roots and anterior horn cells are such as to suggest that some poison is acting on the lower motor neuron, whereas the cases examined in the late stage of the disease suggest that atrophy has resulted from the effect of the poison.

The change found in the muscles is atrophy of some bundles of fibres, leaving other muscle fibres unaffected. Hypertrophy of individual fibres has also been described; but the general appearance of the muscles suggests an atrophy secondary to some spinal change. The absence of the intermuscular fibrosis as compared to that which occurs in cases of myopathy is a striking feature. The peripheral nerves show some degenerate fibres.

In individual cases other changes have been described—viz., degeneration of the fibres of the posterior columns; but in most of the cases not only does the white matter of the cord appear normal, but no change can be found in any part of the nervous system other than that mentioned above.

DIAGNOSIS.—The diagnosis of cases of this disease is by no means easy. Not only have the clinical features to be considered, but the history of the onset is of considerable importance.

An infant who at the time of birth is said to have had a complete flaccid paralysis of both arms, legs, and trunk, is probably the subject of an extensive injury to the spinal cord in the cervical region.

An infant who in the first few weeks or months of life gradually develops a flaccid paralysis of the extremities is probably the subject of a progressive spinal atrophy.

A spinal hæmorrhage, in order to produce the symptoms of a complete flaccid paralysis in the legs, with loss of all reflexes, must be fairly complete, and should involve both motor and sensory paths. The loss of sensation should furnish a distinguishing feature between the two classes of cases. It is, however, often a difficult matter to say whether an infant has or has not lost sensation, nor can any inference be drawn from the condition of the sphincters.

FIGS. 119A, 119B, 119C, 119D.—PROGRESSIVE SPINAL MUSCULAR ATROPHY: A SERIES OF PHOTOGRAPHS OF A CHILD, SHOWING PROGRESSIVE WASTING AND WEAKNESS OF MUSCLES.

The disease started when the child was twelve months old. The first photograph was taken when the child was three years old, the second when four years old, and the third and fourth when five years old (six months before death). The spinal cord showed the characteristic atrophy of the anterior horn cells. (Bruce and Thomson's case.)



FIG. 1191.



FIG. 1192.



FIG. 1193.



FIG. 1194.

It has been shown by Herbert Spencer that spinal hæmorrhage is not uncommon in stillborn infants, and a case of spinal hæmorrhage recorded by Beever shows the difficulty of distinguishing that condition from a muscular atrophy.

It is not, however, in early infant life that the difficulty in diagnosis so often arises as between the ages of six months and two years.

The lack of power and the flaccid condition of the muscles bears a striking resemblance to certain forms of myopathy which occur in early life, and especially to that described by Oppenheim under the name "*myotonia congenita*."

Myotonia congenita is a disease of infancy and early childhood characterized by an extreme degree of laxity of tone in all the muscles of the body, it being possible to place the limbs in most unusual positions in relation to the trunk. Children suffering with this disease can as a rule perform all movements in a feeble manner, and there is not the marked degree of complete flaccid paralysis seen in the case of spinal atrophy.

Another striking difference is the condition of the intercostal and abdominal muscles.

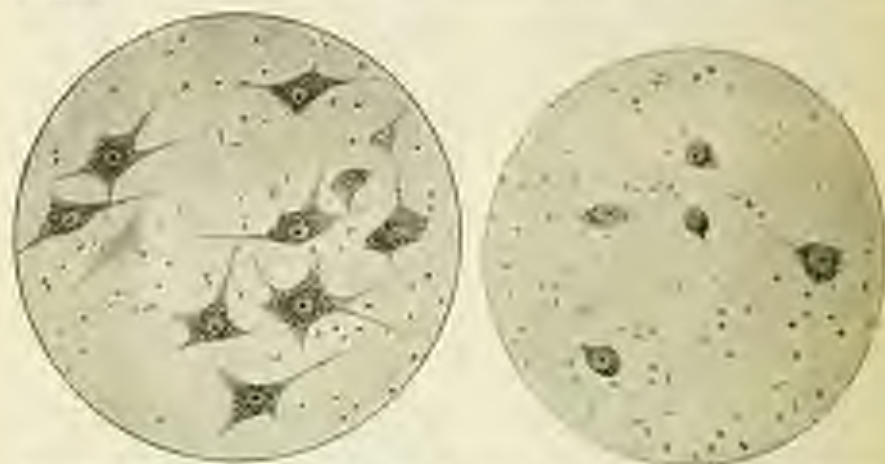


FIG. 120.—PROGRESSIVE SPINAL MUSCULAR ATROPHY: CELLS FROM THE ANTERIOR HORN OF LUMBAR REGION OF THE SPINAL CORD, SHOWING ATROPHY, WITH LOSS OF CHROMATIN SUBSTANCE.

Compare the normal cells on the left with those on the right from same segment of the cord and under the same magnification.

In spinal atrophy the intercostal and abdominal muscles are usually markedly affected, whereas in *myotonia congenita* they are but little affected.

The deep reflexes are absent in both conditions; the intelligence, the sensations, and the control of the sphincters is good in both, and the electrical reaction will not materially help in the differential diagnosis. How difficult the diagnosis may in certain cases be is shown by Finkelburg, who recorded three cases in one family which, on clinical grounds alone, would have been assigned to the Westphal-Hoffmann group, but which on pathological examination were shown to be myopathic in nature.

To put the matter shortly, in spinal atrophy the paralysis of the limbs is more marked and the hypotonia less marked than in cases of *myotonia congenita*.

Apart from the conditions mentioned above, no difficulty should arise as to the diagnosis in a well-marked case of the disease; but the clinical features are by no means constant, and the possibility of a diphtheritic paralysis or diffuse poliomyelitis must be considered.

In a case of diphtheritic paralysis there is seldom complete paralysis of the limbs, the eyes and palatal and laryngeal muscles are commonly affected, and the diaphragm is weak. In a diffuse poliomyelitis the paralysis and atrophy of the muscles is rarely symmetrical, and groups of muscles tend to be picked out, leaving others unaffected. Again, in an early case in which the paralysis has not yet become extensive, a syphilitic epiphyseitis must be borne in mind; for this condition frequently gives rise to a flaccid paralysis of one or both arms. The condition has only to be borne in mind, for the diagnosis is easily made.



FIG. 121.—TRANSVERSE SECTION OF DICEPS PEDORIS MUSCLE, FROM CHILD SUFFERING FROM PROGRESSIVE SPINAL MUSCULAR ATROPHY OF INFANTS. (WERNICKE-HOFFMANN.)

Note the extreme variation in the size of the muscle fibres. The larger fibres are about normal size (*Stow, 1912, BRIT. MED. J.*).

Another condition may rather closely resemble the spinal atrophy of infants, and that is, a condition of flaccid paralysis of the limbs and absent knee-jerks met with in certain forms of mental defect. Such cases, which are probably due to some general defect in development of the nervous system, should not be classed with the spinal atrophies.

Certain cases of spinal caries, in which the tuberculous process has extended into and invaded the spinal cord, may give rise to symptoms closely resembling the cases under consideration. The paralysis and flaccid condition of the legs being due to the extensive lesion of the cord, the presence of constitutional symptoms—of pain, of rigidity of the spine, and the evidence obtained from an X-ray—should make the diagnosis certain.

PROGNOSIS.—Once the diagnosis is established the prognosis is certain. The disease is always progressive, and death usually takes place within five years of the onset. The younger the child the more rapid is the course of the disease.

No line of treatment has been suggested for the arrest or cure of the disease.

REFERENCES.

- BATTEN: *Brain*, 1910, xxxii, 433.
BATTEN AND HUGHES: *Brain*, 1912, xxxiv, 33.
HOFMANN: *Deutsche Zeitsch. f. Nervenh.*, 1903, vi, 477.
PARSONS AND STAMMET: *Brain*, 1912, xxxiv, 50.
WARRING: *Arch. f. Psych.*, 1894, xxvi, 796.

OTHER FORMS OF PROGRESSIVE MUSCULAR ATROPHY DUE TO SPINAL DISEASE.

INTRODUCTION.—Apart from the type of progressive muscular atrophy just described, cases occur in older children which present the features of progressive muscular atrophy and weakness, but which on pathological examination are shown to be quite distinct from the above group of cases. Some of these cases would appear to be due to a condition of so-called "toxic neuritis," others to a diffuse and progressive "myelitis."

Clinical Features of "Toxic Neuritis" Group.

The clinical features of this group are well exemplified by the following case described by Voelcker: A girl, aged three, who had been quite healthy and normal, was noticed one morning to be unable to walk properly. She was able to use the arms well. Gradually her weakness increased, so that she was no longer able to sit up, and could not raise the arms. The legs became wasted, and the muscles gave a reaction of degeneration. All the deep reflexes were abolished. After the disease had progressed for three months an arrest and improvement took place, and a definite gain in power was noticed for the next two months. Then the disease again began to advance, the intercostal muscles and the diaphragm became affected, and the child died some fourteen months after the first onset of the disease. The intermissions of the disease and the variation in the severity of the symptoms from day to day was a striking feature in this case. On one occasion she developed paralysis of the right side of the face, which entirely cleared up in a few days, and did not return.

MORBID ANATOMY.—On pathological examination degeneration of the peripheral nerves and chromatolytic changes in the cells of the spinal cord were found. Fatty degeneration of the muscle fibres of the diaphragm was also present. The changes suggest a toxic degeneration of the lower motor neuron, and bring the case into the group of "toxic neuritis." The nature of the toxin is unknown. Such poisons as lead, arsenic, or diphtheria, might be considered, but neither the clinical features nor course of the disease are like the symptoms usually produced by these poisons.

Clinical Features of "Myelitic Group."

The clinical features of this group may be exemplified by a case recorded by Fletcher and Batten. A girl, aged eleven years, had noticed weakness of the hands for twelve months, and she had been unable to button her clothes for seven to eight months. For four months she had had some weakness in the legs. She complained of some pain in the back, and had some difficulty in swallowing. On examination she showed some atrophy of the tongue, weakness of the neck muscles, weakness of the intercostals. The hand was in a claw position, the wrist dropped, and there was weakness of the upper arm and shoulder muscles. She could walk but feebly. The knee-jerks were active. The weakness slowly progressed, and the child died of respiratory failure eighteen months after the onset of the disease.

MORBID ANATOMY.—The pathological examination of this case showed extensive myelitis of the cervical region of the spinal cord, affecting grey and white matter and destroying the cells of the anterior horn. No cause could be found for the myelitis, which was progressive in nature.

REFERENCES.

- BATTEN: *Brain*, 1910, XXXIII, 423.
HOLMES: *Rev. of Neurol. and Psych.*, 1905, ix, 256.
MASS: *Deutsche Zeits. f. Nervenk.*, 1911, xli, 226.

AMAUROTIC FAMILY IDIOCY (WARREN-TAY-SACH DISEASE).

INTRODUCTION.—Amautotic family idiocy is a progressive degenerative disease of the nervous system occurring in Hebrew children, affecting several members of a family, characterised by progressive mental and physical weakness, blindness, and symmetrical changes at the macula. The word "idiocy" carries with it a wrong conception of the disease, for the children have a head of normal shape and size, a well-developed brain, and are in the earlier months of life perfectly normal babies.

The following facts with regard to the history of the disease should be known: Warren-Tay in 1886 first described the changes in the fundus. Sach in 1887 described the peculiar form of mental defect associated with blindness in four members of a family. Kingdom in 1894 showed that the above conditions were one and the same disease.

The pathology has since been worked out carefully by Russell, Schaffer, Holmes, Parsons, Costes, and many others.

ETIOLOGY.—The cause of this form of degeneration is unknown. Syphilis plays no part. The disease attacks several members of a family in succession, males and females being alike affected, it usually manifests itself between the third and sixth months of life, and death occurs before the end of the third year of life. The disease is limited to the Hebrew race.

It has been questioned whether the disease is limited to the Hebrew race, and cases have been published in which the disease was said to be present in Gentile children. If these cases are examined critically, it is found that they differ, not only in the course and symptoms, but also in the nature of the changes at the macula. These cases will be considered later (see p. 846).

What the nature of the poison is that gives rise to this form of degeneration is at present unknown, but it would seem probable that it is of endogenous and not exogenous origin.

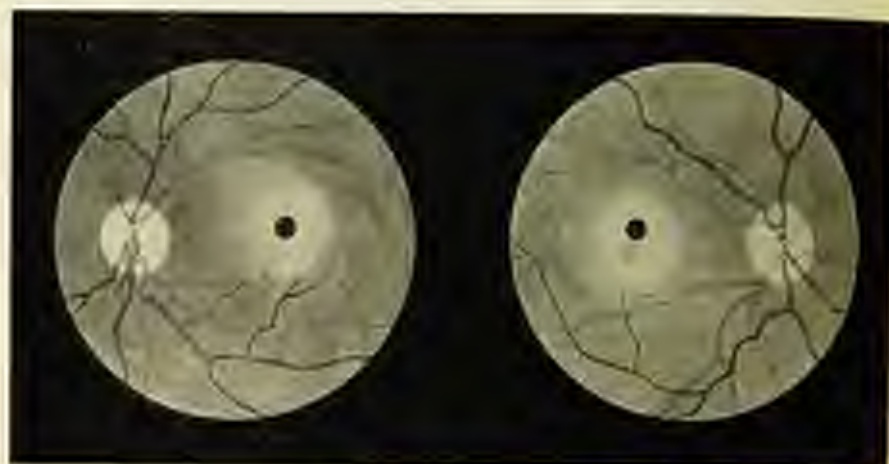


FIG. 122.—APPEARANCE OF THE REGION OF THE YELLOW SPOT IN A CASE OF FAMILY AMAUROTIC IDIOCY.

(FROM MR. WARM-TAY'S ORIGINAL.)

SYMPTOMATOLOGY.—The symptoms of this disease are so characteristic that when once the "clinical picture" has been seen it is again easily recognized.

The child is usually born at full term, and appears at the time of birth and for some three to six months afterwards perfectly normal.



FIG. 123.—PHOTOGRAPH OF A CHILD SUFFERING WITH AMAUROTIC IDIOCY.

Note the extended and pectored position of the arms and the retracted position of the head.
(With Dr. Cahill and Mott's kind permission.)

Signs of weakness then begin to manifest themselves. If the child has sat up, it loses this capacity, the neck muscles become weak, and the head falls about according to the position of gravity.

The child becomes apathetic, and no longer seems to take notice of objects,

and parents who have had previous children similarly affected notice the loss of vision before any change can be seen on ophthalmoscopic examination. Gradually the impairment of vision becomes more obvious, and the eyes wander aimlessly



FIG. 124.—PHOTOGRAPH OF CHILD SUFFERING WITH ANATOMIC DEFECT, SHOWING THE POSTURE OF HANDS.

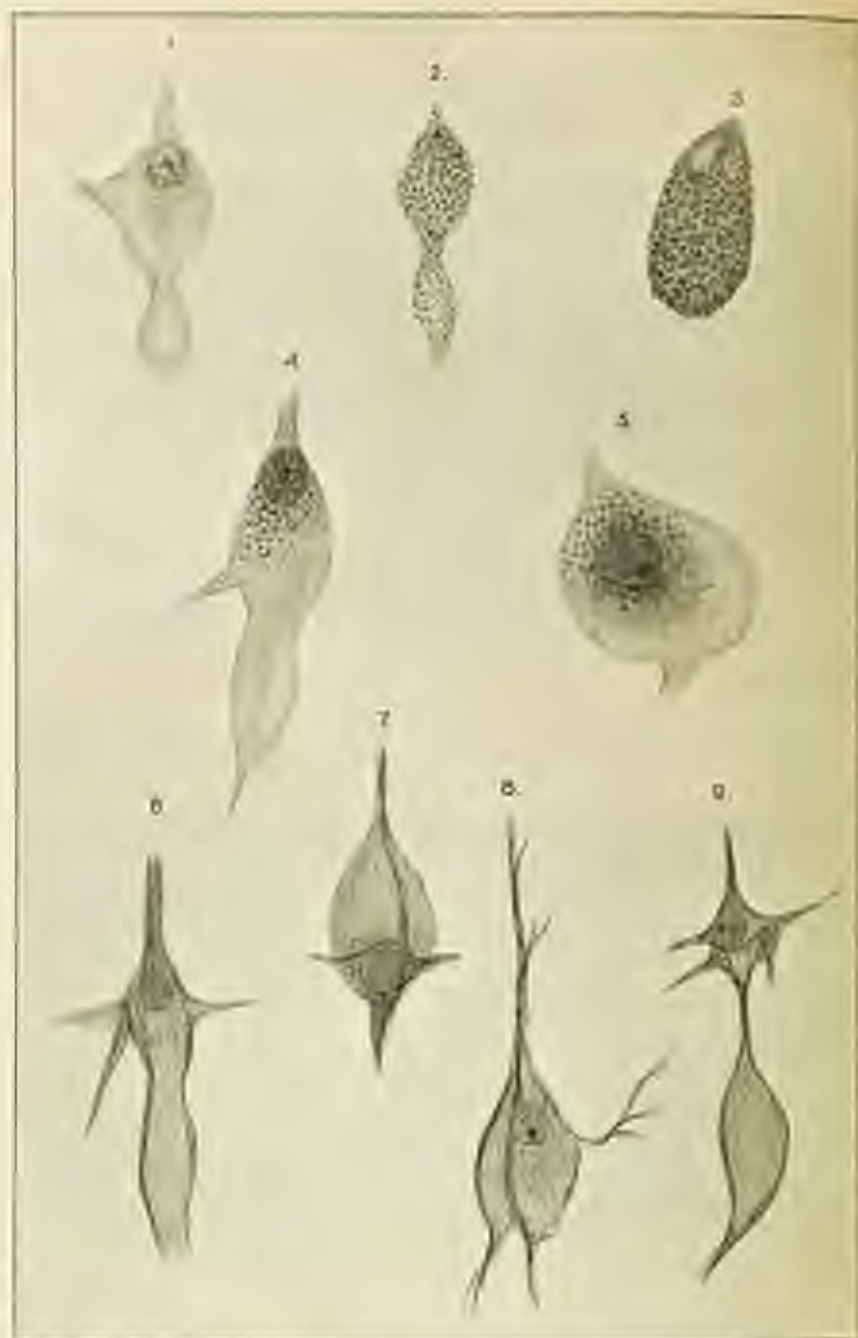
about, the child taking no notice of its surroundings, but being susceptible to sounds.

At this period the characteristic change in the fundus is present. There is optic atrophy, and the cherry-red spot surrounded by a lighter halo at the macula.



FIG. 125.—PHOTOGRAPH OF THE LIMBS OF THE SAME CHILD FIVE MONTHS LATER, SHOWING THE POSTURE AND EXTREME WASTING OF THE LIMBS.

The limbs now become rigid, the arms are extended and rotated inwards at the shoulder, the legs are extended, adducted, and crossed. The head is not usually retracted. The knee jerks at this period are usually exaggerated, and the plantars may give an extensor response. The cerebro-spinal fluid shows practically no change, a few lymphocytes may be present, and the Pehling and Noguchi tests are normal. The child now begins to waste; the hands become flexed and contracted,



2 FIG. 126.—SERIES OF NERVE CELLS, SHOWING THE CHARACTER OF CHANGE MET WITH IN CASES OF AMYOTROPHIC LEOHY.

1, 2, 3, and 4, are pyramidal cells; 5, a large spinal anterior horn cell; 6, 7, 8, and 9, multipolar cells stained by methods to show the neuro-fibræ; 9 shows a large curved swelling at the end of the processes—a characteristic feature in this disease. (Reproduced with Dr. Gold and Mott's kind permission).

as shown in Figs. 124 and 125. The feet become extended, the knee-jerks being at this time difficult to obtain (Fig. 125).

The child lies in a semiconscious condition, convulsions may occur, and a squint may be present. The child gradually loses the power of sucking, and eventually of swallowing. If nasal feeding is resorted to, the child may live for some months in an emaciated condition. Death takes place from pneumonia, or quite suddenly from cardiac failure.

PATHOLOGY.—The brain of a child who has died of this disease is of normal size and average weight. The surface is well convoluted, and the convolutions are well developed. The brain is, however, unusually firm, and has a india-rubber-like consistency, so that it can be freely handled without fear of damage. The spinal cord also appears of normal size and weight.

The membranes of the brain show practically no change.

On microscopical examination, the most striking change is the swollen appearance of the cells and of the dendrites. This condition affects not only the cells of the cortex, but also those of the cerebellum and of the spinal cord. Secondary to these changes there is degeneration of the nerve fibres and medullary sheath.

This change is well illustrated by the diagram taken from Corlill and Mott's paper (Fig. 126). Secondary to this cell change there is a proliferation of the glia, with the development of large glia cells. There is no evidence that the condition is inflammatory; it is essentially a degenerative process.

The retina shows degeneration of the ganglion cells, and the nerve fibre layer is extremely attenuated.

The characteristic appearance of the macula is probably due to an oedema of the macula, the centre of the macula appearing as a red spot in contrast to the pale surrounding tissue.

The chemical analysis of the brain shows that there is a marked relative diminution of the lipid forms of phosphorus and sulphur, with a corresponding increase of the extractive forms, the protein, sulphur, and phosphorus, showing no marked change.

DIAGNOSIS.—When once seen, the disease is easily recognized. The progressive weakness of the limbs, attended by loss of vision, in a Jewish child who has previously been in good health would at once suggest the possibility of Tay-Sachs's disease, and this would be confirmed by the appearance of the macula.

The disease should not be confused with a form of cerebral degeneration occurring in older children, in whom there is failure of vision with a curious pigmented condition around the macula.

PROGNOSIS.—The disease is invariably fatal. When once started, it runs a slowly progressive course till death ensues—nearly always within two years of the onset, and often much sooner.

TREATMENT.—So far all forms of treatment have been unavailing. In the later stages of the disease nasal feeding becomes a necessity if the child's life is to be prolonged.

REFERENCES.

- CORLILL AND MOTT: *Proc. Roy. Soc. Med., Path. Sect.*, 1911, iv, 147.
KENDRICK AND BUSHELL: *Trans. Med.-Chir. Soc.*, 1897, lxxx, 87.
POTVIN, PARSONS, AND HELMER: *Pres.*, 1906, xxix, 390.
SCHAPPEL: *Neurol. Centralblatt*, 1905, xxi, 286.
WARREN-TAY: *Ophthalm. Trans.*, 1881, i, 58.

CEREBRAL DEGENERATION WITH SYMMETRICAL CHANGES IN THE MACULE.

INTRODUCTION.—This condition is somewhat allied to the Tay-Sachs disease, but differs from it, not only in the onset and course of the disease, but also in the appearance of the macule.

ETIOLOGY.—The cause of the disease is obscure. It occurs, like the Tay-Sachs disease, in families. There is no evidence that syphilis plays any part in the production of the typical form of the disease, although in some cases, such as those recorded by Spélmeyer, with dementia and blindness from retinitis pigmentosa, inherited syphilis may be a factor. Consanguinity in marriage is thought to play some part in the production of this condition.

SYMPTOMATOLOGY.—The history obtained in these cases is that the child during the first years of life has been bright and intelligent, has gone to school and learnt to read and write, when about the age of six or seven it is noticed that the child has become dull, fails to advance, and loses the power of reading. It is then noticed that the child does not see well, and the head is turned into a lateral position, so that the object may fall upon the periphery of the fundus. A central scotoma is probably present, but it is difficult to prove, owing to the feeble mental condition of the child. The child becomes irritable, difficult to manage, dirty in habits, but does not in the early stage manifest any weakness of the limbs. The reflexes are normal, and the physical examination shows nothing abnormal with the internal organs.

The striking features of the disease are the progressive mental deterioration and defective vision, associated with pigmentation of the macula, occurring in several members of a family.

PATHOLOGY.—Not one of these cases has so far been investigated pathologically. Two sisters with the disease who were under the writer's care died in Dementia Asylum, but no post-mortem was obtained. Neither has an examination been made in the other reported cases.

DIAGNOSIS.—The diagnosis depends on the examination of the fundus. The association of mental deterioration with the macular changes are the distinguishing features.

PROGNOSIS.—The mental condition slowly degenerates, the disease lasts for seven to eight years, and the child dies from some intercurrent rather than from the cerebral disease.

TREATMENT.—So far no treatment has been found which will arrest the disease.

REFERENCES.

- BAYES: *Trans. Ophthalm. Soc.*, 1903, xxii, 396.
MAYO: *Ibid.*, 1904, xxiv, 142.
MELNBERG: *Munch. Med. Woch.*, 1903, l, 1968.
NEUBERG: 1908, xxvii, 75.
OATMAN: *Amer. Journ. of the Med. Sciences*, 1911, cxlii, 225.
SPÉLMAYER: *Neurologisches Centralblatt*, 1900, 8, 51.

PROGRESSIVE LENTICULAR DEGENERATION.

INTRODUCTION.—This disease, first described by Gowers under the title "tetanoid chorea," has recently been fully investigated by Kinler Wilson, and he has suggested the name "progressive lenticular degeneration." The disease has also been described by Anton under the name "dementia choreo-athetica."

Wilson defines the disease as one which occurs in young people, is often familial, but not congenital or hereditary.

It is essentially and chiefly a disease of the extrapyramidal motor system, and is characterized by involuntary movements, usually of the nature of tremor,



FIG. 177. — PROGRESSIVE LENTICULAR DEGENERATION: PHOTOGRAPH OF BOY AGED TEN, SHOWING THE SPASTICITY, CONTRACTIONS, AND OPEN MOUTH—CHARACTERISTIC FEATURES OF THIS DISEASE.

(From photograph kindly lent by Dr. Wilson.)

dysarthria, dysphagia, muscular weakness, spasticity and contractures, with progressive emaciation. With these may be associated emotionalism and certain symptoms of a mental nature. The disease is progressive, and after a longer or shorter period fatal.

Pathologically it is characterized predominantly by bilateral degeneration of the lenticular nucleus, and in addition cirrhosis of the liver is constantly found, the latter morbid condition rarely, if ever, giving rise to symptoms during the life of the patient.

ETIOLOGY.—The youngest recorded case is that in a boy of ten years. The disease commonly commences during the second decade of life. In one family of eleven children three were affected, and in another family of fourteen three were affected.

There is no evidence that syphilis is an ætiological factor. The exciting causes of the disease are unknown.

SYMPTOMATOLOGY.—A child who has been perfectly normal in mental and physical development is noticed to have involuntary movements and tremor increased by excitement and by voluntary effort. In addition to the tremor, a progressively increasing rigidity of the limbs and trunk takes place. This also affects the muscles of the face, and gives rise to a fixed and furious expression, in which the mouth is held widely open and the patient laughs in a silly manner.



FIG. 128.—PROGRESSIVE LENTICULAR DEGENERATION: CORONAL SECTION THROUGH HEMISPHERES, SHOWING BILATERAL AND SYMMETRICAL CAVITATION OF THE LENTICULAR NUCLEI.

(From a photograph kindly lent by Dr. Wilson.)

The rigidity of the limbs is followed by contraction, so that the patient lies in bed unable to move, and it is impossible passively to straighten the limbs. Difficulty in articulation and difficulty in swallowing is a constant feature.

The muscular power is often strikingly good, in spite of the wasted condition of the muscles; and though the patient is helpless, the muscles cannot be said to be paralyzed. There is no alteration in sensation.

The reflexes are as a rule normal; the knee-jerks are present and active; there is no ankle clonus. The plantar responses are flexor, and the abdominal reflex present.

The mental condition of the patients is changed. They are restless, laugh easily, and are unable to settle to anything. On the other hand, the memory remains good, and on the receptive side but little defect is observable.

The vision is good, there is no nystagmus, and the optic discs are normal.

The disease slowly progresses till death takes place from emaciation.

The cirrhosis of the liver gives rise to no symptoms, but the temperature is often raised and irregular (*vide p. 216*).

PATHOLOGY.—The characteristic change which is found in the brains of these cases is bilateral symmetrical degeneration of the lenticular nucleus, the putamen being more affected than the globus pallidus. Neighbouring structures, such as the caudate nucleus, may be shrunken, but never disintegrated, as is the lenticular nucleus.

The microscopic changes consist in a glia overgrowth, followed by disintegra-



FIG. 129.—MOOREHEAD'S LENTICULAR DEGENERATION: PHOTOGRAPH OF LIVER SHOWING THE NODULAR CHARACTER OF THE CIRRHOSIS, MAINLY OF MULTINODULAR TYPE.

Cirrhosis of the liver is a constant feature in cases of this disease.

(From a photograph kindly lent by Dr. Wilson.)

tion and cavity formation in the lenticular nucleus. This gives the nucleus a finely worm-eaten appearance.

DIAGNOSIS.—Once the clinical picture of this disease is recognized, the features are so distinct that no difficulty in diagnosis should arise.

Cases of bilateral cerebral lesion (diplegia) present somewhat similar symptoms, but in such cases the reflexes show the characteristic changes of a pyramidal affection.

DURATION AND PROGNOSIS.—The disease is always slowly progressive, and may run its whole course in four to six months; but in the more chronic cases two to four years is the usual course of the disease.

REFERENCE.

WILSON - *Brain*, 1911-12, xxvii, 205.

SYPHILIS OF THE CENTRAL NERVOUS SYSTEM.

Introduction.—Congenital syphilis manifests itself in the nervous system of infants and young children in a variety of ways. It will give rise to a meningitis, a hydrocephalus, to an endarteritis, to gummata, to a juvenile tabes, and to a juvenile general paralysis of the insane.

Meningitis and hydrocephalus due to syphilis have already been dealt with in the chapters devoted to these diseases. Endarteritis and gummata produce symptoms dependent on the situation of the lesion. Juvenile tabes and juvenile general paralysis of the insane have a symptom-complex which is distinctive.

Ætiology.—The effects of syphilis in the child are nearly always caused by congenital and not by acquired syphilis, although the latter is an occasional cause of symptoms.

Juvenile Tabes.—This disease is rare. It manifests itself by the same symptoms as those in the adult. Unequal pupils, the failure of them to react to light, and paralysis of one of the ocular muscles, may be the first symptoms, followed by the absence of the knee-jerks and other deep reflexes, unsteadiness of gait, and incontinence of urine. Lightning pains, optic atrophy, and paralysis of the trunk and limbs, may also be present.

The disease tends to run a very prolonged course, and though starting at the age of six to seven, yet progresses slowly, and only gives rise to complete incapacity towards the end of the second decade of life.

Juvenile General Paralysis of the Insane is far more common than juvenile tabes.

The history usually obtained in these cases is that until the age of eight or nine years the child has been normal in its development, both physical and mental, and has not been backward at school. Mott says the mental deficiency may date from birth. The first sign of failure is the loss of the most recently acquired accomplishments; the writing is not so good, he can no longer do the mathematics he used to do, and he becomes dull and listless. Speech becomes affected; the child blurs its words. A fit may now occur, leaving the child with weakness on one side of the body, which passes off in a few days. The child may then develop tremor of the lip, shaking of the limbs, and an unsteady or spastic gait. During this time the child begins to show a marked tendency to get fat.

On examination an Argyll-Robertson pupil may be found to be present, a hesitating and indistinct articulation, a general feebleness of the limbs, with increased knee-jerks, and other signs of pyramidal affection. Further degeneration gradually takes place, the amount of physical and psychical weakness being rapidly increased by the occurrence of epileptic seizures. Hallucinations and delusions are rare in juvenile general paralysis of the insane. A condition of progressive dementia is common. As the disease advances, the child begins to waste, the legs become rigid and flexed, and there is complete incontinence of urine and feces.

Such a condition may develop in three to four years from the initial symptoms. Death during one of the congestive attacks may shorten the duration of the disease; but its progress is slow, and it is extraordinary for what length of time the child will live in a demented and enfeebled condition. Mott states that, of these cases

he has observed, 2 lasted only three months, 2 eighteen months, 1 one year, and 1 seven years; but he also states that when the cases begin young they last longer.

PATHOLOGY.—In some of these cases there is a very marked thickening of the pia arachnoid membrane, in others but little affection of the membrane; the convolutions of the brain are always atrophic, the sulci wide, the ventricles dilated, and the *ependyma* granular. The brain is wasted as a whole. On microscopical examination the fibres of the cortex have to a large extent disappeared, and there is almost complete absence of the tangential fibres of the cortex. The cells of the cortex show various stages of chromatolysis; many have already disappeared or been replaced by calcareous deposits. The pyramidal tracts, and in some cases the posterior columns of the cord, show degeneration.

DIAGNOSIS.—The diagnosis of the condition is not difficult. The slow progress of symptoms, the mental and physical degeneration, the Argyll-Robertson pupils, have little doubt as to diagnosis, even if other signs of congenital syphilis—pugged teeth, interstitial keratitis, and nodes on the tibia—are absent.

The examination of the cerebro-spinal fluid reveals a lymphocytosis, and the examination of the blood and the cerebro-spinal fluid gives a positive Wassermann reaction.

PROGNOSIS.—The disease is slowly and steadily progressive. Various methods of treatment have been tried, but nothing has been found to arrest the progress of the disease.

TREATMENT.—Treatment with mercury and large doses of iodide may be tried, and injections of salvarsan; but no better results are to be expected than those obtained by these remedies in the parasyphilitic affection of the adult.

REFERENCE.

Morr: *Archives of Neurology*, 1898, i, 336.

DISSEMINATED SCLEROSIS.

This disease, which is one of the most common affections of the nervous system in the young adult, is practically unknown in childhood. There are in the literature cases recorded as disseminated sclerosis in children, but no case of the disease with a convincing pathological record has been found, nor has an undoubted case of disseminated sclerosis in a child under the age of twelve years come under the observation of the writer.

There are many cases of intention tremor in children, and some of them may be associated with nystagmus and signs of degeneration of the pyramidal tract, but causes other than disseminated sclerosis account for these symptoms.

In some cases of disseminated sclerosis occurring in early adult life, there is a history of transient squint or some failure of vision occurring during childhood, and these may be brought forward as evidence of the occurrence of the disease during childhood.

MYASTHENIA GRAVIS.

This disease is characterized by weakness and easy exhaustion of the muscles of phonation, mastication, deglutition, and also of those of the eye and trunk.

The disease is certainly most rare in children under the age of twelve, although there are a few such on record. Mailhouse records its occurrence in a boy aged two years and three-quarters, and Renault in a child of twelve—senile with a post-mortem. Buzzard has recorded a case of a girl, aged fourteen, in whom the symptoms first manifested themselves at the age of six years. The weakness was most marked in the pelvic muscles, the face muscles being unaffected.

Only one instance of this affection in a child has come under the personal observation of the writer, and that was a child of twelve who for four years presented symptoms of bulbar paralysis, ocular paralysis, respiratory difficulty, and general asthenia. Whilst at rest the child's condition greatly improved, but from time to time the respiratory difficulty became greater. She died at the age of twelve, having been four years under observation. At the autopsy, and after most careful examination of the nervous system, no lesion could be discovered.

MORBID ANATOMY.—The only change which has been found on microscopical examination is the presence of groups of small round cells in the muscles. These have been designated by Buzzard as "lymphorrhages." They are not, however, peculiar to myasthenia gravis, and have been found under other pathological conditions. The enlargement of the thymus has been described in some cases.

DIAGNOSIS.—The diagnosis is not as a rule difficult, but in those cases in which the weakness is limited to the pelvic muscles, and is associated with atrophy, it may be difficult to distinguish the condition from a myopathy. In the case recorded by Buzzard, above referred to, this difficulty arose, and it was only after the child had been under careful observation, and a definite attack of weakness had been observed, that the diagnosis of myasthenia was made certain.

PROGNOSIS.—Some cases run a most prolonged course, and with care the patient may live for many years. In other cases the disease takes an acute course, and the patient dies of respiratory failure, consciousness being perfectly preserved till the end.

TREATMENT.—No treatment has been found for this affection. Some patients have been benefited with thyroid, others have been made worse. Various glandular extracts have been used, but not with any constant success.

REFERENCES.

Buzzard: *Brit. Med. J.*, 1905, xxvii, 428.

Buzzard: *Boy. Soc. Med., Surg. Sec.*, 1912, v, 128.

HEMIATROPHY OF THE FACE.

Hemiatrophy of the face is a rare affection in childhood; it is characterized by progressive wasting of the subcutaneous tissue, bones, skin, and muscles on one or both sides of the face. There is no weakness of the muscles of the face, and they react normally to electrical stimulation. There is no alteration in the sensation. The wasting affects primarily the fat and subcutaneous tissue, and the absence of these give to the child the appearance of an aged person.

FAMILY PERIODIC PARALYSIS.

INTRODUCTION.—A rare disease occurring in families, and affecting members in several generations. It is characterized by periodic attacks of loss of power, amounting to complete flaccid paralysis, with loss of reflexes and loss of the electrical excitability of the muscles, but without any loss of sensation or loss of consciousness. The attacks last for twelve to forty-eight hours, affect the proximal muscles before the distal, and pass off in the reverse order, so that the distal muscles recover before the proximal.

ETIOLOGY.—The condition is a familial affection. An attack can be produced by exertion or overexertion and by excesses in diet, but the causal agent is not known.

Both sexes are equally liable to be affected; the youngest known case was in a child of two years.

SYMPTOMATOLOGY.—The attack often begins at night; the child has gone to bed well, but wakes up in a condition of more or less complete paralysis of the limbs. The paralysis gradually extends until there is a complete flaccid paralysis of all four limbs, paralysis of the abdominal muscles, and sometimes of the intercostals. In some cases there may be weakness of the levator palpebrarum, but as a rule the cranial nerves are not affected. The child is quite conscious. There may be profuse sweating and dilatation of the heart. All the deep and superficial reflexes are absent, and the muscle irritability is lost, so that no response can be obtained either on percussion or on electrical stimulation.

After the attack has lasted for some hours, the paralysis begins to pass off, the reflexes return, and recovery is complete. Subsequent attacks may be of the same nature, but may vary in severity.

PATHOLOGY.—It is argued that since the muscles entirely lose their electrical excitability, the symptoms must be due to some toxin acting on the muscles. No change has been found in the nervous system, and very little in the portions of excised muscle.

DIAGNOSIS.—In the first attack some difficulty might arise in distinguishing the condition from an ascending poliomyelitis or a Landry's paralysis, but no such difficulty should be experienced if there was a history of former attacks.

Again, although there might be complete flaccid paralysis of all the limbs, in the above two conditions the complete loss of electrical excitability would not be present.

PROGNOSIS.—The prognosis is usually good, but death has occurred in some cases from involvement of the respiratory centre.

TREATMENT.—The attacks can probably be to some extent prevented by avoiding overexertion and certain articles of diet. During an attack the patient should be kept completely at rest, and any symptoms of cardiac or respiratory failure treated in the usual manner. Attention must be paid to the bladder.

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BIRCHALL, E. F.: *Lancet*, 1901, ii, 1595.

FAMILIAL AND HEREDITARY ATAXIA.

INTRODUCTION.—In dealing with the diseases which are included under the above heading, the classification suggested by Gordon Holmes has been adopted. The cases are divided into the following groups:

1. Friedrich's disease—a combined system degeneration of the spinal cord.
2. Spino-cerebellar ataxia, dependent on a primary degeneration of the spinocerebellar tracts.
3. Cerebellar ataxia, in which the morbid change is limited to the cerebellum and its immediate connections.

The clinical features common to all these groups are inco-ordination of voluntary movements and difficulty in maintaining equilibrium.

The disease tends to affect many members of a family, and attacks males and females alike. The transmission occurs through the affected members.

ETIOLOGY.—The cause of the disease is unknown. It is regarded as a primary degeneration of certain tracts of the nervous system. This tendency to degeneration has been described by Gowers under the title "abiotrophy."

Group 1: Friedrich's Disease.—**SYMPTOMS.**—The early symptoms usually appear during the first decade of life. The child may have been backward in learning to walk, and in some cases may never have walked properly. Gradually the walking becomes worse, the child staggers along and seems always in danger of falling, but, curiously, seldom falls. If he attempts to grasp an object, the ataxia of the hands at once becomes obvious.

The head and trunk are in almost constant movement, and if whilst the child is standing the muscles be examined, it will be found that their degree of contraction is always varying.

When the child speaks, he does so in a jerky manner, and his articulation is often defective.

The eyes may show a spontaneous nystagmus, but frequently this is only to be observed by making the child bring the eyes into a position of lateral deviation. The pupils commonly react to light. Ocular paralysis may be present, and rarely optic atrophy with complete blindness. Mental defect may be present. Some observers consider that it is frequent. The manner in which the child talks, and the slowness with which it performs this action, give an appearance of mental defect which is more apparent than real.

The physical examination of these patients reveals a very definite set of signs. Besides the nystagmus already mentioned, there is lateral curvature of the spine, pes cavus, and sometimes equinus, absent knee-jerks, and extensor plantar response. The sphincters are rarely affected, and no alteration can be detected in sensation. In some cases, however, the knee-jerks are increased, and ankle clonus may be present. Lightning pains have been described, but are very rare.

The disease is slowly progressive, and it may take several years to become fully developed. In the later stages wasting of muscles occurs, and the deformity of the back and feet becomes very marked.

MORPHO ANATOMY.—The most striking feature on post-mortem examination in a case of Friedrich's disease is the relative smallness of the spinal cord.

The pathological change which is present in these cases is degeneration of the

posterior column, degeneration of the pyramidal tracts, and also of the spino-cerebellar tracts. In most cases no change can be found in the cerebellum, although exceptionally some change has been described.

The degeneration of the pyramidal tracts is greater in the lower than in the higher segment of the spinal cord, and is not apparent above the medulla oblongata. The cerebrum is normal in appearance, and such changes as have been described are secondary to other lesions.

Group II. : Spino-Cerebellar Ataxia (Sanger-Brown).—This group of cases is characterized by ataxia of an hereditary nature, which commonly occurs at a later period than the Friedreich's ataxia. It is associated with optic atrophy and increased knee-jerks. In the family described by Sanger-Brown, it was only in the fifth generation that the disease began in a child of eleven years. It is certainly a very rare disease in childhood, and if one accepts the occurrence of optic atrophy and increased knee-jerks as a possible phenomenon in Friedreich's disease, then the separation of this group from that of the Friedreich's type is only possible on a pathological basis.

MORPH. ANATOMY.—The spinal cord is small, there is degeneration in the direct cerebellar tract, and some degeneration has been observed in the posterior column, but it is slight compared to the change found in Friedreich's disease. The cerebellum is relatively small in proportion to the cerebrum.

Group III. : Cerebellar Ataxia.—The characteristic feature of this group is a slowly increasing ataxia, due to a primary degeneration of the cerebellum. The course of such a degeneration is unknown, but it has occurred as a familial disease; just as in family amaurotic idiocy there is slowly progressive degeneration of the cerebrum, so in this group of cases there is slow degeneration of the cerebellum.

SYMPTOMS.—The general symptoms are as follows: A child who has made normal progress during the first few years of life gradually loses the power of writing and using the hands for fine movements, begins to walk unsteadily, and eventually becomes so ataxic that he is unable to walk. The articulation becomes indistinct, slow, and jerky. The general intelligence of the child remains normal. There is no affection of the sphincters. The disease runs a slowly progressive course, death being due to some intercurrent disease. The course which the disease runs is usually a long one.

MORPH. ANATOMY.—The striking feature in these cases is the relative smallness of the cerebellum. This smallness must not, as Holmes points out, be confused with agenesis or developmental defect of the cerebellum, in which there may be either no symptoms or ataxia from the time of birth. Neither must it be confused with vascular or interstitial lesion of the cerebellum. It is a degeneration of the cortex of the cerebellum and the fibres which connect this with the central nuclei. The efferent cerebellar tracts are intact, and there is no change in the spinal cord.

DIAGNOSIS.—The diagnosis of these three groups of ataxia from one another is by no means easy. They must be regarded as types of a form of hereditary and familial disease, between which intermediate cases occur having the features, both clinical and pathological, of two or more types.

This group of cases must be carefully distinguished from those cases of cerebellar ataxia which are due to lesion of the cerebellum of a vascular, congenital, or

intestinal nature, for in the former there is no tendency to recovery, while in the latter there is, is a certain number, a tendency to more or less complete recovery.

TREATMENT.—It may be said there is no treatment for familial and hereditary ataxia. Some improvement may be obtained by re-education movements and correction of deformities by tenotomy, but complete recovery does not take place.

OTHER FORMS OF CEREBELLAR ATAXIA.

Apart from the above-mentioned forms of familial and hereditary ataxia, and those dealt with under the heading of Intracranial Tumours and Acute Ataxia due to Acute Polio-encephalo-myelitis, there are other forms of ataxia dependent on lesions of the cerebellum. The most important group is that of *Cerebellar Ataxia*—that is to say, cases in which ataxia is noted in early life, and in which there is a tendency to gradual improvement, and it may be complete recovery.

The cause of this condition is thought to depend on the defective development of the cerebellum, but the pathological evidence on this point is deficient, owing to the length of time elapsing between the occurrence of the symptoms and the opportunity of a pathological examination.

SYMPTOMS.—The first symptom of this disease may be noted shortly after birth. The child is said "to tremble." Such children do not usually come under observation until a later period of life, when they should have learnt to sit up. If an attempt is made to sit the child up, it wobbles to and fro, and eventually over-balances and falls to one or other side. At the time of life when it should grasp an object, it is noticed that the movements are most inco-ordinate. The child generally learns to sit up and to use the hands, but is late in doing so. On standing there is great unsteadiness of the legs, and on attempting to walk they are thrown about in a most ataxic manner. As the child learns to talk, the words are pronounced in a slow, jerky, monotonous way. These children are usually slow eaters, and though they swallow liquids well, they swallow solids slowly.

They are usually clean in their habits, intelligent, well nourished, and have good muscular power. As they grow the ataxia of the limbs becomes less, and in some cases normal development takes place; but this stage of development is not reached till a period long after that of an ordinary child. The child may be six or eight years old before he is able to walk normally. On physical examination the knee-jerks and ankle-jerks are present, and may be active. There is no rigidity of the limbs, but rather loss of tone, and yet the legs are well developed and unusually strong. The abdominal reflexes are present, and the plantar responses are generally flexor. Nystagmus may be present, but is a rare rather than a common manifestation.

In connection with this form of ataxia, which would seem to depend solely upon a cerebellar defect, must be considered an ataxia which is associated with a cerebral defect. The presence of the cerebral lesion in addition to the cerebellar lesion influences the capacity of these children to recover. It has been shown experimentally in animals by Luciani that the symptoms produced by ablation of one lobe of the cerebellum are rapidly recovered from so long as the cerebral cortex is undamaged; but if the cerebral cortex be injured, then the symptoms produced by the removal of the cerebellum persist.

This experimental evidence would seem to explain the well-known clinical fact that there may be atrophy, and even complete absence, of the cerebellum without any symptoms pointing to cerebellar disease being present, and also the clinical fact that in many cases the ataxia persists throughout life, since the damaged cerebral hemispheres are unable to assume the co-ordinating functions of the cerebellum.

This disease may occur in more than one member of a family. In animals, a whole litter of puppies or kittens may be affected.



FIG. 120.—CEREBELLAR ATROPHY: PHOTOGRAPH OF BRAIN OF CHILD TEN MONTHS OLD, SHOWING CONGENITAL ATROPHY OF THE CEREBELLUM—A CONDITION FOUND IN MANY CASES OF CONGENITAL AND PROGRESSIVE ATAXIA, WHETHER OCCURRING IN FAMILIES OR SPORADICALLY.

Compare with normal cerebellum, Fig. 109.

DIAGNOSIS.—The diagnosis of this group of cases from ataxia due to hereditary and familial disease, from tubercle and lesions of the cerebellum and mid-brain, and from diphtheritic paralysis, is of the greatest importance, not only as regards prognosis, but also with regard to treatment; for in the cases under consideration recovery tends to take place, whereas in the others (except diphtheritic paralysis) recovery is unlikely. A careful consideration of the symptoms, their onset, and the course of the disease, should make the diagnosis clear.

If in association with the ataxia there is a condition of rigidity of the limbs,

a mental defect, increased knee-jerks, ankle clonus, and extensor plantar response, signs suggestive of a cerebral defect, a favourable prognosis cannot be given.

PROGNOSIS.—The outlook in uncomplicated cases of congenital cerebellar ataxia is good, but care must be exercised in arriving at the diagnosis "congenital cerebellar ataxia."

TREATMENT.—The education of the movements of the hands and legs is of the greatest importance. A graduated series of exercises must be given. The child should be encouraged to walk with a walking machine.

The piling of little cubes one on top of another is a simple exercise for the hands, and numerous others can be suggested to amuse and educate the child.

Acute Ataxia.—It is probable that most cases of "acute ataxia" are due to a vascular or inflammatory lesion. Those cases occurring in association with poli-encephalo-myelitis have already been dealt with. There are, however, cases in which "acute ataxia" has developed after measles, whooping-cough, or one of the acute specific fevers, or may be due to hemorrhage or convulsions.

The symptom of ataxia may follow on general convulsions, and in such cases the ataxia is only noticed after the child is convalescent from the acute illness, and begins to use its hands or attempts to sit up. There is then marked inco-ordination of the hands, and a wild ataxic gait. The child recovers its general health, but it may take several months for it to recover completely from the ataxia.

DIAGNOSIS.—The occurrence of ataxia in childhood is a condition which may give rise to difficulty both with regard to the cause of affection and also to prognosis.

The ataxia may be due to one of the well-recognized forms above alluded to: hereditary and familial ataxia, to tumour and lesions of the cerebellum and mid-brain, to diphtheritic paralysis, to poli-encephalo-myelitis, to vascular lesion, and to congenital defects.

It is well known that some of the above forms of ataxia run a progressive course; in others complete recovery takes place. In some cases it is only by a careful consideration of the history and watching the course of the case that an opinion can be arrived at as to the nature and course the case will pursue.

The following case illustrates the difficulty:

A male child, aged two years, the first of healthy parents, had made normal progress with walking and talking. The child then gradually became ataxic in the lower limbs, lost the power of walking, gradually lost the power of sitting up, and if sat up oscillated from side to side until he fell over. The arm movements also became ataxic. The general health was not seriously affected. The disease slowly advanced for three months, and then the symptoms began again to slowly pass off, and the child made a good recovery.

The slow onset of the ataxia led to the giving of an unfavourable prognosis, which was not borne out by the course of the disease.

REFERENCES.

- BAYNES: *Ibid.*, 1905, xxviii, 484.
CLARK: *Ibid.*, 1902, xiv, 318.
HOLMES: *Ibid.*, 1907, xxx, 466.
HOLMES: *Albert and Holliston's System of Medicine*, 1900, vii, 779.
WARRISONS: *Ibid.*, 1902, xxv, 444.

PARAPLEGIA.

The term "paraplegia" is used to denote a paralysis of the lower extremities which is usually spastic in character. The term is loosely used, and cases due to widely different causes are classified under this heading.

Ætiology.—The commonest cause in children is undoubtedly spinal caries in the cervical or dorsal region, but injury, tumours of the spine, hæmorrhage, family spastic paraplegia, various forms of cerebral degeneration, tumours of the pons, and even poliomyelitis, may give rise to a spastic paraplegia. All these conditions are considered under their respective headings. Some of those above-mentioned give rise to pressure on the cord, and are often alluded to as cases of "compression paraplegia," in order to distinguish them from such conditions as family spastic paraplegia, in which the condition is due to a degenerative condition of the pyramidal tracts.

The symptoms of paraplegia are, loss of or diminished power in the lower extremities, increased tone giving rise to a spastic condition of the limbs, increased knee-jerk, ankle clonus, and extensor plantar responses. In addition to these signs there may be loss of sensation to a given level, and loss of control over the sphincters.

Diagnosis.—The diagnosis of paraplegia is easily made when the above symptoms and signs are present; but such a diagnosis is insufficient, and it is of the greatest importance to ascertain the cause of the paraplegia, for the line of treatment differs entirely according to the cause. In spinal caries rest is essential, in spinal tumour operation is indicated, whilst in family spastic paraplegia exercises are of importance.

The diagnosis of the conditions which give rise to paraplegia are discussed in detail in the sections dealing with the particular disease which causes this symptom (*cf.* Nervous Manifestations due to Spinal Caries, Spinal Tumour, etc.).

SPINAL TUMOURS.

Introduction.—Spinal growths, apart from those of tuberculous nature, are most rare in childhood, and when symptoms suggesting the probability of such a lesion occur it is commonly found that a more or less diffuse growth is present.

Symptomatology.—The symptoms which such a growth give rise to are pain, loss of power in the limbs, loss of control over the sphincters, and loss of sensation up to the level of the lesion. In some cases the Brown-Séquard syndrome may be present—i.e., loss of power on the one side, loss of sensation, especially to heat, cold, and pain, on the opposite side. The reflexes show the characteristic alterations found in compression paraplegia—viz., increased knee-jerks, ankle clonus, extensor response. The knee-jerks only become lost when the pressure on the cord has increased to such an extent as to abolish its conductivity.

The examination of the cerebro-spinal fluid may furnish evidence as to the presence and nature of the growth.

Pathology.—The growth may by direct pressure give rise to alteration in the vascular supply, and thus lead to softening of the spinal cord; or the growth

may invade the substance of the cord. Sarcoma is the only form of spinal growth which has been observed by the writer within the spinal canal of children.

DIAGNOSIS.—The diagnosis of compression paraplegia is simple; but the diagnosis of a new growth as the cause of the symptoms is most difficult, and in many cases impossible without an exploratory operation. The examination of the cerebro-spinal fluid may furnish valuable evidence, if cells of new growth can be found; but a negative result leaves the diagnosis still doubtful. An X-ray will in some cases aid the diagnosis by demonstrating the presence of caries in the spine; and since spinal caries is by far the most common cause of such a paraplegia in a child, the possibility of a spinal growth should not be considered until every



FIG. 133.—SARCOMA OF THE SPINAL CORD OF A CHILD.

The growth gave rise to symptoms which were localized to the mid-dorsal region, but at the time of operation the growth was found to be too extensive for removal.

means of examination for ascertaining the presence of a spinal caries has been adopted, and has failed to demonstrate that disease.

PROGNOSIS.—The outlook in cases of spinal tumour in childhood is by no means hopeful. If the spinal tumour is circumscribed it may be possible to remove it, but in most cases the growth is diffuse.

TREATMENT.—If the diagnosis of a spinal tumour can be made, the right line of treatment is undoubtedly to attempt its removal. On the other hand, paraplegia in a child is so much more commonly due to spinal caries that no case should be operated on until every means has been adopted to exclude the possibility of that disease. Most observers will agree that, with but few exceptions, a laminectomy in a child suffering from paraplegia due to spinal caries is a most undesirable operation, and one if possible to be avoided.

FAMILY SPASTIC PARALYSIS ASSOCIATED WITH AMYOTROPHY.

INTRODUCTION.—An unusual form of progressive muscular atrophy affecting several members of a family has been described by Gee and Ormerod. The disease begins in the earlier years of life, and is characterized by wasting of the small muscles of the hand and weakness of the legs; there is in some cases a history that the child has never walked well. The disease affects males and females alike, and is transmitted from parent to child.

SYMPTOMATOLOGY.—The clinical features of these cases are spasticity of the lower limbs, with atrophy of the small hand muscles. The weakness of the hand may first be noticed at the time when the child should begin to write. The disease runs a slowly progressive course. The intelligence is usually good. In some cases nystagmus has been present.

PATHOLOGY.—These cases have been shown by Mass to be due to atrophy of the cells of the anterior horns and degeneration of the pyramidal tracts. They are, however, closely related to the cases of family spastic paraplegia, and also probably bear some relation to the cases of familial and hereditary ataxia; for nystagmus has been present, and the small size of the cerebellum was a striking feature in the case reported by Mass.

DIAGNOSIS.—The clinical features of this disease—viz., wasting of the small muscles of the hand and a spastic gait in a child—suggest the possibility of pressure on the cervical region of the cord. The common cause of such pressure would be spinal cancer. Having excluded that disease as a cause, there are but few other conditions which will give rise to this unusual symptom-complex, especially when the slowly progressive nature of the complaint is borne in mind.

In its typical form it is probably easy to differentiate from the other forms of familial and hereditary diseases, but in its aberrant form it presents some of the features of the familial and hereditary ataxia, and is also allied to the familial spastic paraplegia.

PROGNOSIS.—The disease is extremely slow in progress. Some cases may be said to be stationary, many years elapsing between the initial symptoms and complete incapacity.

TREATMENT.—The prevention of deformity by the adjustment of suitable splints, and a careful attention to the general nutrition of the child, are probably the best methods of preventing its advance.

REFERENCES.

- HELANDER: *Rev. of Neurol. and Psych.*, 1905, iii, 256.
MASS: *Deutsche Ztsch. f. Nervenzh.*, 1911, xii, 757.
HUSTON: *Ibid.*, 1899, ix, 1.

FAMILY AND HEREDITARY SPASTIC PARAPLEGIA.

The characteristic feature of this condition is a slowly progressive loss of power of the lower extremities, accompanied by rigidity, exaggeration of the deep reflexes, ankle clonus, and extensor plantar responses, without any affection of

sensation, incontinence of urine, or muscular atrophy. The disease may affect several members of a childhood, and pass from parent to child.

ETIOLOGY.—It may commence at almost any age. Cases occur in children, and it is probable that the "law of anticipation" holds good, in that the children of an affected parent become affected at an earlier age than the parent. Males and females are alike affected. The direct cause of the disease is unknown.

There is no evidence that syphilis plays any part. The acute specific form would seem, however, to be sometimes a determining factor in its onset.

SYMPTOMATOLOGY.—The symptoms of the typical form of the disease are few, and consist in difficulty in walking owing to a spastic paraplegia, without loss of sensation and without affection of the bladder. The knee-jerks and ankle-jerks are increased, ankle clonus is present, and the plantar gives an extensor response. The abdominal muscles may also be spastic.

To this simple clinical picture numerous other symptoms may in aberrant cases be added. Tremors of the arms and trunk and nystagmus have been described in some cases.

Only the simplest form of the disease will be dealt with here; for if the unusual manifestations are included, the clinical picture changes, and becomes that of the aberrant type of Friedrich's disease, or of familial and hereditary ataxia.

PATHOLOGY.—The pathological change which has been found in these cases is a degeneration of the crossed pyramidal tracts of the spinal cord, and this degeneration is not apparent above the medulla. In some cases there is in addition some degeneration of the posterior columns, and especially of Goll's column.

DIAGNOSIS.—The diagnosis depends upon the existence of a spastic paraplegia in more than one member of a family.

The occurrence of spastic paraplegia in a single member of a family may be due to a variety of conditions which are discussed under the heading *Cerebral Diplegia*.

PROGNOSIS.—The disease is but slowly progressive. Even when starting in early childhood, it does not necessarily give rise to complete incapacity for ten to twenty years.

TREATMENT.—Cases have been recorded in which with suitable exercises considerable improvement has taken place; but the general tendency of the disease is to progress, and in most cases treatment is of little service.

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SEILLER: *Philadelphia Med. Jour.*, 1902, ix, 1129.

NEURITIC TYPE OF PROGRESSIVE MUSCULAR ATROPHY

(PERONEAL TYPE—CHARCOT, MARIE, TOOTH).

INTRODUCTION.—This form of progressive muscular atrophy lies midway between the myopathic and the myelopathic affections. In its clinical aspects it is closely allied to the myopathies, but anatomically a definite change in the spinal

cord has been established. The characteristic features of this affection are a slow, progressive muscular atrophy, with weakness, starting usually in the peroneal and anterior tibial muscles, spreading gradually to the other muscles, so that all those below the knee are wasted, whereas the thighs remain in moderately good condition. Following the atrophy of the legs there is wasting of the small muscles of the hands and forearms. There may be some alteration of sensation over the periphery of the legs, and vasomotor disturbances are common.

Ætiology.—*Age.*—The disease usually commences during the first decade, but may appear in the second or third decade of life.

Heredity.—It is transmitted both by males and females, and both sexes are attacked. But in the family reported by Herringham males only were affected, although the disease was transmitted by the females, following closely the hereditary features noticed in the transmission of colour-blindness. In a considerable number of cases no evidence of heredity can be obtained. In some instances the onset has directly followed measles (Ormerod, Batten).

Symptomatology.—The disease starts in the lower extremity, with weakness and wasting of the peronei, which give rise to foot-drop and inversion of the foot, so that the child walks on the outer side of it. The atrophy progresses, and involves all the muscles below the knee, but the patient is usually well able to get about, as the muscles of the thigh remain in good condition. The distribution of the atrophy gives the leg a curious bottle-shaped appearance, the lower portion of the leg representing the neck of the bottle.

Charcot pointed out that it is the distal portion of the muscle which is first affected. Fibrillar tremors have been observed. In association with the atrophy of the muscles there is some loss of tactile sensation, and loss of muscular sense has also been observed. The muscles and nerve trunks are not tender. Vasomotor disturbances are sometimes present, generally in the form of cyanosis, but sometimes there is a marble-white appearance of the extremities. The muscles lose the power of contracting to mechanical stimuli, and there is a diminished reaction to faradism and galvanism, but a true reaction of degeneration is seldom observed. The condition is nearly always symmetrical, although exceptions to this rule occur (Herringham, Ormerod).

The ankle-jerks are usually lost at an early period, and it is difficult to obtain a plantar response. The knee-jerks remain active, and there is very little tendency for them to disappear, as the vastus internus muscle remains in good condition. The sphincters and trunk muscles are not affected. After the atrophy of the legs has been present from one to four years, wasting of the small muscles of the hands is noticed. This gives rise to a typical claw-hand, the two terminal phalanges being flexed. The disease may affect the extensors and flexors of the wrist. The atrophy, as in the legs, falls on the most distal muscles and the distal portion of the muscles. The degree of incapacity to which this affection gives rise varies very greatly. Some patients are able to follow their usual occupation for years. Walking is their main difficulty, and this they surmount by riding a bicycle, which they are able to do perfectly well. The mental condition is good.

Pathology.—There is some confusion with regard to the pathology, owing to the fact that several different conditions have been included under the neuritic muscular atrophy of Charcot, Marie, and Tooth. Sainton carefully reviewed the subject, and excluded many of the doubtful cases. In the case recorded by him

the following changes were found: The cord showed sclerosis of the posterior columns, especially of the columns of Burdach; slight degeneration of both pyramidal tracts; alteration of the cells of Clarke's column; and atrophy of the cells of the anterior horns. There was slight degeneration of the intramuscular nerves, slight sclerosis of the nerves of the forearm and leg, this being well marked in the peroneal nerves only. The larger nerves, such as the sciatic, were normal. The muscular fibres were atrophied, and in some places had completely disappeared, connective tissue being substituted. Almost the same changes were found in

Siemering's case, the degeneration being most advanced in the postero-median column and in the lumbar and lower dorsal regions of the cord. The change in the muscles was that of simple atrophy of the muscle fibres, and not such as is met with in the myopathies.

DIAGNOSIS.—The diagnosis of a characteristic case is not difficult. The slow onset, the character of the weakness and wasting, the absence of pain, and the symmetry of the affection, are the points on which most stress would be placed.

Acute poliomyelitis rarely produces a muscular atrophy so symmetrical as that seen in this disease, and, further, it is not progressive. Multiple neuritis might be confused with it, but the presence of pain and tenderness in the muscles, the more general affection of the muscles, and the greater sensory disturbances, would distinguish ordinary neuritis from peroneal muscular atrophy.

To a progressive muscular atrophy of the motor neurons the disease bears great resemblance, but it differs in its course and



FIG. 132.—PHOTOGRAPH OF A BOY, AGED SEVEN, SUFFERING FROM THE PERONEAL TYPE OF MUSCULAR ATROPHY.

Note the wasting of the legs, below the knee, the feet-drops and inversion of the feet, and also the wasting of the small muscles of the hands.

age and mode of onset from the common type of that affection, which starts in the hands, and produces a spastic condition of the lower limbs. The disease, however, bears a close resemblance to a condition of progressive muscular atrophy seen in several members of a family described by Gee and Omerod. Some of these cases have recently been investigated pathologically, and have been shown to be cases of motor neuron degeneration (Mason) [see p. 861].

The muscular dystrophies, and especially the "distal" type, bear a very strong

resemblance to peroneal muscular atrophy, and, as Spiller has pointed out, it is sometimes impossible to arrive at a correct diagnosis from clinical examination alone.

PROGNOSIS.—Though progressive, the disease advances so slowly that many patients are able to pursue their ordinary work for years. The acute specific foci accentuate the disease, and sometimes call forth its first manifestation.

TREATMENT.—No treatment is known to have any effect in arresting the disease. Mechanical support for the foot-drop will often assist the patients very considerably in walking.

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NERVOUS MANIFESTATIONS DUE TO SPINAL CARIES.

INTRODUCTION.—This section is intended to deal with a class of case in which paralytic symptoms are directly or indirectly due to spinal caries. Most of these cases could undoubtedly be included in a section on compression paraplegia, but in association with these are cases in which the tuberculous process has invaded the spinal cord, and, either by direct destruction or by a spreading myelitis, has given rise to symptoms not easily explicable as due to simple compression.

SYMPTOMATOLOGY.—Spinal caries commonly occurs in the mid-dorsal region, and the manifestations of pressure on the cord in this region are weakness of the lower limbs, spastic gait, increased knee-jerks, ankle clonus, and extensive plantar response. As the pressure becomes more severe, there is loss of sensation corresponding to the seat of the lesion, active incontinence of urine, loss of control over loose motions, loss of the abdominal reflex, and loss of power in the rectus abdominis below the level of the lesion. This weakness of the rectus is shown by the elevation of the umbilicus if the child attempts to raise itself into a sitting position. As the pressure on the spinal cord increases, the spastic condition of the legs is replaced by a flaccid paralysis, the knee and ankle jerks become diminished, and eventually lost, there is overflow incontinence of urine and faeces, and complete loss of all forms of sensation below the level of the lesion.

It is seldom that the third stage is ever reached, for means are adopted to prevent this severe and destructive pressure on the cord.

When the caries is situated in the lower dorsal or lumbar region the pressure comes directly on the lumbosacral enlargement of the cord, and produces wasting of the muscles of the legs, loss of knee-jerks, and an area of anaesthesia of the skin which corresponds to a segmental or root distribution. A tuberculous mass situated within the cord at this region will give rise to similar symptoms, and in such cases the paralysis of the bladder is usually severe.

It is also in cases in which the lesion is situated in the lower dorsal or lumbar region of the spine that the child exhibits the greatest difficulty in rising from the ground into an erect position, and the method of doing so may suggest the diagnosis of pseudo-hypertrophic paralysis. The most careful examination of the back

may in such a case fail to reveal any obvious sign of disease; but such a child will often complain of pain on bending the body forward on the legs, and will often prefer to stand or lie rather than to sit.

When the curv is situated at the level of the lower cervical or upper dorsal region, it is the weakness and wasting of the muscles of the hand which may first attract attention. The hand assumes the claw position, owing to the weakness of the interossei. These and the thoracic muscles show marked atrophy, owing to pressure on the eighth cervical and first dorsal roots.

As the pressure increases, weakness of the legs, spastic paraplegia, increased knee-jerk, and incontinence of urine, are the symptoms which develop. The effects of pressure on the fibres of the sympathetic as they pass out by the first thoracic root are also seen in cases with lesions at this level.

When the curv is situated in the upper cervical vertebrae, the signs exhibited, apart from rigidity of the neck, are weakness, rigidity, transience, and incoordination of the arms and hands. The head is often held in a peculiar attitude, and the child is thought to have a "stiff neck."

With curv in this position, paralysis of the diaphragm on both sides may occur whilst the intercostal muscles act relatively well. It is difficult to see why pressure on the cord should abolish the function of the diaphragm without affecting the intercostal muscles at the same time. The explanation of this unusual manifestation lies in the fact that the tuberculous condition has spread into the spinal cord, and has given rise to softening in the region of the fourth and fifth cervical segments, leaving the conducting paths relatively unaffected.

Curv of the upper cervical vertebrae and of the base of the skull gives rise to unilateral paralysis of the tongue, palate, and vocal cord, and in such a case the earlier blocking of the inferior cerebellar artery by extension of the process may give rise to acute cerebellar symptoms, vomiting, giddiness, dyspnoea, and death from respiratory failure.

NERVOUS MANIFESTATION ASSOCIATED WITH SPINA BIFIDA AND SPINAL MENINGOCELE.

INTRODUCTION.—The nervous symptoms which arise in association with a spina bifida are a partial or complete motor and sensory paralysis of the lower extremities with or without affection of the sphincters. With a spinal meningocele of the upper portion of the spine, weakness of the arms and neck may be present. It is not proposed to discuss the varieties of spina bifida which may be met with, but only the paralytic conditions which are found in association with these defects of development.

SYMPTOMATOLOGY.—In cases of spina bifida of the lumbosacral region the muscles of the feet and legs below the knee are usually most affected, and there is little or no power of movement; whilst the muscles of the thigh and pelvis are relatively intact. The muscles below the knee may be in a flaccid condition and exhibit loss of tone, whilst those above the knee may be rigid and show an increase of tone. The legs are thin and wasted; the knees are commonly flexed, and cannot be either actively or passively extended. The feet are in a position of pes equinus associated with either pes equinus or calcaneus. The great toe is often dorsiflexed. The limbs are commonly cyanosed, and feel cold to the touch. Sensation may be

perfect, but on the other hand there may be partial or complete loss of sensation. Many children seem to feel no pain when the skin is pricked. The sphincters are usually affected, and in those cases where the cone of the cord is involved the sphincter is completely paralyzed, and does not react reflexly. The ankle-jerks are absent, and the knee-jerks also are often diminished or lost. The plantar reflex cannot usually be obtained. On electrical examination no reaction may be obtained either to the faradic or galvanic current, or the muscles may react feebly to faradic stimulation. The skin is usually thin and very liable to trophic sores.

Meningoceles in the upper cervical region may give rise to weakness of the arms, and are commonly associated with defective development of the cerebellum and the posterior column of the cord.

In some cases the paralytic phenomena may be present without any obvious external tumour. These cases are known under the name of "*spina bifida occulta*," and there is in such cases usually a defect in the laminae of the vertebrae, and a defect in the development of the lumbosacral region of the cord, without any protrusion of the membranes of the cord.

DIAGNOSIS.—The diagnosis in those cases in which an obvious swelling is present presents no difficulty; but in certain cases in which there is a paralytic condition of the lower extremities, with wasting and cyanosis, and in which there is a history that the condition has been present from the time of birth, considerable difficulty may arise. Such conditions as defective development, birth injury to the cord, and intra-uterine polyomyelitis, have to be considered as possible causes giving rise to similar symptoms. It is by no means easy to arrive at a certain diagnosis.

PROGNOSIS.—The prognosis as to life in cases of *spina bifida* is bad. Many cases die in the first week of life. Some cases live, and an operation is performed for the removal of the tumour. The removal of the sac is not infrequently followed by hydrocephalus, which is rapidly fatal. In the slighter cases development may take place, and the child may learn to walk.

TREATMENT.—The surgical treatment of the sac of a *spina bifida* does not come within the scope of this work. The paralytic condition is best treated by warmth, massage, passive and active movements, and the application of splints, which keep the leg in a normal position. The celluloid splint is most suitable for this purpose, and should be applied as soon as there is any tendency to the production of deformity. Children who have been neglected learn to walk in the most peculiar attitudes. Some progress on the buttocks, and these cases form one of the groups of "*hutchinson walkers*." The result of getting about in this manner is induration of the skin on the knees and parts which come in contact with the floor, and also of trophic sores which heal with difficulty.

CHAPTER XV

CONGENITAL MENTAL DEFECT (AMENTIA) IN CHILDHOOD

JOHN THOMSON

INTRODUCTION.

THE DIAGNOSIS OF MENTAL DEFECT.

PRIMARY AMENTIA.

SIMPLE PRIMARY AMENTIA.

MICROCEPHALIA.

HYDROCEPH.

TUMORAL SCLEROSIS.

SECONDARY AMENTIA:

EPILEPSY.

AMENTIA DUE TO GROSS CEREbral

LESIONS.

CONGENITAL SYPHILIS.

MORAL IMBECILITY.

GENERAL TREATMENT.

INSTITUTIONAL.

INTRODUCTION AND CLASSIFICATION.

In the following pages the subject of mental defect is dealt with mainly as it affects infants and young children, and it is regarded as far as possible from the points of view of the family doctor and the hospital physician. It is to them rather than to the alienist that parents first bring their children when any of the bodily or mental indications of imbecility are noticed; and it is in their charge that such children are, or ought to be, during the early years of life.

The family doctor is often asked his opinion of the mental condition of little children, so that it is important for him to have such a practical knowledge of the subject as will enable him to discover the early indications of mental defect, and to form a reasonable forecast as to how far the child's intellect is likely to develop. It will also fall to him, in all probability, to guide the parents as to the main lines to be followed in training the child, and to supply from time to time the encouragement necessary to enable them to persevere in sensible treatment in spite of difficulties.

TERMINOLOGY.—The words "idiocy," "imbecility," "feeble-mindedness," "mental defect," and "amentia," do not, of course, denote a disease. Like "diarrhoea" and "paralysis," they only refer to a very striking and common group of symptoms which is evidently present in a series of cases, many of which cannot be named more accurately because their real causation and pathology are still imperfectly understood. The three first mentioned terms are now usually made use of to indicate different degrees of severity of the same kind of intellectual weakness. Their exact meaning has been defined as follows by a Committee of the Royal College of Physicians of London for the Royal Commission on the Care and Control of the Feeble-Minded:

"'Idiot' means a person so deeply defective in mind from birth or from an

early age that he is unable to guard himself from common physical dangers such as prevent us from leaving young children alone.

"Imbecile" means a person who is capable of guarding himself against such common physical dangers, but who is incapable, by reason of mental defect existing from birth or from an early age, of earning his own living.

"Feeble-minded person" means a person who is capable of earning a living under favorable circumstances, but is incapable, from mental defect existing from birth or from an early age, (a) of competing on equal terms with his normal fellows, or (b) of managing himself and his affairs with ordinary prudence, such as the peddler and the fakir.

"Moral imbecile" means a person who displays from an early age, and in spite of careful upbringing, strong, vicious, or criminal propensities on which punishment has little or no effect."

Between idiocy, imbecility, and feeble-mindedness, therefore, the difference is merely one of degree. Between slight feeble-mindedness and mere intellectual dulness or backwardness, however, there are real and important distinctions, although in individual cases it is often difficult, or even impossible at first, to differentiate between the conditions. The word "feeble-mindedness" is used frequently in America, and sometimes in this country also, to include all degrees of mental defect. "Amentia" is a serviceable term which is being increasingly employed in the same general way. It has the advantage that it possesses, as yet, no disagreeable association for the lay mind. It need scarcely be said that a child should never be alluded to in his parents' presence as an "idiot" or "imbecile."

CLASSIFICATION.—The difficulty of making a really satisfactory and scientific arrangement of the numerous varieties of disease, of which mental defect is a prominent feature seems to be insuperable, and it is as easy to criticize effectively all the classifications that have as yet been put forward as it is difficult to frame a better.

Some sort of a classification, however, is necessary, and the most practically useful for clinical purposes seems to be that of Tredgold. He divides all cases of mental defect into two classes, according as they are *primary* (i.e., due to "an inherent incapacity for perfect development owing to defects within the embryonic element") or *secondary* (i.e., due to "the mental development having been arrested by some extraneous or accidental cause"). Among older children and adults the first of these groups includes, according to Tredgold's estimate, about 90 per cent. of all the cases, and the second about 10 per cent. Among children under five years who are recognizable as mentally deficient, the proportions are, for various reasons, rather different from these. Probably about 60 and 40 per cent. respectively would be nearer the mark at this age.

The following further subdivision of these two groups, which is founded on Tredgold's classification, will serve to give some idea of the number and variety of the clinical types of disease which are characterized by mental defect, although it does not include every possible variety:

PRIMARY AMENTIA.—Due to—

"Simple primary" arrest of development of cerebral neurons. (This group probably includes many types not yet differentiated. Many of the cases are characterized by epileptiform convulsions.)

Microcephalus.

PRIMARY AMENTIA (continued).—Due to—

- Mongolism.
- Congenital hydrocephalus.
- Congenital porencephalus.
- Tuberous sclerosis.

SECONDARY AMENTIA.—Due to—

- (A) Epilepsy.
 - Encephalitis and meningitis.
 - Acquired hydrocephalus.
 - Trauma (especially during delivery).
 - Congenital syphilis (including juvenile general paralysis).
 - Infantile cerebral degeneration (amaurotic family idiocy).
- (B) Cretinism.
 - Nutritional defect.
 - Isolation, or sense deprivation.

It is probable that the cases which fall under the Subclass (B) should, if uncomplicated, be ranked as instances of "acquired mental dulness" rather than as varieties of true mental defect.

Complicated cases which seem to belong to more than one of the above types are not uncommonly met with.

THE DIAGNOSIS OF MENTAL DEFECT IN GENERAL.

The recognition of the presence, and of the degree of mental defect in a child must usually depend on what we notice and on facts we ascertain, rather than on any opinions given us by his relatives, although these must not be disregarded. If a child's parents think him less clever or less sensible than others of his own age, this may indeed be of great importance in the diagnosis of a doubtful case; but if they speak highly of his intelligence, their opinion has often to be largely discounted, because most parents are curiously slow to notice and to admit any weakness of this sort in their own children. If, however, we can manage to find out what the patient's companions really think of him, that is always very significant, for even little children are extraordinarily quick to mark and to resent any mental want or peculiarity in their playmates.

In investigating the intelligence of boys and girls of school age, we naturally proceed by questioning their parents as to the usual indications of mental capacity present. We ask how the children behave, and if in nursery and family life they show ordinary common sense and propriety, and get on well with their fellows. We also wish to know if they can be trusted to do an errand, and if they can carry more than one message in their heads at a time. We inquire how they have done such lessons as they have had, and if they can recognize pictures or letters of the alphabet, can count at all, can repeat nursery rhymes, and so on.

Such methods of inquiry, although useful in older cases, take us only a very short way in dealing with infants and little children. In the first year or two of life the directly mental manifestations of amentia are sometimes both few and indefinite, so that we have to trust largely for guidance to bodily symptoms. We

may divide the most important indications of mental defect at this age into four groups.

Certain Abnormalities of Bodily Conformation.—Mental defect is invariably present to some extent in all cases of microcephalus and mongolism, also in severe cases of cretinism, hydrocephalus, and spastic diplegia. The recognition of any one of these conditions, therefore, implies a diagnosis of amentia. In such cases the medical man often recognizes that imbecility is present long before the mother can see anything amiss in the baby's behaviour.

Minor bodily malformations, such as hare-lip, abnormally vaulted or cleft palate, deformed and asymmetrical ears, marked epicanthic folds, epina kila coccia, congenital malformations of the heart and other congenital abnormalities, are in themselves no proof of mental deficiency, for all of them are often met with in children of perfectly normal intellect. They are, however, commoner in children of neurotic families, and, especially when several of them are found together, they may be looked upon as providing some slight confirmatory evidence in doubtful cases. It has been thought that the presence of such stigmata is an imbecile should suggest that the case probably belongs to the "primary" group.

Recurrent Convulsions.—The periodic recurrence of convulsions in an infant without obvious cause, when it continues over a long period of time, generally indicates the presence of mental impairment (*vide infra*). This may be simply the effect of the fits, but much more commonly the mental defect is just another symptom of the brain lesion which is causing them. All kinds of imbeciles are more or less liable to convulsions on slight occasions. In cases of juvenile general paralysis convulsive attacks sometimes occur, and they are apt to be mistaken for epileptic seizures.

Abnormal Gestures.—In many cases the child's want of intellect is at once obvious from his abnormal gestures and actions. In early infancy there may be continuous senseless crying and fretfulness, which is apt to be attributed to all sorts of supposed internal pains and discomforts before the real cause is suspected. Again, we may find airless gaping movements of the jaws, or a constant restless rolling of the eyes, the child never looking straight at anything as a normal baby should do. Constant protrusion of the tongue in a young baby is another suspicious sign, and should suggest mongolism or cretinism. Sometimes there is unmeaning laughter, with exaggerated gestures, wayward and impulsive actions, and in older children dirty and disgusting ways of eating food. Continual dribbling of saliva is a common occurrence in mentally defective children. A very grave indication of arrested intelligence is a state of utter apathy to surrounding circumstances, with absence of the normal reflex muscular movements. In such cases the baby feels like a lifeless thing in his mother's arms when she tosses him up and down, instead of making springing movements with his limbs in his enjoyment of the motion, as a healthy child would do.

Delay in the Development of the Bodily and Mental Powers.—In many mild cases of amentia the first sign that anything is wrong is to be found in the child's being far too long in gaining a proper control over his voluntary muscles and the use of his special senses. Thus, we should always think of mental defect when a baby, without obvious cause, is backward in learning to hold up his head, when a baby, without obvious cause, is backward in learning to hold up his head, to grasp things, to sit, stand, and walk. It is also a particularly bad sign if any infant whose body seems normal does not show the natural craving of the healthy child to be constantly exercising all his growing faculties. It has a similar signifi-

cance when a baby whose eyes and ears are normal shows no interest in bright and moving objects, is backward in responding to the smile, the voice, and the touch of his mother or nurse, and, at a later stage, in beginning to speak. Undue delay in acquiring the usual control over the action of the bladder and bowel is another circumstance which may confirm our suspicions as to the state of the intelligence. Occasionally the earliest abnormality noticed is refusal from birth to take the breast.

Backwardness in the acquisition of normal actions often, of course, depends on the temporary weakness which accompanies and follows exhausting disease. If however, it is present to a marked degree, without any evident debility to account for it, we shall probably be right in suspecting mental deficiency.

It is sometimes no easy matter to distinguish a deaf and dumb idiot who has been badly spoilt from a mentally defective child. The intelligence of his gestures and expression, his obvious interest in his surroundings, his clearly habits, and the ready use of his hands, will, however, usually help us to recognize when nothing worse than deafness is present.

Among older children it is sometimes very difficult after a single examination, as already said, to decide whether we have to do with a mild degree of amnesia, or merely with a backwardness of mental development which is due to some more or less temporary past or present cause.

Acquired Dulness of Mind.—This class of dull children, although not a very large one, is worthy of careful consideration, because they are often taken for cases of amnesia, which is a serious mistake. It is so partly because it leads to the children being wrongly treated, and still more because, when they get well, their recovery is widely quoted to prove that real mental defect may recover completely; and this is a source of much painful disappointment.

Backwardness of mental development may arise from mere lack of teaching and other forms of mental stimulus in an ailing child. We see this often, for example, in weakly cripples. Defects of sight and hearing may have a similar effect. In such cases the mental powers may in time improve indefinitely, if the abnormal conditions can be removed. The most important cases, however, arise from a clinical point of view, are those in which the mental development has been retarded by a prolonged period of malnutrition and profound ill-health. Cases of chronic tuberculosis, recurrent dyspepsia and diarrhoea, severe rickets, and congenital syphilis, are sometimes instances of this. These children are pale and badly nourished, and show very little energy or enterprise. At first sight, individual patients of this kind may closely resemble mentally defective children. We generally find, however, that they differ essentially in disposition, in common sense, in the feeling of responsibility, in correctness of behaviour, and in moral sense.

Another group of children whom I have known mistaken for imbeciles are those whose nervous system has given way completely, and who have lost their brightness, and even all signs of intelligence, for the time being, under the strain of long-continued worry and overwork, along with a lack of sympathy and affection.

In all cases of acquired dulness from ill-health or overstrain, the child is to be regarded strictly as a patient, and not as a scholar. What his brain needs is rest and relaxation—not instruction, however skillful. His mind must be completely fallow while his bodily health is being fostered by a prolonged period of tranquil routine, which should include abundance of fresh air, plenty of sleep, simple amusements, warmth, massage, tonics, and good food. At the same time, if there has been

worry and overstrain, his nerves must be soothed by kindly mothering and encouragement.

The prognosis as to ultimate recovery is often very good, especially where there has been much bodily ill-health to account for the weakness. Cases of nervous breakdown generally do very well under proper treatment, but the future life of a child who has suffered from such a breakdown should be regulated, so as to put as little strain as possible on his nervous system.

PRIMARY AMENTIA.

Simple Primary Amentia.—Although in some respects less interesting to the medical man than many of the more clearly defined groups of mentally defective children, this class is far more important than any of them from a practical point of view. It is, for one thing, larger than all the others put together; and, for another, it includes within it many of the epileptics, and nearly all the slightly feeble-minded children whose treatment forms in after-life such a grave and puzzling problem, both from the patient's point of view and from that of the public.

Ætiology.—The causation of this variety of mental defect is a very difficult subject, and one which cannot be satisfactorily discussed here. Most writers are agreed that a *neurotic heredity* is by far the most important influence at work in its production. Tredgold found this in 80 per cent., Ashby in 75 per cent., of the cases. *Alcoholism and tuberculosis* in the parents may exert some influence, but their importance has probably been enormously overrated. A history of parental alcoholism is not very uncommon in these cases, but it is probably to be regarded less as a cause than as a manifestation of the neurotic strain which is at the root of the trouble. A tuberculous family history is another evidence that the stock is a weak one, but it can scarcely be blamed as a direct cause of amentia. *Consanguinity of parents*, which has such an important influence in determining the occurrence of disfunction and certain other congenital abnormalities, seems to have little ætiological importance in this type of amentia. I have found it present only in between 2 and 4 per cent. of my cases. *Congenital syphilis* has hitherto been generally regarded as a rare cause of primary amentia. A number of bacteriologists of repute in various countries have recently made use of Wassermann's test to investigate this point. Their results have as yet, however, differed so widely from one another (from 1/2 up to 55 per cent. of positive reactions) that we can only gather from them that syphilis may possibly be a more important ætiological factor in amentia than we had formerly suspected.

Symptoms.—This class includes all degrees of mental impairment from the slightest to the most profound, and the symptoms vary considerably according to the amount of intelligence present.

Severe Cases.—In the worst cases of simple primary amentia the appearances are those of utter idiosy. The baby is evidently abnormal from the very first—dull and apathetic, paying no regard to his surroundings. He often does not know his mother by sight, though he may turn at her voice; sometimes he only recognizes her, if at all, by the way she holds him. The abnormal gestures and actions of these babies, and their want of the natural automatic movements of infancy, have already been referred to (p. 871). A considerable majority of them suffer also from frequently recurring convulsions. These are sometimes of a regularly epileptiform

character, but very often in young halos they take the form of petit mal; and when this is so, their serious significance is very apt to be overlooked by the parents. The child gives from time to time a sudden start, the head, shoulders, and lower limbs being momentarily jerked forwards; a short period of loss of consciousness may follow, with a little stertorous breathing and on waking up the baby often cries bitterly, as if much distressed.

Less Severe Cases.—In these the symptoms may be much less noticeable, especially during infancy. Dentition and the closure of the fontanelle proceed as in normal children; and, as there is no paralysis or gross deformity and no markedly abnormal gestures, the child's want is often overlooked during the early years of life. Someone, however, generally notices that the baby is strangely long in showing an interest in bright lights and moving objects, and is late in recognizing his parents and the other children. He is also backward in learning to use his hands and in holding up his head, in sitting up, in standing, and in walking. When he does learn to walk, his gait for long remains clumsy and like that of a younger child. Such children often go with their heads slightly forward and their arms hanging in front; their feet are planted far apart, their pelvis moves too much in walking, and the joints of their lower limbs too little. Their hands, too, remain clumsy and nerveless. A common symptom is a more or less constant dribbling of saliva, which is very troublesome. Proper control of the bladder and bowel is often long in being acquired—sometimes not till four, six, or even eight years old—and yet it may become quite normal in time. Convulsive attacks are common, though not so frequent as in the severer cases.

The mental symptoms may not be very prominent at first, but the children are generally too long of talking, their speech is often indistinct and the words badly pronounced. When they reach an age at which they should be beginning to learn some sort of lessons, they are found to be very hard to teach, because they cannot give their attention continuously for any time to the same thing. Even when they are doing what they like, they seem incapable of perseverance. Many are very deficient in affection, and all in common sense. They lack self-control, laugh and cry without sufficient reason, and often get into senseless passions. A common indication of their defect is their inability to play peacefully with normal children, who have little patience with their thoughtless ways, and are very apt to tease and irritate them.

Slight Cases.—Very mild cases of mental defect, such as form the proper pupils of the "special classes" in the public schools, generally pass quite unrecognized through infancy and early childhood. They have wit enough to perform passably all the duties of ordinary babyhood. It is only when the time comes that they should leave hold of their mother's apron strings that they show their utter inability to guide themselves properly and to keep pace with their fellows. The facts that they are late in walking and talking, and that they are long in losing their baby speech and their babyish ways of behaving, are thought little of. If they have fits, these are apt to be attributed only to peripheral causes and unusual smallness of the head, and any other abnormalities, if present, are also easily overlooked. When their school-time comes, however, the teachers soon discover their weakness. The mental and manual work they do are both below the average—usually very much so. They are probably amiable, but very feeble and unsatisfactory, with no strength of purpose, little practical sense of right and wrong, and no proper realization of their responsibilities.

In later life their weakness of will and lack of self-control and of common sense are constantly landing them in trouble whenever they leave the shelter of home and come in contact with life outside. If they can be kept carefully guarded in wisely-managed homes, they remain for life good and happy children. The provision of suitable institutions, where such individuals can be shielded from the evil outside influences which they are powerless to resist, is at present one of the most urgent needs in our public life. Until some such provision is made, the members of this class among the poor will continue to go from one folly to another, while the public pay for keeping them for a large part of their lives in prisons, workhouses, and Magdalene asylums. When they reach adult life, they are usually very prolific, and their children are often mentally defective.

Simple primary amentia is frequently met with in several members of a family.

PATHOLOGY.—To the naked eye the brain in simple primary amentia presents as constant characteristic appearance. The microscopic structure still requires further investigation; but Bolton, Tredgold, and others have shown that there is evidence of imperfect or arrested development of the cerebral neurons. The nerve cells in the cortex are ill-grown and often abnormally pigmented; they are also relatively few in number, and their arrangement is irregular. The degree of these defects varies directly according to the amount of the intellectual impairment. There is also a diminution in the number of the nerve fibres, and an overgrowth of the neuroglia (schwann) is frequently present.

The **DIAGNOSIS** and **PROGNOSIS** have been already sufficiently dealt with, and the management of the cases will be fully considered under the heading of General Treatment.

Microcephalism.—The term "microcephalus" is used by many as synonymous with "small-headed," and any child the circumference of whose cranium is less than 17 inches is called "microcephalic." It is probably better, however, to keep the term for the small and fairly well defined group of cases sometimes called "true microcephalus." These are distinguished by very small heads, which are also of a peculiar form, the forehead being narrow, the vertex pointed, and the occipital region flat. They are likewise characterized by extremely early closure of the fontanelle. The brain is dwarfed, and its different parts are out of proportion, but it is of normal consistence, and shows no visible signs of past disease and no gross congenital malformations or defects.

ÆTIOLGY.—The causation of the condition is still quite obscure, although various theories have been put forward to explain its origin. One of these, for which a good deal has been said, regards microcephalus as an atavistic variation—a reversion to a simian type. This view was first suggested by C. Vogt, and has been supported in recent times by D. J. Cunningham and Telford-Smith. Another interesting theory is that the dwarfed growth of the brain is the direct result of abnormally early closure of the sutures and fontanelle. This hypothesis was formerly supported by Virchow and other great authorities, but it has now been universally given up, as it is certain that the arrest of cerebral growth is the primary, and the cranial change merely a secondary, phenomenon. It has also been shown by Cunningham and Telford-Smith that the interference with growth of the brain begins at too early a period for this theory to be true—some time between the third and fourth month of intra-uterine life. It is generally considered that a neurotic

inheritance has much to do with the etiology of this, as of other forms of primary amentia.

Symptoms.—The parents of these children usually seem healthy, although they are often found to belong to a nervous stock. There is no peculiarity with regard to the age of the mother at the time of conception, the sex of the child, or his order in the family. Sometimes the patient is a twin, and in that case the other twin may or may not be microcephalic.

The child's appearance is very characteristic if the case is at all a well-marked one (Figs. 133 and 134). The head from early infancy is obviously too small, and, as the body grows at a fairly normal rate and the cranium very slowly, this disproportion soon becomes very striking. As he gets older the child has a tendency to hold his head stretched forward; and this, along with the rapid way in which he jerks it from side to side, gives him the curious "bird-like" aspect which has often been remarked upon.

By the sixth month of life, when the normal child's head should have grown from a birth measurement of 13 or 13½ inches to one of 16 inches or thereby, in the microcephalic it is still only 13 or 14 inches



FIG. 133.—MICROCEPHALUS; CHILD, SEVEN AND FIVE YEARS AND NINE MONTHS.



FIG. 134.—MICROCEPHALUS; GIRL SEVEN TWO YEARS.

At twelve months it may be 14 to 14½ inches instead of 18 inches; at two years, 15 or 16 inches instead of about 19 inches. In older children the circumference may remain only 15 to 17 inches, although sometimes it is a little larger. Besides being small, the head, as already described, is of peculiar shape. The condition of the fontanelle is an important point in the diagnosis. In many cases it is entirely closed at birth, so far as can be felt; in others it is much too small. The palate may be very highly arched. The features are usually well formed and of normal size. The ears are noticeably large and well grown. Although the body looks big during early childhood, it is rare to see a microcephalic in later life who is not small in stature.

The first teeth appear at the usual time, but the child generally does not walk until the third or fourth year, or even later. Speech is scanty and slowly acquired;

control over micturition and defecation is sometimes late in coming, and in bad cases may never be quite satisfactory. In one-third of my cases there was a history of convulsions; and had the children been older when I saw them, this proportion would probably have been much larger. In true microcephalus there is generally no real paralysis, but during the early months of life many of these children show a sinking degree of spasticity. This apparently represents an exaggeration of the normal hypertonicity of healthy new-born children, and probably depends on the backward development of the cortex. It passes off completely as the children grow older.

The child's intellectual attainments and his disposition vary greatly in different cases. In the worst cases he is often very unobservant and apathetic during infancy, and in later childhood of a low mental type, although quick and active in his movements. He is generally restless, inquisitive and greedy, quite devoid of affection, and dangerous to smaller children and to animals. When the head is not so small, however, there may be a very fair degree of intelligence combined with a kindly disposition, and the child may be capable of a considerable amount of education.

PATHOLOGY.—The general hypoplasia of the brain and spinal cord which is characteristic of these cases is most marked in the posterior portions of the cerebrum, so that the cerebellum is left uncovered as in the brains of the lower animals. The cerebral surface shows a simple arrangement of the convolutions and a lack of secondary gyri. The nerve cells in the cortex and spinal cord are imperfectly developed. Microcephalus is sometimes complicated by the presence of hydrocephalus and by inflammatory lesions.

DIAGNOSIS.—Complete absence of the anterior fontanelle at the time of birth, or very soon after, always indicates microcephalus. Its closure by the sixth or seventh month is occasionally met with in cases of arrest of cerebral development due to gross cerebral lesions. In the great majority of cases, however, such premature closure indicates the presence of microcephalus. In later childhood the characteristic physiognomy is easy to distinguish from that of advanced spastic diplegia. When extreme smallness of the head is found without spastic condition of the limbs, true microcephalus may always be diagnosed.

PROGNOSIS.—The outlook, both as to length of life and as to mental improvement, is generally bad. The children are usually more or less delicate and very prone to tuberculosis, so that most of them die in early childhood. In rare cases, however, they live to old age.

TREATMENT.—Extreme cases are ineducable, but in those that are less severe institution treatment is sometimes very successful in improving the intelligence. A wholesome routine of happy occupations with a more or less open-air life may very strikingly lessen the abnormal restlessness and the tendency to convulsions.

Mongolism.—In 1866 J. Langdon Down drew attention to a group of imbecile children whose features had a curious resemblance to those of the Mongolian race. These cases were further investigated by Arthur Mitchell and John Fraser and many others, and it soon came to be recognized that they formed quite a distinct class and presented a large number of points of interest.

FREQUENCY.—The condition is very common in this country. Langdon Down regarded it as responsible for 10 per cent. of all imbecile children. Shuttleworth states the proportion as 5 per cent., while Still found no fewer than 22 per cent.

of mongols among 350 imbeciles. The variations between these estimates is easily explained by the difference in the ages of the children examined. This acts in two ways: (1) A considerable majority of mongols die during the first few years of life, and (2) many of the milder cases of the other kinds of *anentia* remain quite undiagnosed in infancy and early childhood. It is therefore easy to understand how statistics drawn from children's hospitals give a far larger proportion of mongols than those taken from institutions to which only older children are admitted. Among 300 consecutive cases of mental defect in children of all ages, seen as out-patients at the Edinburgh Children's Hospital, 37 were mongols (i.e., 12.3 per cent.). On analysis of the ages, however, it was found that while 150 consecutive cases under four years old included 29 of mongolism (i.e., 19.3 per cent.), another 150 of four years and over showed only 8 of them (i.e., 5.2 per cent.). In Germany the proportion seems to be much lower, as H. Vogt, Abt. and Weygandt all give it as 1 per cent. of all imbeciles.

Ætiology.—Although mongolism is so common, almost nothing is known about its causation. There seems to be no reason whatever to think that either tuberculosis, syphilis, or alcoholism, has any special influence in producing it. A neurotic heredity is often, although not always, discoverable. The only facts known which can be claimed as having any ætiological significance are the child's order in the family, the age of the mother at the time of conception, and the frequency with which congenital malformations are met with, especially cardiac abnormalities. The exact bearing of these on the matter is, as yet, far from clear. It is quite exceptional to find any history of special ill-health in the mother during her pregnancy.

Shuttleworth has spoken of mongols as "exhaustion products," and there seems to be good reason for holding this view. For one thing, a large proportion of the cases come at the end of large families. Out of 138 cases I found that 22 were the result of first, 16 of second, and 39 of third, fourth, or fifth pregnancies; while 25 resulted from sixth, seventh, or eighth, 29 from ninth to thirteenth pregnancies, and 7 from fourteenth to seventeenth pregnancies.

Another and even more important point is that the great majority are the offspring of mothers who are near the end of their child-bearing period. In 79 of my cases in which this was inquired into, the average age of the mother when the mongol child was born was thirty-seven and a half years, and in only 7 of them was the mother under thirty years. In none of these young mothers could any special disease or debility be discovered, and several of them had healthy children afterwards. One instance of mongol twins has been reported (Shuttleworth), and several of twins one of which was a mongol (Shuttleworth, Mitchell).

Symptoms.—Mongolism affects the conformation of the entire body, but its signs are most readily seen in the face, head, and hands. The *facies* (Figs. 135, 136, and 137) is generally easy to recognize, even from birth; but, although a single glance is usually all that is required to make sure of it, it is very hard to describe its characters helpfully in words. These consist of many small peculiarities, and so one of them is invariably present or really pathognomonic. Indeed, we generally recognize mongols not so much because we have noted in them any special feature of the disease, but rather because they have so strong a "family likeness" to other mongols, when they resemble much more than they do their own relatives. They seem, indeed, as if they belonged to a distinct species of human beings.

The cranium is small, brachycephalic, rounded, and devoid of eminences. The features also are small and rounded, and they are often reddish in tint. The eyes are frequently too near to one another, and in most cases the axes of the palpebral fissures are oblique, their outer angle pointing somewhat upwards. In many the epicanthic fold of skin at the inner canthus is strongly developed (Figs. 135 and 136). The eyelids are often thickened by blepharitis. Squint is common (9.4 per cent.), and nystagmus even more so (12.3 per cent.). It has recently been pointed out by Pearse, Rankine, and Ormrod, that a peculiar spotted form of lamellar cataract is often present in children over nine years old. The nose is usually snubbed, and the nostrils in early life have often a peculiar triangular shape. Adenoids are frequently present, and in some cases they produce severe symptoms, even when scanty, owing to the small size of the naso-pharynx.

One of the most characteristic features is the protrusion and sucking of the tongue. The protrusion, which is seen mostly in young children, seems to be due rather to the shortness of the mouth than to any special largeness of the tongue. In babies under six or nine months the surface of the tongue appears quite normal. After this age, however, the papillae enlarge in increasing numbers, until it assumes a raw, granular appearance. Later, usually during the third or fourth year, deep transverse fissures form across the dorsum. It is rare to see a mongol over six years in whom this fissuring is not present. It is therefore a marked characteristic of mongolism, and it has a special interest, because, unlike the other typical features of the condition, it is acquired, and not congenital. Its occurrence may possibly be partly due to an inherent vulnerability of the organ. There is good reason, however, to believe that it is mainly, and perhaps entirely, the result of the vigorous and constant tongue-sucking which these children practice, with very few exceptions (Thomson). An exactly similar condition of the tongue is occasionally seen in patients who are not mongols. In these cases it is, in my experience, always found that the children have been addicted to sucking their tongues for years.

The teeth are small, and usually tend to become yellow and to decay early. In many cases the central incisors are twisted round on their axes, so that the surfaces of the crowns form an angle with one another, with the concavity forwards. The mucous membrane of the lips is often dry and fissured.

The ears may be almost normal in form, but generally they show some deformity. They are often small, rounded, and simply convoluted, and they also stand out from the head more than usual. In older children the hair is apt to be dry and poor, and occasionally alopecia develops. In babies both the skin and the hair are soft and natural.



FIG. 135.—MONGOLIAN BOY AGED FOUR, SHOWING PUPILS, EPICANTHIC FOLDS, AND PROTRUDED TONGUE.

The limbs are small-boned and soft, and rather shorter than normal compared with the trunk. Their ligaments are so lax that the joints can usually be easily hyperextended (Fig. 137). The hands are often thick and clumsy, but less so than in cretinism. The fingers, though thick for the size of the hand, are not so square-tipped as those of cretins, nor is the skin much wrinkled. The thumb is short, and the little finger, in more than half the cases, is both dwarfed and somewhat curved towards the ring-finger (Figs. 136 and 138).



FIG. 138.—MONGOLISM: GIRL, AGED NINE YEARS, SHOWING EPICANTHIC FOLDS, FINISHED TONGUE, AND CHARACTERISTIC HANDS.

In addition to the numerous small defects of conformation above described, we often meet with other congenital abnormalities of various parts. The following is a list of those found in the examination of 138 cases:

	Cases		Cases
Cleft palate	1	Hypospodias	1
Congenital cataract	1	Undescended testicle	2
Supernumerary teats	2	Foot-heel dimple	1
Congenital heart disease	22	Congenital dilatation of recto-abdominal ..	1
Club-foot	4	Umbilical hernia	2
Gross oesophagus and no palate ..	1	Inguinal hernia	1
Cubitus varus	4	Congenital stenosis of rectum	1
Syndactyly of toes	1	Congenital stenosis of large intestine ..	1
Dwarfed terminal phalanges of fingers with deformed nails	4		

Far the most interesting of these abnormalities are the heart conditions, to which attention was first drawn by A. E. Garrod. The proportion of heart cases met with

varies according to the age of the patients seen, because the presence of a cardiac defect greatly lessens a child's chance of survival. In 194 mongol children under four years 20 (19.2 per cent.) had congenital heart disease, while in 34 over that age only 2 (5.9 per cent.) were affected in this way. The most frequent heart lesion, in my experience, has been patency of the interventricular opening, the ductus arteriosus, and the foramen ovale; but in two of my cases there were extensive adhesions of the tricuspid valve apparently from intra-uterine endocarditis.

The first dentition is generally very backward. In two-thirds of the cases the first teeth did not appear until after the end of the first year, and often not until the child was eighteen months or two years old. In the others, although the first few teeth came early, the later ones were usually much delayed, and often appeared in a wrong order.

The voluntary muscular movements are slow of being acquired. Often the baby does not hold up his head until the sixth or even ninth month, or sit until



FIG. 131.—MONGOLISM: CHILD, USED FOR OTHER MOTIONS, SHOWING GENERAL APPEARANCE AND HYPEREXTENSIBILITY OF JOINTS.



FIG. 132.—MONGOLISM: HANDS OF BOY, AGED FOUR YEARS.

the end of the first year or later. The average age of learning to walk is about three and a quarter years (one and a half to five and a half), and the gait is often very clumsy. The grasp, also, is usually feeble and fumbling. Speech is late of being acquired in most cases, generally about three and a half years (two to seven years). It is guttural and indistinct, and often very scanty. In the worst cases the child can scarcely say anything. The characteristic disposition is bright and lively, and the child, although imbecile, may be very quick at doing many small things which are learned by imitation. Many mongols can in time be taught to read, but few derive much pleasure from the accomplishment.

At birth the baby is small, and, as his rate of growth is abnormally slow, the dwarfing becomes more noticeable as he grows older. He is very apt to take infective diseases, and to have them badly. Convulsions sometimes occur (6 times in 128 cases), but they are rarer in mongolism than in most other kinds of amentia. Obstinate catarrh of the eyelids, nose, and throat, is very common. Bronchitis is especially frequent, and always tends to end in collapse and broncho-pneumonia. When the chest affection is primary, it is surprising how many weakly-looking

infants recover from it; but when secondary to measles, hooping-cough, or influenza, it is generally fatal. Indeed, in my experience, this accounts for more than two-thirds of the deaths in mongols who die during the first few years of life. Most of the older patients die of tuberculosis. Mongol infants are so apt to take acute diseases badly that only a small proportion of them survive infancy. When they do so they are often fairly robust. Those who live beyond forty, however, soon get to look very old and frail.

PATHOLOGY.—At first sight the mongol's brain looks like that of other children. There is no gross, naked-eye malformation, and no trace of inflammatory change or sclerosis. On closer examination, however, various slight abnormalities may be noted. Thus, Wilmarth and G. A. Sutherland found the pons and medulla only half the normal size, while Waterston and others have described "a retentive of the infantile type in the convolutions of both the frontal and parietal lobe." No constant or characteristic change has been found on the microscopic examination of the brain. There seems to be no ground for the idea that a pathogenic locus is to be found either in the *basia cranii* or in the *cortex cerebri*. The thyroid is normal.

The effects of mongolism on the body are, indeed, so numerous and so universal that it seems most unlikely that they can all be secondary to a localized lesion in the brain. In all probability the cerebral changes, and those in the other organs and structures, are all to be regarded as secondary to the same cause. What this can be is entirely unknown, but during early intra-uterine life something certainly interferes with the normal influence which makes for the healthy development of all the tissues. Although mongols seem to be in a sense "unfinished children," as Shuttleworth says, yet they are not quite so like premature infants as one might have expected them to be had their development merely come to a standstill a few months before birth. It is certain that whatever goes wrong in their growth does so in very early intra-uterine life—probably by the second month, if we are to judge by the nature of the cardiac defects. The later development would appear to go on continuously, though badly. "Ill-finished" would perhaps be a better adjective than "unfinished" to apply to these children.

The **DIAGNOSIS** and **PROGNOSIS** have been dealt with under **SYMPTOMATOLOGY**, and need not be further alluded to here.

The **TREATMENT** is on general lines. Owing, however, to the good-natured disposition of these children, their limitations as to learning, and their delicacy of health, it is quite often found better to keep them at home than to send them to an institution.

Congenital Hydrocephalus and Porencephalus. Vide p. 815.

Tuberous Sclerosis.—This is a rare developmental disease, which is characterized anatomically by rounded areas of sclerosis in the brain, and by peculiar lesions in the skin and several of the internal organs, and clinically by convulsions and mental deterioration. The name "tuberous sclerosis" was first given to the disease by Bourneville in 1880, and much of our knowledge of it is derived from his writings and those of H. Vogt. Nothing is known regarding its etiology.

SYMPTOMS.—The convulsions, which occur in nearly all cases and form the most striking manifestation of the disease, may begin during the first weeks or months of life, but just as often they are deferred till later childhood. They are generally

anxious, and have no peculiar characters by which they can be distinguished from fits due to other causes.

In most of the published cases there seems to have been some mental defect present from birth, but in a few the intellect has remained normal for a varying period of years, and has only shown signs of deterioration after the convulsions had persisted for some time. Spastic paralysis and contractures are only occasionally found. Various malformations, such as congenital heart disease, spina bifida, and hydranrylia have been recorded as complications.

PATHOLOGY.—The characteristic cerebral tumours, which are usually numerous, are scattered throughout the cortex. They are rounded in contour, forming projections on the surface, and also extending into the subjacent tissue. Their periphery is ill-defined, and their consistence, especially towards the centre, is denser than that of the surrounding tissue.

On microscopic section, numerous large, irregularly-shaped cells are found in them, which apparently are ganglionic nerve cells, and are most numerous towards the periphery of the tumours. Small hard growths, the size of henop-seeds, may also occur in the ependyma, projecting into the ventricles. The tumours in the heart are rhabdomyomata (Fowler and Carnegie Dickson), while those in the kidney, according to Fischer, who reports on eight cases, are mixed growths consisting of net-striped muscle, fat, arteries, and sometimes primitive kidney cells in varying proportions. Tumours have also been found in the thyroid, thymus, pancreas, breast, and duodenum. The most important of the growths from a clinical point of view are those in the skin which form the well-known eruption called "adenoma sebaceum." This occurs mostly on the face, round the nose and mouth, and on the forehead, and is composed of little red nodules of sebaceous gland tissue embedded in a vascular matrix.

It is said that the brain condition in tuberous sclerosis probably dates from the seventh month of fetal life, or soon after it.

DIAGNOSIS.—Tuberous sclerosis can only be distinguished from ordinary epilepsy if adenoma sebaceum is present, or renal symptoms, such as a palpable kidney tumour or peculiar cells in the urine.

PROGNOSIS.—This is invariably bad, as the patients always die either during childhood or in early adult life. Death may be due to the convulsive seizures, to the renal tumours, or to some intercurrent disease. There is, of course, no treatment beyond that of the convulsions.

SECONDARY AMENTIA.

Epilepsy in Relation to Mental Defect.—As we have already seen, epileptic seizures are among the commonest clinical phenomena met with in simple primary amentia and many other types of mental defect. They occur, indeed, in all its varieties, not excluding cretinism and mongolism, although in these they are less frequent than in the other types. In the great majority of instances, when fits occur in mentally deficient children, they are secondary to the main organic disease which is causing the amentia, and are not its cause. At the same time, their repeated recurrence has generally an obviously bad effect on the mental state; the child comes to insipide while they go on recurring, and brightens up in every way when they pass off.

There is, however, an important, although smaller, group of cases which we have now to mention, in which more or less severe mental impairment takes place as the result of recurring convulsions which are not due to any ascertainable organic cause. In this connection it is important to note that it is only certain types of convulsion which lead to deterioration of the intellect. The fits, of doubtful origin, which are met with in young infants, and which often recur many times a day for weeks, are apt, when they come to an end, to be succeeded by a long period of drowsiness and apathy; but they do not usually leave any permanent mental defect behind them. The same absence of permanent damage is noticed in the case of the so-called "rickety fits" which are so commonly met with, when in association with tetany and laryngismus, and also in the convulsions which occur from time to time along with fever and digestive derangements in nervous children.

In true epilepsy in childhood the outlook is very different, for in it the intellect is almost invariably damaged. The degree to which this occurs varies in different instances, partly with the frequency and character of the fits, but chiefly with the age of the patient. In children under five years even a few apparently slight epileptic attacks may have an obviously bad effect on the mind and character. Chronic cases of epilepsy in early childhood always show some degree of mental deterioration or peculiarity, and frequent and severe convulsions have always a rapidly demoralizing influence on the intellect.

In the intervals between the attacks the child becomes dull and apathetic, and seems unable to concentrate his attention on things. When this stupifying effect wears off, he is often found to have changed somewhat in mental attitude and character. There is a tendency for him to lose the normal shyness of childhood and the usual sense of responsibility and propriety. He is at the mercy of sudden moods and impulses. Sometimes he goes into wild passions; at other times, it may be without any appearance of anger, he will suddenly strike another child on the head with a stone, or fling his jug of milk across the room. All sorts of reckless mischief seem to attract him, and he is constantly getting into trouble for upsetting ornaments, setting fire to clothes or furniture, tearing books, kicking doors, etc. Sometimes the patient's condition is one of mental exaltation, and in rare cases maniacal symptoms develop; indeed, most cases of mania in young children *arise* in epileptics. The excited phase, if not soon recovered from, tends to pass on to a state of dementia.

Amentia due to Gross Cerebral Lesions.—Any form of gross cerebral disease or injury which causes paralysis is apt to be accompanied by more or less impairment of the mental functions. Cases of the kind in which no intellectual weakness can be made out do, indeed, occur, but they are quite exceptional. Even when the parents regard the child as normal in mind, we usually find at least a degree of mental instability, a tendency to epileptic fits, some emotional weakness, or some other slight mental peculiarity which shows that the psychic centres have not altogether escaped damage. In many cases the mental symptoms are more marked than the paralysis. When the anterior lobes of the brain only are affected, there may, of course, be severe amentia without any paralysis.

Far the commonest type of paralysis associated with amentia is spastic diplegia. This may arise from various lesions, but is certainly in many cases attributable to meningeal hemorrhage of birth origin, with or without laceration of the brain. Children affected in this way are very helpless, and, by reason of their frequent grimacing and grotesque movements, they look much more foolish than they really

are. Many of them, indeed, have great determination and not a little common sense, so that they sometimes improve surprisingly under careful training.

In cases of hemiplegia from birth injury the mental symptoms are usually less severe than in those of diplegia.

The *amentia* associated with chronic hydrocephalus of congenital origin is generally of rather a mild type, and the children are docile and well disposed, although lacking in energy and initiative. In the form of chronic hydrocephalus which follows *basis meningitis* the mental defect is generally more severe than in the congenital cases.

Cases of cerebral damage from cerebro-spinal meningitis, apart from hydrocephalus, and cases of *polioencephalitis*, are usually characterized by serious mental enfeeblement, and the prognosis as to improvement under training in these conditions is very bad.

Infantile cerebral degeneration (*amaurotic family idiocy*) is discussed elsewhere (p. 844).

Congenital Syphilis in Relation to Mental Defect.—The importance of the part played by hereditary syphilis in the causation of mental defect in childhood has, until recent years, been somewhat underestimated. This has been partly due to the question having been looked at too exclusively from the point of view of the insane asylums, in which as a rule only from 1 to 2 per cent. of evidently syphilitic children are to be found, as most children with syphilitic brain lesions are far too ill for institution treatment. The cerebral condition, if it does not end in early death, generally leads to such severe dementia and bodily debility that the child is kept at home because he is obviously quite unfit for any special training.

Congenital syphilis acts in various ways in producing mental defect.

1. It may, like any other weakening influence, interfere with the development of the cerebral tissues, and produce either a deformed brain or ill-grown brain cells. The use of the Wassermann reaction has recently shown that a number of cases of apparently idiopathic primary *amentia* and *epilepsy* are probably syphilitic in origin.

2. Syphilitic endarteritis may occasion cerebral lesions which manifest themselves in various ways—in hemiplegia or other forms of paralysis with *amentia*.

3. Various gross cerebral lesions of a congenital syphilitic nature produce severe mental symptoms. Of these, far the most important and common is the form of *meningo-encephalitis* which in older children produces "juvenile general paralysis," and in younger ones simple progressive dementia with spastic diplegia. These types of disease occur, in more than half of the cases, in children who are congenital inbreds; and in practice, when forming a prognosis regarding an inbred who has signs of syphilis, it is always well to bear in mind the probability that the case may end in progressive dementia.

Congenital syphilitic gummata of the brain occasionally occur, but they are very rare.

Classic hydrocephalus of hereditary syphilitic origin in infants is by no means rare in the out-patient departments of children's hospitals. The cranial enlargement in these cases is not usually very great, and, under active specific treatment, recovery often takes place quite satisfactorily.

Aitby has described a form of convulsion of Jacksonian type, followed by *idiocy*, which is met with in young infants, and is due to syphilitic *meningo-encephalitis* of the cerebral cortex.

MORAL IMBECILITY.

The ordinary mentally defective child is very often notably deficient in moral sense, although it is his intellectual want that is his main characteristic. Occasionally, however, we meet with what are called "moral imbeciles"—children who, in spite of an average or almost average capacity for lessons and for looking after themselves, seem to have been born without a conscience, and not to have the power of acquiring one, like other children, as the result of experience. An ordinarily careful upbringing fails to give such children any due sense of the difference between right and wrong, and no amount of punishment or of persuasion is able to break their habits of telling lies, stealing, and doing cruel and indecent actions. Marked instances of this kind are, fortunately, very rare, but they are occasionally met with, even in children as young as five or six years old.

DIAGNOSIS.—The recognition of moral imbecility is not usually difficult, but there are certain cases of moral defect in epileptics which may be mistakes for it. It is also important to remember that some school-children, especially girls who are approaching puberty, are apt to distress their parents and teachers by going through a phase of untruthfulness or pilfering, which passes off within a short time, and has no very serious significance.

TREATMENT.—For their own sakes, as well as for that of the community, morally defective children should, as soon as possible, be sent to some institution where they will be constantly under proper supervision, and where they can be supplied with sufficiently interesting and useful occupations to keep them out of mischief. When this is done, the extent to which improvement takes place is sometimes surprising. If they are not taken in hand in this way, they go from bad to worse, and eventually join the ranks of the criminal classes.

GENERAL TREATMENT.

The treatment of the mentally defective child is, of course, mainly a question of his training. In considering this, two things must be borne in mind. The *first* of these is that his condition is *essentially* an incurable one—he was born defective, and he will remain so, whatever we do. However well he is treated, there is no chance that he will ever quite make up on his normal companions in the race of life as we might have expected him to do had he merely been mentally backward. Our ultimate aim for him has, therefore, to be different in some ways from what it would have been had his intellect been normal, and many things that an ordinary child must learn may in his case be wisely omitted, or, at least, laid little stress on.

The *second* point to remember is a mere encouraging one—namely, that practically all mentally defective children, however bad they are, are capable of *some* kind of improvement under judicious training, and that most of them can be very greatly bettered in many ways. It is to this second point that we must always try to draw the parents' attention. Many of them find it exceedingly difficult to believe that any child of theirs can really be wanting in intellect. If the fact is urged upon them by a medical man before their own observation has prepared them to receive it, they are apt to reject it with more or less annoyance; while they

lead a ready ear to foolish people who tell them that, at seven, or fourteen, or some other age, there is sure to be complete and spontaneous recovery. It is, therefore, extremely desirable to induce parents to concentrate their interest, as much as possible on such aspects of their child's deficiency as are likely to be improved by individual attention from them. This is not only the best thing for the child, but also for the parents themselves, because it brings them, in the easiest and most natural way, to see what the child's limitations really are. Such knowledge will prevent them from forming false hopes such as often lead to much unnecessary pain afterwards; at the same time it will prepare their minds to face sensibly the hard problems of the child's future.

The object of our treatment, so far as the mentally defective child himself is concerned, is to make him as happy and as good as possible. As his happiness will largely depend on how many things he can and does do and notice, and on whether his ways and manners are pleasing to others, or the reverse, our chief aims must be to make him more capable of performing the common actions of everyday life gracefully and like other people, and so to form his manners and conduct that they may be unobjectionable to those he associates with. It is also important as far as possible to cultivate a sense of responsibility in all that he does, and to show him that his duty lies in doing things that he is quite able to do. We must remember the great pleasure that a child always derives from the active use of his ordinary functions. We may succeed in pleasing the defective child by providing pleasant sensations—soft and warm clothing, sweet tastes and smells, bright colours and bright lights, and cheerful music (or chatter if he prefers it)—but, if we wish him to get all the happiness he can out of life, we must go farther, and make him more than a passive receiver of benefits. So far as possible he must be induced to exert his own powers of body and mind, and, above all, his affections. It is a very true saying that occupation is the key to success in dealing with mentally defective children, while idleness is to them the root of all evil.

The main indications for the treatment of these children in early life may be stated as follows:

1. *Attend to the General Health.*—This includes the giving of good plain food suited to the child's powers of mastication, and the careful regulation of his bowels. Frequent baths are necessary, and a modified cold douche, along with much open-air exercise, warm clothing, and a sufficiency of fat in the food, form the best means of encouraging the feeble circulation.

If contractions occur, their treatment is, of course, of great importance. It is also very important that if rickets, anaemia, cardiac weakness, tuberculosis, or dyspepsia, are present, they should be energetically dealt with, for they may be interfering greatly with the child's mental as well as with his bodily vigour. Local defects, such as refractive errors, adenoids, and contracted tendons, are well worth attending to, except in very low-grade cases. Massage and musical drill are often useful in slighter cases. Operations on the cranium have hitherto proved of no value whatever.

2. *Awaken the Child's Faculties.*—(a) *Bodily*, and (b) *Mental*.—(a) He must be encouraged in the voluntary use of all his muscles regularly and carefully. This improves his strength and his co-ordination, and it is very helpful in stopping the purposeless automatic actions which are sometimes present. For older and more intelligent children, musical drill, skipping, dancing, trundling hoops, playing at home and at soldiers, dumb-bell exercises, ball games, bean-bags, walking between

the rings of a ladder, nail-boards, threading beads, building with bricks, and all sorts of kindergarten games, are useful. Singing and reciting are also good for these children, and so are playing with dolls, and various forms of golfing.

For the younger ones, and those who are severely affected, very simple actions, such as clapping hands and playing with a rattle or with a suspended ball, answer to some extent the same purpose.

Mentally defective children should, if possible, be taught to chew their food. Sometimes this is a great difficulty, but if the jaws are not put to their normal use the teeth are apt to decay badly, and much ill health may result from this. If the child dribbles, an attempt should be made to strengthen his lips by making him hold a fat piece of wood between them for a given time every day, and getting him to blow a whistle or trumpet, inflate balloons, etc.

(b) Teach the child to notice things, and to compare their characters—roughness and smoothness, hardness and softness, heaviness and lightness, heat and cold, different colors, shapes (circles, squares, triangles, spheres, cubes, cylinders, etc.), distances, sounds (musical and other), tastes, and smells. Take him out, or to the window, where he can see the people, houses, dogs, cats, etc. If such objects do not attract him, perhaps bright lights and colors will. Let him have whatever is found to arouse his attention. Encourage him to look at, listen to, and handle anything that he is taken up with. Any sort of interest will help to brighten him.

3. Encourage him to use his *Acquired Faculties in giving himself Pleasure*.—The mentally defective child needs to be taught to do many things which normal children do of their own accord without any teaching. He should be incited to do things, and at first easy successes should be planned for him. If he deliberately wants a thing, tries to get it, and succeeds, this is a most valuable and a very pleasant lesson for him; but he must not be disheartened by being given tasks beyond his capacity. In the case of a baby, if he likes a noise, let him have something that he can make it with for himself. Never let the mother or nurse go on doing for the child anything that he can himself be made to do for his own pleasure. For most mentally defective children mere memory knowledge is of little use, but the more things they can do the better for them.

4. *Provoke Self-Control and cultivate the Moral Character*.—This is very important, and includes many things. One of the first of these is the training of the child in keeping himself clean, and in letting his mother know when he requires to be attended to. Some mentally defective children can never be taught cleanly habits, but in most cases, even when there has been complete incontinence in early childhood, the child may in time, by persevering attention, be trained to have completely normal control. General cleanliness and tidiness in person and dress are also to be constantly insisted on, and the child should be encouraged to take a pride in having everything about him nice.

Another point of great importance is that all sorts of "bad habits," to which these children are particularly prone, must be watched for and checked at their earliest beginning. This refers not only to such habits as thumb-sucking, dirt-eating, and masturbation, but also to unpleasant tricks of manner and expression (grimacing, unconstrained gestures, making unpleasant noises, etc.). These do the children much harm, because they draw attention to their defect and make them very objectionable to other people.

The acquisition of good manners, including good temper, and of decency of conduct is, in most cases, of far more consequence to the child than that of reading

and writing, and the ability to speak clearly and nicely, and to use a knife and fork like other people, are for him invaluable accomplishments.

Thoroughness in everything, so far as it is practicable, is of the greatest importance. The mother must never acquiesce in the child's doing less than his best, because he is weakly. A habit of prompt obedience is, of course, of the greatest value. At the same time, especially in the case of the older children, it is well to remember that, as Miss Dearly says, "almost any work that is within the physical and mental capacity of feeble-minded children can be got from them by allowing them to do it as a favour, instead of imposing it as a duty."

In most mentally defective children the moral sense is much dulled, if not apparently absent. Even in comparatively low-grade cases, however, we may find some ideas of fairness, honesty, truthfulness, reverence, unselfishness, and affection, and these should be watched for and encouraged as far as possible. Occasionally the moral character is developed to a surprising degree.

INSTITUTIONAL TREATMENT.

The question whether the mentally deficient child should be kept at home, or sent to an institution to be with others of his kind, is at times a very difficult one. During the first six or seven years of life there can, of course, be no doubt that the advantages of a good mother's constant care outweighs all the benefits that an institution can offer; and in some cases the same may be said with regard to older children. Generally, however, if they are at all educable, there are strong reasons in favour of institution treatment as age advances. If the child has been carefully brought up, he will probably do no harm at all to his normal brothers and sisters; but this is not always the case. For himself, the routine of a well-conducted institution is generally far happier than life at home. For one thing, it is extremely discouraging for him, as he grows older, to be associated in his work and play with normal children. Not only are they apt to tease him, but the fact that by no effort can he do anything nearly as well as they do is very demoralizing for him. For another, the home surroundings of most mentally defective children are such that they cannot be allowed much freedom while living with their parents, and therefore even their limited capacities are apt not to get full scope. Further, in his home the child misses the great advantage and stimulus of society and healthy rivalry, unless he can associate with children who are somewhere about his own level, and this is only possible in an institution. For these reasons institution life is, for the majority of mentally defective children, much happier and more wholesome, as well as more instructive, than treatment at home.

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CHAPTER XVI

DISEASES OF MUSCLES

F. E. BATTEN

CONGENITAL DEFECTS OF MUSCLES.
 MYOTONIA OSTEOMALIA.
 MYOTONIA FIBROSA.
 MYOTONIA.

MYOPATHY.
 MYOTONIA CONGENITA.
 INHERENT MUSCULAR PARALYSIS AND CON-
 TRACTION.

CONGENITAL DEFECTS OF MUSCLES.

INTRODUCTION.—Children are from time to time seen in whom there is congenital absence of a muscle or part of a muscle. The condition is not associated with any form of progressive weakness, and frequently does not give rise to any disability. The muscle which is most frequently absent is the lower portion of the pectoralis major. Bing collected 102 cases in which some portion of this muscle was absent, as well as 18 cases in which the trapezius was absent, and 16 in which the quadriceps was absent. The absence of the lower portion of the pectoral is shown by asking the child to press the hands together whilst the elbows are abducted from the side. Instead of the lower border of the muscle running in an oblique line downwards, it runs transversely. This portion of the pectoral muscle may be absent on both sides; it must, however, be remembered that bilateral absence of the lower part of the pectoral is a characteristic feature in many cases of pseudo-hypertrophic paralysis.

The trapezius, serratus, quadriceps, are the muscles which, after the pectoralis, are most commonly absent. Bing has collected many cases of congenital defects of these and other muscles, and four cases in which the abdominal muscles were absent.

Congenital Absence of Abdominal Muscles is one of the most interesting groups of these cases. The absence gives to the abdomen a strange appearance; the whole surface of the skin is wrinkled, the abdominal wall extremely thin, and the child is quite unable to make any loud cry or forced expiratory effort. These children often live for many months and put on weight, but they usually succumb to the first attack of bronchitis owing to the difficulty of coughing. In association with this abnormality, other defects in the development of the ureter, bladder, and testes, have been shown to exist.

The abnormality is primarily a muscular defect, and not secondary to a nervous lesion; for it has been shown that the cells of the spinal cord are normal.

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FIG. 179.—CONGENITAL ABSENCE OF MUSCLES OF THE ABDOMINAL WALL: PHOTOGRAPH SHOWING THE FISSURES IN THE ABDOMINAL WALL AND THE DEFORMITY OF THE THORAX. (GARROD AND DAYLEN.)

MYOSITIS OSSIFICANS PROGRESSIVA.

INTRODUCTION.—The name "myositis ossificans" is applied to a condition in which there is a tendency for several muscles of the body to undergo ossification. It must not be confused with the condition of multiple exostoses.

The disease is one of early childhood. It often manifests itself in the first decade of life, and Garrod has described this condition in a child of five months. It does not in itself tend to shorten life, and may persist for many years, some patients having been afflicted with the condition for over forty years.

ÆTIOLOGY.—The cause of the disease is not known. It is often attributed to injury; but though a blow may be the exciting cause of a single centre of ossi-

calion, it can hardly be the cause of the universal ossification which occurs. The disease is probably an "inborn error of metabolism."

SYMPTOMATOLOGY.—The first symptom is usually a local swelling on the back, which is often attributed to injury. The swelling is situated in the muscles, is firm in consistency, sometimes painful, and the onset may be accompanied by fever. There is often more or less edema round the swelling, but the skin is not involved.

After a few days the swelling tends to subside, leaving a hardness in the muscles which may eventually become bone.

Garrod, who had the opportunity of watching carefully the development and course of these swellings in a child under his care, says that they were of various sizes; they were most numerous on the back, but were also situated on the front of the chest or on the limbs. The swellings formed round or oval bosses, very firm and elastic and with sharply defined margins; they were attached to the deeper tissues, and the skin was freely movable over them. They were not tender. From the base of some of the more defined swellings slender processes could be traced for some distance from the main boss. Swellings of a second kind appeared as diffuse thickenings of the soft parts, with indefinite outlines, and over these the skin was not movable. Some of the masses underwent fusion, a single boss becoming converted into two distinct bosses connected by an area of diffuse swelling. These swellings might last for nineteen to



FIG. 140.—MYOSITIS OSSIFICANS. BONY NODULES ON THE BACK, NECK, AND LIMBS. THE FEET SHOW MICRODACTYLY. (SIMPSON.)

thirty-seven days, and then completely disappear. After some weeks fresh attacks occurred, with further swelling in the muscles. Some of these did not subside, and left a residual hardness which eventually became bone. In the initial stages of the disease the bony tumours are at first small and multiple. They gradually enlarge, coalesce, and form long bony masses of irregular shapes. These masses may be independent of bone, but as the disease progresses they generally

become attached to bone, and give rise to a great limitation of movement. Whenever a bony growth is formed, it never disappears.

The disease may affect any part of the body, but usually begins in the muscles of the neck and back. It advances slowly; the shoulder-blades become fixed to the thorax, the neck becomes rigid, and finally the limbs, the masseters, and temporal muscles, become so fixed that the patient cannot eat. Such an advanced condition is rarely if ever seen in childhood.

Symmetrical deformity of the great toe has been present in many cases. The great toe is shorter than the second toe, and this deformity is due to an absence of the proximal phalange or a synostosis of the shortened phalanges of the toe. Shortening of the trunk has also been described.

PATHOLOGY.—This form of myositis is one which goes on to the formation of true bone. What the cause may be is quite unknown. It has been regarded in the nature of a new growth, but neither from a clinical nor pathological standpoint is such a view justifiable.

DIAGNOSIS.—In the earlier stages it is possible to mistake the disease for rheumatism or the effect of injury; but in the later stages, when bone is formed, no difficulty in diagnosis can arise. It must not be confused with that of multiple exostoses. The latter is a familial and hereditary disease, whereas there is no evidence that myositis ossificans is so. An X-ray photograph would be of service in the diagnosis of any difficult case, but care must be taken to distinguish the shadow of "multiple calcification in the subcutaneous tissue" from true ossification (Weber).

PROGNOSIS.—The disease always runs a slowly progressive course. Starting in childhood, it advances by acute exacerbations, followed by periods in which improvement occurs; finally, in adult life, leaving the patient in a rigid position, incapable of movement.

TREATMENT.—No means has been found of arresting the disease.

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WEBER: *Proc. Roy. Soc. Med., Clin. Sec.*, 1902, vi, 14.

MYOSITIS FIBROSA.

INTRODUCTION.—This is a rare condition in which a slow and progressive fibrous infiltration of the muscles occurs, similar to that found in myositis ossificans, but without ossification.

SYMPTOMATOLOGY.—The disease begins in early life, starting in the lower limbs, affects the sterno-mastoid muscles, and spreads to the muscles of the neck, back, limbs, intercostal and abdominal muscles. The following case of a boy, aged six years, who was under the care of the writer for many months, illustrates the leading features of the disease as well as the pathological condition.

This boy was well up till nine months old. His mother then noticed that the back was growing out and the legs were drawn up. The condition slowly progressed, so that the child became more and more bent up. When six years old the boy could sit up in bed with the legs flexed, the back curved, and the head bent forward on the chest, the face turned to the left owing to the contraction of

the right sterno-mastoid. The spine was flexed in a curved position, and could not be straightened. The abdominal muscles were extremely hard and contracted. The arms were fixed so that they could not be extended or adducted. Any attempt to move the limbs resulted in the moving of the body as a whole.

The boy was rigidly fixed in a flexed position, unable to move. The great toe was shorter than the second, third, and fourth toes (Fig. 141).

PATHOLOGY.—The condition depends entirely on a change in the muscles, no change being present in the central or peripheral nervous system. The muscles when removed from the body are firm and hard, and when cut across are found to consist of a tissue which grates under the knife and appears hard and white on section. In less affected portions the muscle tissue appears as reddish-yellow



FIG. 141.—MYOSITIS FIBROSA.

Note the curvature of the back, the flexion of the neck and limbs. The child lay constantly in this position, and any attempt at passive movement only resulted in movement of the child as a whole.

points on a white ground. Microscopical examination shows a mass of the interstitial tissue between the muscle fibres, which have in part undergone granular degeneration, in part simple atrophy. In the most severely affected portions the muscles are composed entirely of tendinous tissue.

The cause of this affection is quite unknown; in fact, the condition is so rare that little opportunity for investigation has presented itself.

DIAGNOSIS.—The clinical resemblance to myositis ossificans is sometimes very close, and it is only the complete absence of any evidence of bony formation within the muscle, as shown by X-ray, that justifies the diagnosis of myositis fibrosa. The condition might be mistaken for the later stages of myopathy, but in that disease the wasting of muscle is more general. To the *dissieie arthritica* of children the disease bears some resemblance, but the implication of the joints in that disease

serves to distinguish it from that under discussion. In cerebral diplegia there may be marked rigidity of the limbs and contraction of muscles, but the presence of well-marked signs of cerebral and spinal disease would serve to distinguish the two conditions.

PROGNOSIS.—The disease is generally slowly progressive. Some cases have been reported in which recovery has taken place.

TREATMENT.—Massage, passive movements, hot-air baths, and electricity should be tried. Recovery has been reported after this line of treatment.

REFERENCE.

BITTEN: *Chil. Soc. Trans.*, 1904, xxvii, 12.

POLYMYOSITIS—DERMATO-MYOSITIS.

INTRODUCTION.—This disease is an inflammatory condition of the skin, subcutaneous tissue, and muscles, often starting acutely, but tending to run a chronic course. It is rare, and but few instances have been recorded in childhood (Schüller and Batten).

ETIOLOGY.—The cause of the infection is unknown. It is often attributed to rheumatism, but has neither the clinical features nor pathological changes of



FIG. 142.—DERMATO-MYOSITIS; PHOTOGRAPH OF GIRL AGED NINE YEARS, SHOWING THE WASTED CONDITION OF MUSCLES AND THE GREATEST EXTENT TO WHICH THE ARMS AND LEGS COULD BE EXTENDED.

that disease. There is no evidence that it is due to syphilis, though it is possible that it may be due to a protozoan. The evidence of bacterial infection is at present negative.

SYMPTOMATOLOGY.—The disease is usually of acute onset, attended by swelling and redness of the skin, with pain and tenderness in the underlying muscles. The swelling of the skin gradually subsides, leaving the muscles hard and infiltrated.

The swelling of the muscles subsequently slowly subsides. Fresh attacks occur which eventually leave the skin and muscles in a more or less permanently indurated condition. There is no evidence of affection of the joints, and the heart is unaffected.

The disease tends to run a long course, with recurrent exacerbations. The skin becomes greatly thickened and loses its elasticity. No sign of visceral disease or affection of the nervous system is present, and the child may live for months in a wasted condition, with contraction of the limbs, eventually dying of some intercurrent disease, such as a *B. coli* cystitis.

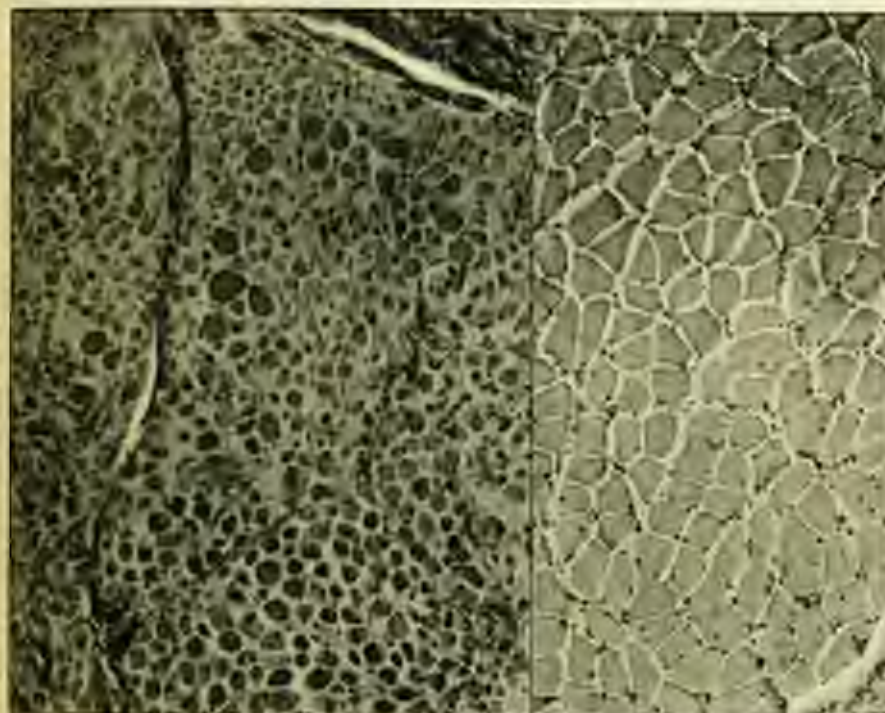


FIG. 145.—DERMATO-MYOSITIS: SECTION OF SKELETONAL MUSCLE OF THE ARM, SHOWING AFFECTION OF THE MUSCLE FIBRES, WHICH IN SOME PLACES HAVE ALMOST DISAPPEARED.

Note the small amount of interstitial tissue. A normal portion of the same muscle under the same magnification is seen on the right.

In other cases, after the disease has been in progress for some months, complete recovery takes place.

Pathology.—Fluid withdrawn from the muscles and the blood during life shows no change, and cultures made therefrom remain sterile. The superficial portion of the muscles and those parts which lie close to the intramuscular septa undergo the most marked atrophy and parenchymatous degeneration. The deeper muscle fibres remain comparatively normal. There is a slight increase of the intermuscular connective tissue. Small groups of lymphocytes ("lymphothaps") are found between the muscle fibres, and there is a considerable amount

of perivascular lymphocytosis. The walls of the vessels are thickened, and some have become obliterated. The thickening of the vessel walls is a most constant feature, and affects not only the vessels of the skin and muscles, but those of the visceral organs.

The skin shows atrophic changes, and the subcutaneous fat is replaced by fibrous tissue.

DIAGNOSIS.—In its early stages the disease most closely resembles acute rheumatism, but is distinguished therefrom by the absence of any affection of the joints and the presence of swelling and tenderness in the muscles. The tenderness which occurs in certain cases of poliomyelitis must be borne in mind, but in such cases there is paralysis, and in addition other symptoms pointing to affection of the nervous system.

PROGNOSIS.—The disease will sometimes run a progressive course which terminates fatally after an indefinite period. In other cases complete recovery occurs; but it is never rapid, and only takes place three to six months after the onset of the disease.

TREATMENT.—During the acute stage complete rest in bed is essential. Much can be done, by careful nursing, to relieve the pain. Salicylate, phenazon, and other coal tar products, are of service.

In the more chronic stages of the disease, hot baths, massage, and passive movements, are useful, and if contractions occur extension and splints should be used to prevent deformity.

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- BAYNES: *Proc. Roy. Soc. of Med., Neurolog. Sec.*, 1912, v. 805.
SCHEFFNER: *Festschrift des Kaiser-Wilhelms-Instituts*, 1903, p. 132.

THE MYOPATHIES (MUSCULAR DYSTROPHY).

INTRODUCTION.—Under this title are included those cases in which muscular weakness is the leading feature, such weakness being independent of a spinal lesion. The weakness may be attended by atrophy or dystrophy of muscles; it may be present at birth or may develop in infant or child life; is usually progressive, but may show remission; is unattended by pain or any alteration in sensation.

The cases which comprise this group differ considerably in their clinical manifestation, yet they possess the one characteristic feature—viz., progressive muscular weakness.

Various classifications of the different types have been adopted, but the following is suggested as applicable to the disease as it affects children. It must, however, be realized that the myopathies form one great class; and though for the sake of clinical description it may be convenient to divide them into "types," yet there is no hard-and-fast line of division between the various types, and cases occur not infrequently which possess the features of two or more types.

The following classification is suggested:

1. Simple atrophic type (Erb, Leyden-Moebius), and with this should be included the condition described by Oppenheim under the name "*myotonia congenita*," or *myotonia congenita*.
2. Pseudo-hypertrophic type.
3. The juvenile type (Erb).
4. The facio-scapulo-humeral type (Landouzy-Dejerine).

Ætiology.—The cause of myopathy is unknown. Certain facts in connection with its occurrence are, however, of considerable interest.

The pseudo-hypertrophic form is both an hereditary and familial disease—that is to say, it is transmitted from parent to child through several generations, and will affect several members of a childship. Females are rarely affected, but the disease is usually transmitted by the female.

The *homo-scapulo-humeral* form, again, is both an hereditary and familial disease; it affects males and females alike, and is transmitted both by males and females. The family tendency is by no means so striking in this type as in the pseudo-hypertrophic type. The juvenile form shows the same tendency.

In the simple atrophic type the family character is not marked, and in the majority of recorded cases no family tendency can be traced. This form tends to affect males and females alike, and, owing to its crippling effect, there is no evidence that the disease is transmitted from one generation to another.

The ages at which these various types show their first symptoms differ considerably. In the simple atrophic type the weakness may be noticed in the first



FIG. 144.—MYOPATHY: SIMPLE ATROPHIC TYPE (SAME CASE AS FIG. 143), SHOWING THE "FOLDED-UP" POSITION CAPABLE OF BEING MAINTAINED Owing TO THE MARKED HYPOTONIA OF MUSCLES.

few months of life, though the difficulty which the child has in sitting up or in standing are often the first symptoms for which it comes under observation. In the *homo-scapulo-humeral* type the weakness of the face is often observed shortly after birth or in the first months of life, though the muscular weakness of the arms and legs is not noticed till years later, often after the first decade of life.

The age of onset of symptoms in the pseudo-hypertrophic form is usually about five or six, but a history of the child being late in learning to walk, or never having walked well, is not unusual.

In the juvenile type the symptoms usually manifest themselves about the eighth or sixth year of life.

1. **The Simple Atrophic Type ("Myotonia Congenita," Oppenheim).**—This type of myopathy is characterized by smallness, lack of power, and loss of tone in all the muscles of the body, without localized atrophy or hypertrophy of individual muscles or groups of muscles. The disease starts in early infancy, and advances but slowly, for the child may, as development takes place, learn to sit up, and possibly to stand, with support. When seen in the earlier months of life, the child is

able to move the arms and legs in all directions so long as no great power is required. He will move the head in all directions, but on account of the weight of the head he often fails to hold it up. Owing to the marked loss of tone in the muscles, the arms



FIG. 145.—MYOPATHY: SIMPLE ATROPHIC TYPE, SHOWING A POSITION FROM WHICH THE BOY IS UNABLE TO RISE.



FIG. 146.—MYOPATHY: SIMPLE ATROPHIC TYPE.

The frog-child walked in the position seen in the photograph. He was unable to assume the erect attitude, owing to the contraction of the flexors. Note the general atrophy and the long feet and hands.



FIG. 147.—MYOPATHY: SIMPLE ATROPHIC TYPE.

Note the general wasting of muscles, and the long feet and hands. This child had never been able to walk, and owing to contraction of the ham-strings the legs could not be fully extended.

can be placed in most unusual position in relation to the trunk, and frequently even be folded behind the back. The legs can be flexed on the trunk so that the feet lie over the shoulders. The feet and hands often appear unduly long, and, owing to the flexibility of the muscles, can be bent to unusual angles on the legs and forearms.

All movements can be performed, but in a feeble manner. The movements of the face are usually perfectly good, but some cases have been described in which the face has been affected.

The child learns to talk at the usual age, and intellectually is often in advance of his years. These children frequently adopt strange manoeuvres for getting



FIG. 148.—*Myomaxillary Scurvy Anomalous Type* (Lafont-Martin Type).

Note the general weakness and wasting of muscles, as shown by the various postures. (Reproduced with Professor Handshuter's kind permission.)

about; sometimes they roll round on the long axis of the body in order to cross the floor, or other children assume the "frog" position, as seen in Fig. 146.

If these children are placed on the floor and asked to stand up, they are frequently unable to do so without the help of a chair; sometimes they go through the manoeuvres adopted by the pseudo-hypertrophic, sometimes they can raise the pelvis, but are unable to erect the trunk on the thighs (Fig. 145).



FIG. 149.—MYOPATHY: PSEUDO-HYPERTROPHIC TYPE.

Typical case illustrating the relative large calves as compared to the thighs. (With Dr. Collier's kind permission.)

In the later stages of the disease some contraction of the flexors occurs, so that the legs cannot be fully extended.

Cutaneous sensation is usually perfect. The deep reflexes are very much diminished or abolished. The control of the sphincters is perfect. The electrical reaction of the muscles is such that a very strong faradic current is needed to obtain a contraction; but this strong current does not seem to cause any pain, whilst the galvanic current gives rise to pain.

Into this group are placed also cases which were described by Leyden and Morhiss, and are sometimes known as the "Leyden-Morhiss type." These authors emphasize the familial characters of the disease, as distinguishing them from other cases, but it has been shown that this tendency cannot be held as a distinctive feature.

The cases which have been described by Oppenheim under the title "myotonia congenita" also belong to this group, possessing all the characteristic features. Myotonia congenita has been described as an "entity," but clinical and pathological evidence assigns these cases to the myopathies.

2. The Pseudo Hypertrophic Type.—This is the best known and most easily and generally recognized form of myopathy, since it possesses certain very distinctive features.

The usual history obtained in such cases is that the child was late in learning to walk, and has never been able to go up stairs well; on the other hand, cases are met with in which the history is that the boy was quite well, active, and strong, till the onset of the weakness, when five to six years old. It is generally recognized that this form of paralysis may commence in childhood, adolescence, or even adult life, but the history as given above is the common one. It not infrequently happens that, if the younger members of a family are examined, they are found to show signs of the disease which have escaped casual observation.

SYMPTOMATOLOGY.—Children affected with this disease frequently fall down and may have considerable difficulty in rising. This type is characterized by weakness of groups of muscles, with hypertrophy and wasting of certain other muscles. The muscles commonly hypertrophied are those of the calf, the glutei, the infraspinati. The muscles commonly atrophied are those of the thigh, the



FIG. 150.—MYOPATHY: Pseudo-Hemiparesis Type.

A series of photographs illustrating the exercises adopted to attain the erect position. (With Dr. Cox's kind permission.)

lower portions of the pectoralis major, the biceps, and latissimus dorsi. Although the above are the commonest muscles to show atrophy and hypertrophy, yet in individual cases other muscles are found to be affected; the masseter, tongue, the vastus internus, erector spinae and the triceps, are sometimes enlarged.

The appearance of a child with well-marked pseudo-hypertrophic paralysis is striking. When he stands there is marked lordosis of the lumbar spine, the shoulders are held well back, and the scapulae usually project somewhat. The large size of the buttocks often makes the lordosis appear more accentuated than it is in reality.

The large size of the calves is in contrast to the relative wasting of the thighs, and when the feet are placed together a space exists between the adductors which is not present in the normally developed fat child. The size of the calves is also in contrast to the usually poor development of the muscles of the shoulder girdle and to the wasted condition of the pectoralis major. When walking the child waddles, swinging the trunk from side to side like a child with congenital dislocation of the hip. This waddle has a double object—first, in order to get the centre of gravity directly over the hip-joint, and, secondly, to get over the difficulty of raising the thigh. In walking the trunk and head are usually held very erect, for unless this position is maintained the balance of the child is liable to be upset. The feet as a rule can be placed flat on the ground, but in some cases a degree of cavus may be present, and a talipes equinus, so that the child walks on tiptoe.

If an attempt is made to get up stairs, a considerable amount of difficulty is experienced, owing to the weakness of the flexors and extension of the thigh on the trunk. Without the help of the balusters the child as a rule cannot mount the stairs. With the baluster on the left the child raises the right leg on to the stair; the right hand is then placed on the thigh just above the knee, and by pulling with the left hand and straightening the knee with the right the trunk is raised on to the first step, and the manoeuvre repeated.

In sitting, the lordosis, which is present on standing, passes into a kyphosis, the back being bent according to the degree of weakness of the dorsal muscles.

If an attempt is made to lift up the child by placing the hands in the axillae the looseness of the shoulders is a striking feature. When placed flat on his back on the ground and asked to rise, the child goes through a definite series of movements to accomplish the act. The normal child when asked to get up, sits up, flexes the legs under the trunk, and rises into the erect position by the extension of the legs and thighs. The pseudo-hypertrophic child rolls upon his face on his long axis; he then draws the legs up under him, keeping his head on the ground. He now extends the legs, and, by working the hand backwards along the floor, brings the trunk to above the thighs. The trunk has now to be brought into the erect position; this is done by bringing the hands on to the legs and then on to the thighs and thus climbing up the legs, pushing the trunk into the erect position. With a little swing of the head the shoulders are thrown back and the trunk is balanced on the thighs, the lordosis appearing in the place of kyphosis.

The above description applies to a marked case of the disease. There are, however, on the one hand, the early and slight cases which may show but few of the above signs, except, possibly, the difficulty in rising from the ground; and, on the other hand, the advanced cases in which the paralysis is so extreme that but few movements are possible, and all, or nearly all, the muscles have passed into a wasted condition, contraction and deformities being usual at this stage.

In the earlier stages of the disease the deep reflexes are preserved; the knee

jerks may in some cases appear unusually brisk, and in cases in which the *extensor internus* has been hypertrophied they may be very active. The knee-jerks commonly disappear before the ankle-jerks. The superficial reflexes are preserved, and plantars are present and have the normal characters. Sensation is usually perfect to all forms of stimuli, and, although sensory changes have been described in connection with pseudo-hypertrophic paralysis, such alteration must be regarded as exceptional.

The control of the sphincters is as a rule perfect. Fibrillary tremors have been described in association with myopathy, but their presence is generally regarded as diagnostic of a myelopathic rather than a myopathic affection. The fact that modern pathology recognizes that some spinal cord changes may be found in cases of myopathy would also make one prepared to accept the occurrence of fibrillary tremors in some cases of myopathy.

The affection of the muscles of the face is most unusual in cases of pseudo-hypertrophic paralysis. Such cases do, however, occur, and they form the connecting link between this group and the facio-scapulo-humeral. The presence of the facial weakness is so characteristic a feature of the facio-scapulo-humeral group that cases showing this feature should be regarded as aberrant types of that group rather than as aberrant types of the pseudo-hypertrophic group.

3. Juvenile Type (Erb, "Scapulo-Humeral").—This type was first described by Erb, and it derives its name from the period of life—viz., early youth—at which it most commonly starts. The disease may be present in childhood, and manifest itself about the sixth to seventh year of life. This form is both hereditary and familial, and affects males and females alike. It is closely related to the facio-scapulo-humeral type—Landouzy-Dejerine—but differs from that in that the face is not affected.

The most prominent feature of this disease is the "winging" of the scapula, and the difficulty of raising the arms above the head. The winging of the scapula is dependent on the weakness of the serratus magnus and trapezius muscles, and the difficulty in raising the arms is dependent on the weakness of the lower portion of the trapezius. Some of the muscles affected are atrophied; others are hypertrophied. The muscles which usually show atrophy are the upper portion of the pectoralis major and minor, the latissimus dorsi, the lower part of the trapezius, the serratus magnus, and the rhomboids. The deltoids, supra- and infra-spinati,



FIG. 111.—MORRIS: Erb's Juvenile Type.

Note the winging of the scapula, due to weakness of the serratus magnus and trapezius muscles; the other muscles of the body were normally developed.



FIG. 132.—MYOPATHY: FACIO-SCAPULO-HUMERAL TYPE (LANDOUZY-DEJERINE).

The photograph shows some of the typical features of the disease—i.e., the weakness of the facial muscles, the weakness and winging of the scapula, the lordosis produced by weakness of the abdominal and lumbar muscles. (With Professor Henshaw's kind permission.)

usually appear large in comparison to the wasted muscles, and may be actually hypertrophied.

As the disease advances, the muscles of the upper arm become affected and show atrophy, those of the forearm remaining normal. Weakness then manifests itself in the muscles of the pelvic girdle, and there is wasting of the quadriceps extensors and adductors of the thighs and weakness of the psoas and iliacus group of muscles, so that the child has difficulty in flexing the legs on the abdomen. The muscles of the leg below the knee remain well developed. The degree of atrophy to which a patient may advance, and yet have power to get about, is extraordinary. The face and neck muscles do not become affected. The presence of the deep reflexes depends upon the stage of the disease and the condition of the affected muscles. In the early stages of the disease the knee-jerks, ankle-jerks, and the arm-jerks, are present; in the late stages all these disappear.

This type of myopathy is, in the experience of the writer, in children the least common of the four types here described. Only a few cases in children under twelve years of age have come under his observation, and in none of these was the disease advanced, nor did it make rapid progress.

Trevelyan gives a good account of the disease in a family of eight, in which the father and three children were affected, the first symptoms being noticed in the youngest child when eight years old.

4. The Facio Scapulo-Humeral Type (Landouzy-Dejerine).—This type is characterized by a weakness of the muscles of the face and winging of the scapula, due to weakness of the muscles of the shoulder girdle. The weakness of the face is especially marked in the orbicularis oris and palpebrarum. It may be present at birth, and is sometimes the only sign of the disease for many years. Other features are the weakness and wasting of the muscles of the shoulders and pelvic girdle, and wasting of the muscles of the upper arm and thigh.

The disease is both hereditary and familial, males and females being alike affected.

As seen in infancy, it often escapes notice, and the symptoms which the child presents—viz., the difficulty in sucking the breast or bottle, and the fact that it does not close its eyes when asleep—are put down to general weakness. In children of two to three years old, however, the lack of facial expression is very striking. They are unable to raise the eyebrows, unable

to close the eyes tightly, unable to blow out the cheeks or blow a "penny trumpet." The child smiles with its "eyes," but the face remains motionless. The muscles of the eyeball, tongue, palate, and those of deglutition and phonation are normal, and the movements of the head and neck are also good.

A child with such an affection of the face may present no other symptoms of the disease, and no further weakness may manifest itself till between twenty to thirty years of age. On the other hand, the weakness of the muscles of the shoulder girdle may show itself at an early age by the winging of the scapulae. The muscles first affected are the trapezi, the serratus magnus, then the pectorals, latissimus dorsi, biceps, triceps, leaving the deltoid, infra- and supra-



FIG. 123.—MYOPATHY: Pseudo-Scapulo-Humeral Type (LANDOLZY-DEJEVINE).

The photographs illustrated the marked weakness of the orbicularis palpebrearum and risorius muscles. This weakness produces to a face devoid of expression. (With Professor Harnshtalter's kind permission.)

spinal and forearm muscles relatively intact. The weakness then manifests itself in the pelvic girdle, and is attended by wasting of the glutei and muscles of the thigh.

Children with this form of disease are often able to get about fairly well. They are able to perform most movements, and do not experience the difficulty of going upstairs or in getting up from the floor that is seen in some of the other types of myopathy.

This type of myopathy is one of the most slowly progressive, and often seems to remain stationary for years. The case on which Landolzy and Dejevine based their original description died at the age of forty-five, and the disease was known to have existed for over thirty years.

The illustrations taken from Harnshtalter's excellent paper on the myopathies of childhood illustrate the clinical features well.

PATHOLOGY.—The characteristic feature of the pathology of the myopathies is that the nervous system shows no change, whilst the muscular tissue shows advanced degeneration.

The above statement is perfectly true for most cases of myopathy; it is not, however, universally true, and cases of myopathy have been described in which changes have been found in the peripheral nerve and the cells of the ventral horns of the spinal cord. These changes are, however, always slight as compared to the marked change that is found in the muscular tissues, and it is quite reasonable to regard such changes as have been found as secondary to the changes in the muscles. The question is too long to be argued in a textbook of this nature, but is admirably dealt with in a paper by Gordon Holmes.

In the case on which Landouzy and Dejerine based their description of the type which bears their name, no change was found in the nervous system, although the disease had existed for thirty years.

On examination of the nervous system of cases of "myotonia congenita" it has been found to be normal.

With regard to the changes in the muscles, it is unnecessary to consider the various types separately, for the changes are similar in all the types, and from the appearance of a muscle it would be impossible to say from which type it was taken. On removal from the body their appearance may differ. There are the large hypertrophied muscles which consist of masses of fat lying between layers of fibrous tissue, in which are embedded small groups of apparently more or less normal muscle fibres; and there are the atrophied muscles, only represented by a thin layer of fibrous tissue, which feel very tough and firm, and on section cut like fibrous tissue.

Certain muscles—for instance, the glutei and gastrocnemii—almost always show fat deposition; whilst others, such as the pectorals and latissimus dorsi, always show atrophy without any fat deposit.

On microscopical examination of transverse sections, the muscle fibres are seen to have lost their polygonal outline. Some of the fibres are enlarged, have a diameter of twice, and sometimes three times, the normal, and others of the fibres that are atrophied having a diameter of a third or a sixth of the normal size. All these fibres are circular or oval on section and are surrounded by fibrous tissue, and often separated from one another by fat. Some of the fibres show a central nucleus and vacuolation. The nuclei of the sarcolemma sheath of the muscle fibres are relatively or actually increased in number. The muscle fibres as a rule preserve their transverse striation until atrophy has proceeded to an extreme degree. There is always a considerable thickening of the walls of the bloodvessels in the muscles. The intramuscular nerves are usually remarkably well preserved when the atrophic condition of the muscles is taken into consideration. The appearance of the muscle spindles in myopathic muscles is also a striking feature, for these structures do not seem to undergo atrophy, and appear increased in numbers in relation to the size of the muscle.

DIAGNOSIS.—Two points arise in the diagnosis of cases of myopathy: First, the diagnosis of the disease from progressive muscular atrophy of spinal origin; and, secondly, the diagnosis of the various types of myopathy from one another.

The diagnosis of myopathy from a progressive muscular weakness of spinal origin may be a simple matter, or, on the other hand, may be a matter of the greatest difficulty.

The simple atrophic type of myopathy bears a very close resemblance to the type of muscular atrophy described by Werdnig and Hoffmann, due to a spinal lesion. The loss of tone in the muscles is very similar. In the myopathic disease, however, the paralysis is much less than in the spinal disease, whereas the loss of tone is greater in the myopathic disease than in the spinal disease.

Both these diseases occur in infancy or early childhood, and both are progressive. Both may affect more than one member of the family. No cases illustrate the difficulty of diagnosis better than those described by Finkelburg, which on clinical grounds would be assigned to the progressive muscular atrophy of spinal origin, but on pathological grounds are shown to belong to the myopathic group. In older children the presence of spinal caries and psoas abscess must always be borne in mind, for such children often have difficulty in going upstairs, raise themselves from the floor with difficulty, and climb up their legs, etc. There is progressive weakness. On examination, no rigidity of the spine may be detected, lateral flexion and extensor movements of the spine being normal. Such children often, however, object to sit, and will rather stand or lie. If sat on the floor and bent forward, they evince signs of pain. The myopathic child can be bent forward easily, and will often lie with the head resting on its leg with the greatest comfort. Other lines of investigation will be adopted in a doubtful case, and an X-ray photograph will often reveal a caries which cannot otherwise be detected.

Cases have come under the observation of the writer which have presented the above symptoms, but in which the most careful investigation has failed to reveal any disease of the bodies of the vertebrae or of the spine. Such cases have, after a few weeks or months of treatment, entirely cleared up, and the only diagnosis arrived at is an inflammatory condition of the intervertebral cartilages and joints. A slowly progressive symmetrical weakness and wasting of muscles not corresponding to those supplied from a spinal segment or nerve trunk, the absence of any sensory impairment, and the absence of fibrillary contraction, are the signs on which the diagnosis of a myopathic affection should be based.

It must, however, be borne in mind that cases of myopathy have been described in which not only fibrillary tremors, but sensory changes have been present.

The diagnosis of the various types of myopathy from one another is not, as a rule, a matter of any difficulty, when the features of the various types are borne in mind. Each type has certain characteristic features, and the presence of these in any given case indicates the type to which the case should be assigned—e.g., the affection of the muscles of the face would indicate the "facio-scapulo-humeral type," and the enlargement of the calves and buttocks would indicate the pseudo-hypertrophic type. It is fully admitted that cases of myopathy occur which cannot be assigned to a "type," and on this ground certain writers have suggested that all cases of myopathic atrophy should be classed together.

Prognosis.—The prognosis differs considerably in the various types described. In the simple atrophic type the children usually die of some intercurrent disease before the age of twelve. Some of the cases show improvement in their muscular power as development takes place, and this has been urged as a point of distinction of the cases known under the title "myotonia congenita" from the true myopathic cases. No case of myotonia congenita has yet been reported in which recovery has taken place. The disease is usually very slowly progressive, and a child who was seen after an interval of six years showed but little, if any, increase of weakness.

In the pseudo-hypertrophic type the disease is almost always slowly progressive,

the loss of power being so great after the disease has lasted for six to seven years that the child can no longer walk. Life may be prolonged to the end of the second decade, and may extend even into the third decade, but is usually terminated by some pulmonary affection before that time.

In the "juvenile" type the progress of the disease is much less rapid, and the patient may go on for many years with but little increase of the weakness. Erb states that he has seen cases in which the disease has become stationary and has not advanced in later life, and he has recorded one case in which complete recovery took place, and that was in an English child who presented typical signs of the disease when seven years old, and in whom gradual recovery took place, so that, when ten years old, the child was quite healthy, and six years later was still in good health and perfectly strong.

In the "sacro-scapulo-humeral" type the progress of the disease is very slow. A child who presents the typical weakness of the face at birth may show no other sign of the disease till the second, third, or even the fourth, decade of life. On the other hand, the weakness of the muscles of the shoulders and hips may develop during the first decade, and almost completely incapacitate the patient during its second decade of life.

Considering the myopathies as a whole, it may be said that the prognosis as to recovery is more favourable in the juvenile type than in any of the other types. With regard to life, the prognosis in the pseudo-hypertrophic form is less favourable than in other types.

TREATMENT.—Various animal extracts have been tried in cases of myopathy without any material benefit. The extracts of thyroid and pituitary gland have been given, and some improvement is stated to have taken place in one case after the last named.

Injections of diastysin have been given over a considerable period without any effect.

Massage and exercises, with galvanism and faradism to the spine and affected muscles, together with the use of baths, was the treatment under which recovery took place in the case recorded by Erb. The general management of these cases is important. Exercise, both active and passive, and massage should be carried out daily, in order to prevent the development of contraction, and to keep the muscles in as good a condition as possible. These patients should be encouraged to walk, even if only for a short distance, daily, as long as it is possible for them to get about, for when once they take to bed contraction occurs, and the loss of power becomes more marked.

In some cases it is advisable to recommend tenotomy, especially when the contraction is such that it prevents the patient from walking.

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MYOTONIA CONGENITA (THOMSEN'S DISEASE).

INTRODUCTION.—This is a rare disease in adults, and only a few cases have been described in children. The characteristic feature of the disease is that, upon attempted execution of a voluntary movement, the muscles concerned contract, but slowly, and also relax but slowly. The result of this slow contraction and relaxation is that movements cannot be quickly performed. On repetition of the movement, however, it is more quickly performed, and after it has been carried out several times it is then easily and quickly performed.

ÆTIOLOGY.—The condition is familial, the disease passing from parent to child, males and females being alike affected.

In many cases the condition is known by the patient to have been present in childhood, but little or no notice has been taken of the affection till the later years of life.

SYMPTOMATOLOGY.—Difficulty in sucking has been observed in an infant a few hours after birth. This symptom was noticed in a case recorded by Friis, the father and one other child being affected with the same disease. The child was able to swallow perfectly well. Some stiffness of the limbs was noticed when the child began to walk, and some hypertrophy of the muscles was also present. In the case of a boy described by Meera, when starting to walk he did so on tiptoe, but after walking a few steps his gait became more normal. The affection was in this case at its maximum between the ages of three to six, and at the age of ten the disease was present in its typical form.

The slow contraction of muscles is usually well exhibited in the hand-grasp, the grasp being slowly executed, and relaxation taking several seconds.

The physical examination of the nervous system reveals nothing abnormal, but the electrical examination of the muscles shows a long-drawn and slow contraction to faradism.

PATHOLOGY.—The only change found in these cases is enlargement and feeble striation of the muscle fibres.

DIAGNOSIS.—The diagnosis of myotonia congenita should only be made in those cases in which the symptoms are present in a characteristic form. Cases of infantile hemiplegia and diplegia exhibit hypertonia, movements are slowly performed, and relaxation of contracted muscles slowly takes place; but in such cases the increased tone is constantly present, and does not pass off after repeated movement. Such cases often exhibit loss of power and irregular movements, neither of which symptoms occur in Thomsen's disease. The occurrence of increased muscle tone on one side of the body only would suggest the involvement of the pyramidal path in some part of its course, but it must be borne in mind that increase of tone depends upon the failure to control normal efferent impulses, and is not necessarily associated with a lesion of the pyramidal tract.

PROGNOSIS.—The disease persists throughout life, and is not appreciably affected by any course of treatment.

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ISCHEMIC MUSCULAR PARALYSIS AND MUSCULAR CONTRACTION.

INTRODUCTION.—Ischemic muscular paralysis and muscular contraction is produced by a primary necrosis of the muscular substance due to the cutting off of the blood-supply.

Following the necrosis there is an increase of the connective tissue between the muscle fibres, which leads to a shrinking and shortening of the muscle.

The condition is commonly the result of injury to the limb, or to too tight application of splints or plaster of Paris bandages.

SYMPTOMATOLOGY.—The hand and forearm are most commonly affected. The skin is blue and cold, has a glossy appearance, and frequently is the seat of superficial ulceration. The wrist is usually flexed, owing to the contraction of the flexor muscles. The fingers are bent into the palm of the hand.

There may be complete loss of power in all the muscles below the elbow. The arm is thin and wasted. Cutaneous sensation is sometimes lost over the whole of the peripheral portion of the limb, but in other cases, although the paralysis of the muscles may be profound, yet sensation may be relatively good. The limb may be excessively painful and tender on pressure. The affected muscles fail to react to electrical stimulation. The deep reflexes of the affected limb are lost.

DIAGNOSIS.—The diagnosis in well-marked cases is not difficult, but in the slight cases difficulty may arise in distinguishing the condition from a monoplegia of cerebral origin, such as occurs in some cases of infantile hemiplegia. The normal electrical reaction and the increased reflexes in the latter condition would serve to distinguish it from the former.

A lesion of or injury to one or more of the peripheral nerves may give rise to a somewhat similar symptom-complex, but the grouping of the affected muscles in association with anesthesia in the area of supply of a peripheral nerve will serve to distinguish such a lesion from the diffuse and widespread paralysis caused by ischemia.

PATHOLOGY.—The cutting off of the blood-supply from a muscle rapidly gives rise to loss of function, and this is soon followed by destructive changes in the muscles, from which recovery does not take place. The nerves and nerve termination are damaged at the same time as the muscles, but the sensory fibres of the nerves are less readily affected than the motor fibres, and more readily recover their functions.

In the later stage of the disease fibrous tissue takes the place of the damaged muscular tissue.

TREATMENT.—Warmth, massage, passive movements, and hot baths may in the earlier stages of the disease do much to relieve the condition and assist recovery. The limb should be placed in a well-fitting splint to prevent the occurrence of deformity.

In the late stages little can be done to relieve the condition.

CHAPTER XVII

DISEASES OF BONES AND JOINTS

H. D. ROLLESTON

DISEASES OF BONES

ATROPHYPLASIA.

SECONDARY CRURIO-CLAVICULAR DYSOSTOSIS.

CONGENITAL ELEVATION OF THE SCAPULA.

FRAGILITAS OSTIUM:

1. OSTIOGENESIS IMPERFECTA.

2. MORBIDUS OSTIUM.

TUBERCULOUS DISEASE OF BONES AND JOINTS.

STYLOIDED DISEASE OF RIBS.

CRANIOTOMY.

BOWING OF THE SKULL.

LEONTIASIS OSSEA.

NEW GROWTHS OF BONE.

ACHONDROPLASIA.

SYNONYMS.—Chondrodystrophia fetalis; Micromelia.

DEFINITION.—A disease usually of fetal life, characterized by shortness of the long bones, trident hands, and large head. It depends on interference with endochondral ossification, especially at the extremities of the long bones.

Although earlier observers, such as Müller (1866) and Urtel (1873), recognized that the disease was not ordinary rickets, Parrot (1878) first definitely separated it from rickets and from cretinism, and called it "achondroplasia." The striking characteristics of achondroplasia have been reproduced in art, and can be recognized in the representations of the Egyptian gods Bes and Ptah, and of various Court dwarfs. There is evidence that achondroplasia has existed for at least 5,000 years in Egypt (Raffet).

ETIOLOGY.—Heredity.—Delivery in achondroplastic women usually necessitates Cæsarian section, and so interferes with the propagation of an achondroplastic race. The disease may be handed down through the male line, and its occurrence in males in three generations has been recorded (Peloquin, Porter). A pedigree given by Retz and Weil showed achondroplasia in the first and fourth generations, and partial achondroplasia in the second and third generations on the male side. The disease may also be familial, but of twins one may be achondroplastic and the other normal. Certain species of animals, such as dachshunds and Aneon sheep, have been regarded as examples of achondroplasia fixed by inheritance. But the "hulldog" calves also described as achondroplastic have been shown by Seligmann to be cretins. It is often stated that the disease is more frequent in females than in males, and of Kassowitz's collection of 29 cases 4 only were males; but in 100 other collected cases I found 55 males and 47 females.

PATHOGENY.—From a Mendelian point of view there is a loss of the determinants, in part, which govern the length of the bones developing in early endochondral ossification—namely, those of the limbs, the osseous innominate, the ribs, and the basi-occipital (Berry Hart). Endochondral ossification occurs after the sixth month of intra-uterine life, and the bones thus laid down—viz., the vertebrae, carpus, tarsus, sternum, patella, and costal cartilages—are unaffected. It has therefore been thought that the disease runs its course between the third and sixth months of intra-uterine life. Various hypotheses have been propounded. It has been thought that the condition is a sudden alteration in type of the species—a reversion to the ancient pygmies (Agouti). Several writers have considered that achondroplasia is the outcome of a maternal toxemia, which acts on cartilage either directly, or indirectly through the ductless glands, such as the thymus or thyroid, or through the nervous system. Shattuck regarded it as a paracretinoid state.

There is no evidence that the condition is determined by tuberculosis, syphilis, alcoholism, or consanguinity in the parents. Disease of the placenta has been recorded in a few instances, and it has been argued that achondroplasia is due to high amniotic pressure (Janet), but this has not been proved. The disease is not absolutely congenital, for it may advance after birth, and in rare instances it has been noticed to begin after birth (Schorr). The relation of the disease to rickets has been much discussed, and formerly the condition was called *fatal rickets*.

MORPH. ANATOMY.—(1) In stillborn achondroplasia the long bones (except the clavicles, which are usually normal), especially the humerus, femur, tibia, and ulna, are shortened, sometimes to half their normal length; the shafts are firm, show excess of periosteal bone, and instead of being smooth present prominent ridges for the attachment of muscles. The normal curves of the bones are exaggerated, and symmetrically so. The diameter of the bones is about normal, but the medullary canal may be obliterated. The epiphyses are of normal size (Dixon), but the shortening of the shaft produces a striking deformity, which has been compared to an hour-glass (Emerson). They are unusually vascular, and may be soft. The diaphyses of the long bones are expanded at their ends, and fit in a cuplike manner over the heads of the epiphyses; this accounts also for the beading of the ribs.

The skull is enlarged, suggesting hydrocephalus, the fontanelles may be widely open, and there are bosses on the frontal and parietal bones. The most important change is synostosis of the basi-sphenoid and occipital, which leads to shortening of the base of the skull and to a rigid, funnel-shaped foramen magnum. The viscera and the thymus and thyroid do not show any constant changes. Hydranencephaly may occur. The skin may be thickened.

(2) In older achondroplasia the changes are on the same lines as in the newly-born. The long bones of the limbs, especially the humerus, are very short, but the clavicle is as long as the radius. The scapula is dwarfed, and the glenoid fossa is proportionately too small for the head of the humerus. Two groups of cases have been described on the basis of the state of the epiphysis: (a) Those in which union with the shaft is much delayed, so that it is still ununited in middle life; (b) those in which ossification is much advanced. But in both instances dwarfing of the bones results. The sacral promontory projects into the pelvis and narrows the true conjugate, which is seldom more than 2½ inches.

MORPH. HYSTOLOGY.—The microscopic changes in the epiphyseal cartilages are the diagnostic feature of the condition. There is aplasia of chondral ossification



FIG. 154.—DRAWING OF SKELETON OF ACHONDROPLASTIC INFANT, LONG IN SC. GEORGE'S HOSPITAL MUSEUM. NO HISTORY.
 Notice the infant hands and the shortening of the long bones, except the clavicles, which are normal.

The cartilage is strikingly abnormal, the cells are few and irregular, and the intracellular substance fibrous or fibrillar. The cartilage is vascular, and is vascularized by the numerous vessels. A most important change is the presence of a transverse layer of vascular fibrous tissue which passes in from the pericentrum and divides the zone of ossifying cartilage from the remainder of the epiphyseal plate. Kaasman describes three forms of achondroplasia: (1) Hypoplastic, in which proliferation of the cartilage cells is diminished and their columnar arrangement defective, the epiphyses being of normal size; (2) hyperplastic, with excessive proliferation of the cartilage cells and defect in their columnar arrangement, the epiphyses being enlarged; (3) malacia, with softening of the cartilage, defective columnar arrangement of the cells, and a gelatinous and vascular matrix.

CLINICAL PICTURE.—The most striking feature is the contrast between the stunted limbs and the trunk, which is normal in length, though narrow. Hence

the shortness of the body as a whole is not so obvious in the sitting posture. The hands may not extend below the great trochanters, and, from the shortness of the lower extremities, the subjects can often when standing stoop down and kiss their toes. The shortening of the limbs (micromelia) is greater in the proximal segment (for example, the upper arm) than in the distal segment (such as the forearm); this is spoken of as "rhizomelic micromelia." There may be furrows around the limbs, as if the soft parts were too large for the bones, and the transverse diameter of a segment may almost equal its length. The muscles of the limbs are well developed, the strength is often above the average, and the subjects resemble small athletes. In the erect position the buttocks are prominent, and there is lordosis; the gait is waddling, and walking is often a late accomplishment. The long

bones, which are stunted and thick, may show sharp curves at the junctions of the shaft and epiphyses. The head of the femur is much nearer to the knee-joint than normal; this is a persistence of the normal condition of the eighth month of intra-uterine life. In some cases extension at



FIG. 155.—ACHONDROPLASIC GIRL, AGED SIX YEARS.

the elbow is restricted (Turner), in others there is hypotonia. The hands are broad and very short, and the fingers, which are also short, thick, and somewhat conical, diverge from each other, the index and middle fingers being deflected to the radial side, and the ring and little fingers to the ulnar. This appears to be due to enlargement of the heads of the proximal phalanges. This condition, which recalls the spokes of a wheel, has been called the "trident hand," and compared by Thomson to the shape of the hand in some gorillas. In some cases the fourth digits are much shortened, as the result of dwarfing of the fourth metacarpals and metatarsals, but this localized dyschondroplasia (Robin and Weil) is not peculiar to achondroplasia (Chevallier).

The head is brachycephalic, relatively large, and the frontal region bulges; the chin and lower jaw are well developed, and the lips are thick, but the face is

small and the root of the nose depressed. At birth the face has a cretinoid aspect, but it is that of an adult, not of an infant, cretin. The tongue often projects from the mouth at birth, but this is not noticeable later.

Intellect begins at the normal time. Mentally achondroplasias are generally well up to the average, but in some instances a form of mental weakness, which is peculiar in being accompanied by love of relations, has been described (Zosin). Secondary sexual characters develop normally, or even rather early. Sexual activity is considerable in adult achondroplasias; Caesarian section has been performed four times on the same achondroplastic woman (Frühholz and Michelf). Affected girls are often obese, but boys are rarely so. The abdomen is prominent, and, owing to the shortness of the legs, the umbilicus is below the middle point of the body. Inguinal hernia is not uncommon.

PROGNOSIS.—Many achondroplasias are stillborn, dying at the eighth month of intra-uterine life. A considerable number are born prematurely, and die soon afterwards, death being probably in most instances connected with the narrow, rigid foramen magnum and the resulting pressure on the spinal cord. Even general oedema or ascites there may be great difficulty in the birth of achondroplastic infants. In achondroplastic mothers the difficulty of delivery may lead to the death of the offspring. When the achondroplastic survives the first year of extra-uterine life, the disease does not shorten life, except in pregnant women. The prognosis as to growth is bad; the height is not likely to exceed $4\frac{1}{2}$ feet.

DIAGNOSIS can be made at a glance by recognizing that the dwarf has a trunk of normal dimensions with extremely short limbs (micromelia).

DIFFERENTIAL DIAGNOSIS.—From Cretinism.—In a true cretin there are no marked facial changes at birth, no prominence of the buttocks, and no want of proportion between the trunk and the limbs. In achondroplasias the thyroid is usually palpable, there are no supraclavicular pads of fat, and in older ones the mental and sexual characteristics are normal.

Rickets.—Achondroplasias cut their teeth normally, their general health is good, and they do not present any rickety deformities. But it must be remembered that rickets may complicate achondroplasia. *Dwarfs* are small in all dimensions, and their proportions are normal; achondroplasias are deformed dwarfs, the limbs being unduly short as compared with the trunk. This same difference holds good between achondroplasia and the various forms of *infantilis*, and it is generally possible to find a cause for the infantilism.

In *osteogenesis imperfecta* the distortions of the long bones occur in the shafts and not at the epiphyses, as in achondroplasia, and there are multiple fractures. In *osteogenesis imperfecta* the ossification of the membrane bones of the cranial vault is delayed and deficient, whereas in achondroplasia it is normal.

Inflammation of the epiphyseal plate, due to various causes, may arrest the growth of a long bone, and so imitate achondroplasia, but this change would be confined to one, or at most very few, of the bones, and so be easily distinguished from achondroplasia.

The Mongolian idiot has the characteristic oblique palpebral fissures, red cheeks, and idiotic expression.

Mey and Babonneix described as hyperchondroplasia, or the converse of achondroplasia, the changes of elongation and thinning of the long bones of the extremities. Skiagraphs showed that the epiphyseal cartilages were perhaps more

developed than normally. Marfan had previously described this case, apparently unique, as one of "dolichostenomelia."

TREATMENT is of no use. Thyroid extract has been tried, but without benefit.

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HEREDITARY CLEIDOCRANIAL DYSOSTOSIS.

This condition was first described by Sir T. Barlow in 1883, but did not attract any attention until Marie and Sinton gave it the above name in 1898. In 1905 Villaret and Franco tabulated forty-five cases, and in 1910 Fitzwilliams analysed sixty cases.

DEFINITION.—Congenital absence or imperfect development of the clavicles, with deficient and defective ossification of the vault of the cranium.

ETIOLOGY.—It is often hereditary, and several members of a family may suffer; thus, in Fitzwilliams's series eight families provided thirty-one examples, and in the family described by Heinicke seven members were affected; but it is rarely seen in more than two generations. Incomplete forms occur, especially in affected families. It can be transmitted both by the mothers and by the fathers. Sex does not exert any influence: in fifty-four cases there were twenty-eight females and twenty-six males (Fitzwilliams).

It has been suggested that consanguinity of the parents, tuberculosis, syphilis, rickets, and various infections or intoxications, may be responsible for the condition, but there is no convincing evidence in support of any of these causes. The developmental defect begins early (second month) in fetal life. It has been suggested that it is a partial and hereditary variety of osteogenesis imperfecta (Ducote).

PATHOGENY.—Nothing is really known, but several hypotheses have been put forward. It has been thought (1) that the causal factor of the underdevelopment of the membranous bones is deficiency in some chemical constituent (Fitzwilliams); (2) that the condition is a reversion to the type of animals without clavicles, such as the osteosperm, or to the type of animals with rudimentary clavicles, such as the carnivora; (3) that it is an example of an abrupt variation in the human type termed "mutation" by Vries.

MEDIAN ANATOMY.—Clavicle.—Both may be completely absent (six out of sixty cases); it is very rare for one clavicle only to be absent or deficient. More often the sternal end is present and the rest of the clavicle is absent, the sternal end having a ligament attached to it which is thought to be the costo-caroid membrane. In very rare instances the sternal portion is absent and the acromial end present. In a considerable number of cases the sternal and acromial ends of the clavicle are both present but not united, so that there is a congenital pseudo-arthritis. The condition of the muscles attached to the clavicle—the subclavius, the sterno-clideo-mastoid, the trapezius, pectoralis major, and deltoid—naturally varies with the state of development of the clavicle; thus the clavicular portion of the trapezius is often absent.

The Skull.—The defective development of the membrane bones leads to large anterior and posterior fontanelles or to the presence of one large fontanelle. Closure is long delayed, and, though usually complete at the age of twenty, may not occur at all. The biparietal diameter is increased, and the vertical and fronto-occipital diameters are diminished. Apparently, as the result of an attempt to compensate for the deficient ossification, bones are formed over the frontal, parietal, and occipital bones—three on each side, so as to produce a hot-cross-bun or antelope skull. The growth of the superior maxilla is interfered with and the palate is highly arched and sometimes cleft. This stunting of the facial bones is secondary to mal-development of the cartilaginous base of the skull (Pitrewilliams). The frontal sinuses are exaggerated, whereas the sinuses of the face are diminished in size. According to Moschetti, anomalies of the teeth are almost constant; the first dentition is delayed, and often persists to the twelfth year; some teeth, especially the upper incisors, are missing; the teeth are small, irregular, badly embedded and become carious early and rapidly.

Other bony deformities, such as scoliosis, lordosis, kyphosis, defects in the shape of the sternum, and dwarfing of the little finger, may be present.

CLINICAL FEATURES.—The patients, who are usually short in stature, are often unaware of their deformity, which is naturally much more obvious in thin than in fat subjects. Their undue mobility allows the shoulders to be brought in contact under the chin.

The head is enlarged, the forehead bulging, with a depression between the frontal bones, but the face is small; the supra-orbital ridges are prominent, and from depression of the roof of the orbit there may be some exophthalmos, so that the general aspect resembles that of slight hydrocephalus. As a rule the mental faculties are normal and the general health good.



FIG. 156.—Boy with HEREDITARY GROSS CRANIAL DYSOSTOSIS (VILLIERS AND FRANÇOIS).

Incomplete forms of the condition are not uncommon.

DIAGNOSIS.—This depends on the association of defective development of the skull bones formed in membrane, and of the clavicles. It must be distinguished from intra-uterine fracture of the clavicle.

NO TREATMENT is necessary.

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CONGENITAL ELEVATION OF THE SCAPULA.

SYNOXYM.—Sprengel's deformity.

Of this condition, described by Sprengel in 1891, there are more than a hundred examples on record, but the cases described under this heading are not all of the same nature.

ETIOLOGY.—Sprengel's view that the condition is mechanical and due to prolonged fixation of the arm behind the foetus's back *in utero*, on account of a deficiency in the amount of amniotic fluid, is no longer accepted, for in many cases the quantity of amniotic fluid has been normal. It has been thought to be due to injury during labour, or to muscular defects, especially absence of the lower part of the trapezius which allows the upper fibres of the muscle to elevate the scapula. This defect, however, is not constant. Another view is that the scapula is malformed and abnormally situated as a result of a congenital malformation involving the cervico-scapular region and upper ribs, the scapula retaining its fetal position in the neck. In some instances, as first recorded by Willett and Walsham in 1883, the vertebral border of the scapula is united to the spine of the seventh cervical or first dorsal vertebra by a bridge of bone or rod of cartilage, which has been regarded as a reversion to the suprascapular bone of lower vertebrates. In favour of the view that elevation of the scapula is a congenital malformation due to errors in segmentation of the mesoblast is the occasional presence of other abnormalities, such as fusion or suppression of ribs, absence of half a vertebra or of a whole or more vertebrae, polydactylism, suppression of the radius and of the corresponding thumb (Fairbank), hydrocephalus (Nageotte-Willcocks-witch), talipes, torticollis, local hypertrichosis (Greig), idiopathic dilatation of the colon (Boyd).

CLINICAL PICTURE.—The deformity is usually unilateral, though it has been found symmetrical, and is commoner on the right side and in girls. The scapula is raised, small, and its inferior angle nearer to the spine. In cases with a bony bridge between the scapula and the spine the arm cannot be raised above the right angle owing to the restriction of the movements of the scapula. There may be some scoliosis.

DIAGNOSIS.—Congenital elevation of the scapula must be distinguished from elevation of the shoulder which may be due to various acquired factors, such as

glosses or caries of the spine. Tie of the rhomboid muscles may elevate the shoulder, and as the result of paralysis of the shoulder muscles—for example, that due to birth palsy—the other shoulder may appear to be elevated.

TREATMENT is hardly necessary, but exercises, massage, and surgical interference, such as division of shortened muscles or removal of bony bridges, have been recommended.

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FRAGILITAS OSSIUM.

INTRODUCTION.—Fractures on slight provocation occur in children in various conditions, such as rickets, inherited syphilis, scurvy, and new growth. The titles "fragilitas ossium" and "osteopathosia" (Lobstein, 1833) are restricted to cases in which multiple fractures without any obvious cause, either existing or underlying, constitute the disease. The nomenclature of these cases is confusing, and it appears more satisfactory to describe two groups: (1) *Osteogenesis imperfecta*, in which fractures are primary and deformities and bending secondary; and (2) *melnie ossium*, in which softening is the primary and fractures the secondary features, thus to include them in one category.

1. *Osteogenesis Imperfecta*.

SYNOXYSM.—Idiopathic osteopathosia: Annular rickets; Periosteal dysplasia (Daranté); Periosteal aplasia.

DEFINITION.—A rare condition in which multiple fractures occur in intra-uterine or infantile life.

As various conditions have been grouped under this heading, great confusion has resulted. In some cases thus described the bones are soft and pliable so that they can readily be bent (congenital osteomalacia); in others the shafts of the long bones are composed of alternate segments of cartilage and bone; and in others the bones are brittle and fracture easily. The description here will be confined to the cases showing multiple fractures. In the past it was thought that *osteogenesis imperfecta* was incompatible with extra-uterine life, and the cases which survived and continued to show multiple fractures were called by some other name—*fragilitas ossium* or *idiopathic osteopathosia*. To cases commencing in late childhood the title "*osteogenesis imperfecta tarda*" has been applied by Looser. The name "*osteogenesis imperfecta*" was introduced by Vrolich in 1849; and the masses of callus round the fractures give rise to the title "*annular rickets*." In 1897 Griffith collected 67 cases, and in 1906 Lovett and Nichols estimated the published cases at 130.

ETIOLOGY.—Griffith estimated that the condition was hereditary in 27 per cent. of the cases. It may be met with in several members of the same family

without any hereditary history. Like hereditary cleido-cranial dysostosis, it is a congenital disturbance of the periosteum; and Durante considers that they are variants of the same process.

MORPHO-ANATOMY.—The shafts of the bones are short and thick, and, as the result of past fractures, may show deformities or annular thickenings due to rings of callus. The periosteum is thickened and adherent to the shaft of the bone, which has not any proper outer layer of compact bone. The periosteum of the long bones produces little true bone, but gives rise to a considerable formation of cartilage and connective tissue, whereas in achondroplasia it is the cartilaginous formation of bone at the epiphyses which is at fault. The essential fault appears to be that the perichondrium of early fetal life does not undergo the normal metaplasia into the periosteum of the long bones. There is either a lack of the proper stimulus to this hyperplasia or an unknown inhibitory force acting on the osteogenetic tissues (Klotz). In one case the epiphyses were small (Lovett and Nichols). In an infant dying at birth the placenta was fibrous and showed areas of calcification (Klotz).

CLINICAL PICTURE.—As many as 100 fractures have been seen in a new-born child (Klotz). In Chaumier's case there were 115 fractures, 70 of which were in the ribs. The fractures occur suddenly in children, previously healthy, as the result of insufficient causes, such as muscular exertion. The fractures may be incomplete or greenstick, may involve the shaft, or occur at the junction of the shaft and epiphysis. As the causal injuries are usually slight, compound fractures are rare.

The fractures, which are usually in the long bones and ribs, unite rapidly, but by fibrous tissue, which may become calcified but not ossified; the amount of callus varies much. Deformity, however, is prone to occur as the result of union; for the fractures, being due to very slight injury, are less painful than in health, and may therefore, especially when numerous, escape medical attention.

Apart from the deformities due to faulty union of the fractures, malilias osium may supervene, so that the bones bend and thus imitate the changes in rickets. There is frequently deficient ossification of the bones of the vault of the skull; and other developmental defects, such as spina bifida and club-foot, may exist. Electrical examination of the muscles shows a diminished response to the faradic current, and the reaction of degeneration with the galvanic current (Larot).

PROGNOSIS.—When fractures are present at birth, the infant, if not stillborn, will probably die soon; but in cases in which fractures do not occur until some years have elapsed there is little danger to life, although there may be much deformity.

DIAGNOSIS is based on the presence of fractures at birth, or their occurrence soon afterwards on inadequate provocation. It is inadvisable to elicit *crepitus*, as fresh fractures may thus be induced.

From rickets, *osteogenesis imperfecta* differs in the absence of bowing of the ribs, enlargement of the epiphyses, and of the nervous and visceral symptoms.

From separation of the epiphysis in congenital syphilis the diagnosis depends on the position of the fracture, which can be best seen by skiagraphy. Multiple fractures in rare instances occur in syphilitic infants (vide p. 923).

From achondroplasia (vide p. 917).

TREATMENT.—Fractures should be treated in the ordinary way; and when union has occurred, massage to obviate muscular atrophy should be employed. No benefit has followed the use of thyroid extract, calcium salts, or cod-liver-oil.

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2. *Mollities Ossium.*

SYNONYM.—*Osteomalacia*.

DEFINITION.—Spontaneous softening of bone resembling that seen in adult females.

The bending of the bones so characteristic of rickets must always be excluded before admitting a case to be one of mollities ossium. Some doubt as to the existence of mollities ossium in children has been expressed (Ziegler); but cases have been recorded by Rein and von Recklinghausen, Davies-Colley, Mosley, Dent, Siepert, Broca, and others.

ETIOLOGY.—In adults the disease is almost confined to females, but this is not so absolute in the case of children, among whom it is rare, for Hatfield and Tixeront refer to twelve cases only. It is not hereditary, but more than one case may occur in the same family (Broca).

PATHEGENY.—The hypothesis of excessive internal secretion of the ovary, causing hyperemia of the periosteum and decalcification, which has been evoked to explain the disease in women, appears improbable in the case of children. It has been ascribed to adrenal insufficiency, mainly on the evidence that adrenalis has benefited the patients (Bossi).

Evidence has been brought forward to show that in children between ten and fifteen years mollities ossium may follow infectious diseases, such as scarlet fever and whooping-cough, and that this softening, which causes permanent deformity, may pass off and the bones become rigid. The softening may be due to the local action on the bones of infective micro-organisms (Dent). Osteomalacia of tuberculous origin has also been described (Poncet and Leriche). This softening may occur in patients who had rickets earlier in life and had recovered. It seems probable, therefore, that some cases described as late rickets are examples of mollities ossium.

MORPH ANATOMY.—The periosteum is thickened, the underlying bone is soft, and the medullary cavity is enlarged, so that the bones become very thin, and can be readily cut with a knife. The specific gravity of the bones is diminished. There is a rarefying osteitis with decalcification of the bone. The joints and articular surfaces are normal.

Clinical Picture.—The onset is gradual; pain always appears early, and may involve the whole skeleton, but is specially severe in the lower limbs. The pain is aggravated by pressure and exercise, and there is excessive fatigue on slight exertion. The bones bend, and may do so to an extraordinary extent. When

these deformities have appeared, fractures occur very readily. Skiagraphy shows that the shafts of the bones are unduly translucent and the medullary canal large.

DIAGNOSIS.—Osteogenesis imperfecta and mollities osium may both show fractures and bending of the bones; but in osteogenesis imperfecta multiple fractures occur *in utero* or soon after birth, and may be followed by some deformity of the bones; whereas in mollities osium, which is an acquired disease, the bones, previously healthy, soften, bend, and then fractures may occur. Mollities osium may supervene in a case which begins as osteogenesis imperfecta. This is explained by Hastings Gilford's view that "any organ or tissue affected with infantilis is prone to premature degeneration or senilism. Hence osteogenesis imperfecta (infantilis) is liable to be followed by osteomalacia (senilium) or osteitis deformans (senilis)." It is doubtful if mollities osium occurs in infants, and there is practically no way of differentiating severe rickets with extreme bony softening in infants from mollities. Late rickets, which is rare, resembles mollities, but in the former disease the bending is not so extreme as in mollities.

TREATMENT.—The patients should, obviously, be protected from all causes which favour fracture, and are therefore safest in bed. The general health should be maintained by fresh air, good food, and cod-liver-oil and calcium hypophosphites may be given. Phosphorus, subcutaneous injections of adrenalin, and ethylchloral anaesthesia, are stated to have given good results in some cases, but they may fail.

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TUBERCULOUS DISEASE OF BONES AND JOINTS.

As tuberculosis specially attacks the cancellous tissue of bone, and thence readily spreads to the adjacent joints, and, further, since tuberculous disease of joints is usually secondary to disease of the adjacent bones, the two conditions are closely related, and will be considered together.

Although tuberculosis in bones and joints usually appears to be primary during life, it is really secondary to some focus, often of small size, the bacilli being conveyed by the blood-stream. Injury, by reducing the local resistance of a bone, favours the growth of tubercle bacilli. According to most of the available evidence the bacilli of bone disease are of the human and not of the bovine form, but out of seventy cases of tuberculosis of bones (thirty-one) and of joints (thirty-nine) of children in Edinburgh, forty-one, or 58 per cent., were due to the bovine form, twenty-six to the human form, and three to both forms (Frazer). Tuberculosis of bone is rare in the first three months of life. The favourite sites of tuberculosis of bone are the vertebrae, the ends of the long bones, the tarsus, carpus, ribs, and sometimes the skull. It has been estimated that a third of the cases in children occur in the spine, another third in the hip, and then with decreasing frequency in the knee, ankle, elbow, shoulder,

Tuberculous Dactylitis.—The metacarpals and phalanges are enlarged, spindle-shaped, and tender from the presence of caseous material in the interior of the bones. The lesions are multiple. The shafts of the bones become thinned and expanded (*spina ventosa*), and the caseous material may soften into an abscess and perforate, thus setting up some periostitis. The appearances must be distinguished from those of syphilitic dactylitis and of multiple enchondroma.

Tuberculous Osteo-periostitis of the frontal, parietal, and occipital bones may occur. Reber has collected one hundred and fifty-eight cases. It may occupy the centres of ossification, and so imitate syphilis, especially in the absence of bony lesions elsewhere. There may be numerous soft nodes on the cranial bones; as many as twenty-nine have been recorded (Ménard and Bafnoir). Tuberculous nodes on the skull may coexist with lesions of the long bones, caseous tubercles in the viscera, and widespread tuberculosis.

Tuberculous Spondylitis (Synonyms: *Spinal Caries*; *Pott's Disease*).—Tuberculous disease of the vertebrae attacks the cancellous tissue of the bodies and destroys the bone, with the production of caseous material. It is most frequent in the lower dorsal and rarest in the cervical regions. As the arches and spines commonly remain, even when the bodies of several vertebrae have been destroyed, the onset of kyphosis is gradual; evidence of pressure on the spinal cord is much less prominent than in fracture dislocation, and is due to the pressure of an abscess.

The Symptoms vary very considerably according to the position of the lesion, and may be misinterpreted; thus, there may be persistent or recurrent abdominal pain imitating colic; or pain in the limbs, pamplogia, or nervous symptoms, which in the absence of any spinal deformity may give rise to difficulty in diagnosis (vide Chapter XIV., p. 855). A retropharyngeal, lumbar, or psoas abscess may result. Muscular rigidity of the back is a constant and valuable sign. Local pain in the spine is caused by jarring the head or the spine, as in jumping, but not by pressure on the spines. Kyphosis is a late result.

In children the Prognosis of spinal caries is good, much better than in older subjects, provided that treatment is undertaken early.

Diagnosis.—The kyphosis of rickets, myopathies, and ankytonia congenita, is painless, forms a wide curve instead of an acute angle, and disappears when traction is made on the legs, except in severe and long-standing cases of rickets.

Treatment.—Vide p. 326.

Tuberculosis of the Long Bones of the limbs occurs in the epiphyses, and may then be spoken of as *para-articular*. It may attack one bone or several, but is seldom symmetrical. The tuberculous process in the epiphyses is accompanied by marbling osteitis, and there may therefore be an absence of any bony enlargement. Skiagrams show an increased transparency of the bone. The tuberculous infection readily spreads to the articular surface; the rapidity of extension depends on whether the epiphyseal line is inside or outside the synovial membrane; thus, tuberculosis of the lower end of the femur and of the upper end of the tibia are associated with early invasion of the joint, whereas in tuberculosis of the lower ends of the fibula and radius the joints may remain healthy for some time. In the latter event a *para-articular* abscess may form, and is then likely to infect the joint (Tollby). Pain and tenderness in such extra-articular tuberculosis are localized to one spot, and the clinical picture is quite different from that seen in tuberculous arthritis.

Tuberculous Arthritis is usually secondary to, and the main clinical manifestation of, tuberculous disease of the neighbouring epiphyses. In the knee, however, Stiles finds that the disease begins much more frequently in the synovial membrane than in the bone.

MORBID ANATOMY.—The synovial membrane containing miliary tubercles becomes swollen, reddened, and covered with flakes of fibrin; it ulcerates and adheres to the cartilages which are eroded, and eventually the bone is exposed. The joint becomes distended with fluid—sero-fibrinous, fibrino-purulent, or purulent—and may contain sequestrated bodies showing concentric laminations. In neglected cases peri-articular abscesses form. Tubercle bacilli are somewhat rare in the effusion, but inoculation into animals proves their presence.

CLINICAL PICTURE.—The onset is usually gradual, though it may be quite acute, and the course is chronic. One or more joints may be affected, but the disease is seldom symmetrical. Rigidity with limitation of movement in all directions is an early and important sign. The joint becomes swelled and swollen, the outline of the synovial membrane is obvious, and thickening may be detected. There may be local heat, but the skin is not reddened in the early stages. Pain is a symptom of little value, as it may be absent or slight; on the other hand, it may be severe, especially in the hip, in which pain is often referred through the obturator nerve to the inner side of the knee; jerking pains at night may occur, and limping is an early symptom. Muscular wasting accompanies all forms. Suppuration may occur in or about the joint. With X-rays the joint appears fluffy and the picture is characteristically blurred (Shenton).

Prognosis is better in children than in adults, provided suitable treatment is adopted early. Dissemination is not so likely to occur as in adults, but death may result from tuberculous meningitis. Mixed infections, as in discharging abscesses, add greatly to the gravity of the prognosis.

DIAGNOSIS.—In the early stages acute rheumatism must be differentiated by the effect of salicylate treatment. Rheumatism of the hip may simulate tuberculous disease, but it should be remembered that cases of tuberculous disease, if neglected on the ground that they are rheumatic, suffer irreparable damage. Cases with an acute or subacute onset may simulate pneumococcal, gonococcal, or other forms of infective arthritis, and to arrive at a diagnosis it may be necessary to aspirate the joint and examine the fluid bacteriologically. The use of tuberculin tests is open to the fallacy that there may be tuberculosis elsewhere. Determination of the opsonic index before and after massage of the joint, or the application of Bier's cups, may be tried; and help may be obtained from skiagraphy. Tuberculous arthritis of the knee must also be distinguished from bilateral effusions into the knee-joints in delayed congenital syphilis (vide p. 943). Infantile scurvy, on account of the pain and fear of movement, may imitate tuberculous disease of the knee and hip.

TREATMENT.—Though tuberculosis of bones and joints is commonly regarded as a surgical disease, the treatment should be mainly medical. The first essential is absolute rest to the part, which should be immobilized by a suitable and simple splint without traction except in so far as this may be required to correct deformity. The patient's resistance should be increased by all available means; children should be treated in the open air at the seaside or in the country, and should not be kept in towns. Good food, with plenty of meat, cream, and fats, should be given.

Good results have been obtained, especially by Maynard Smith, from tuberculin treatment, and discharging sinuses or abscesses with mixed infections may be treated with corresponding mixed vaccines. But vaccine treatment should be combined with, not substituted for, the all-important methods of rest, open air, and good food. Eber's hyperæmic treatment whereby auto-inoculation is induced has also been employed. Massage to obviate atrophy of the muscles may be useful.

Operative interferences should be avoided as far as possible, in order to exclude pyogenic infection. If necessary, joints may be aspirated rather than opened. Robert Jones deprecates hurry in opening abscesses, because if the joint be kept at rest they may disappear. Maccewen advises injection of tuberculous foci in the epiphyses with sterilized emulsion of iodoform.

Tuberculous Pseudo-Rheumatism is described by Poncet and his followers as a form of acute synovitis, resembling that of rheumatic fever though not reacting to salicylates, and apt to terminate in ankylosis or in an ordinary tuberculous joint. It has been thought that the synovitis is due either to tuberculous toxins absorbed from other parts of the body or to tubercle bacilli, as in a tuberculous pleural effusion. It has been suggested that some cases of rheumatoid arthritis in children are thus due to tuberculosis or constitute a juvenile form of Poncet's tuberculous pseudo-rheumatism, but there is little real evidence available to support this.

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SYPHILITIC DISEASE OF BONE.

Although infants may acquire syphilis, the cases seen in practice are nearly always inherited; the following description will therefore deal with that form.

The bony lesions of inherited syphilis, which have been stated to be as frequent as the cutaneous manifestations, differ somewhat from those of the acquired disease in adults. This is connected with their occurrence during the period of growth of bone, for the syphilitic changes are most marked at the epiphyseal ends of the long bones and the centres of ossification of the flat bones of the cranial vault. The bones formed in cartilage are affected earlier than those laid down in membrane; thus, syphilitic disease of the epiphyses (osteochondritis) has been seen in stillborn infants, whereas the nodes on the cranial bones occur later.

Syphilitic Epiphysitis (Synonyms: Osteochondritis (Wegner); Parrot's Disease).—This is a lesion of very early life, generally within the first three months, and may be present in stillborn infants. It is said to occur in 5 per cent. of infants with congenital syphilis (Milnes); but other estimates are much higher. Several members of a syphilitic family are sometimes attacked, thus illustrating the liability of certain tissues in such families to be specially affected (Thomson).

MORBID ANATOMY.—There is syphilitic inflammation of the epiphyseal plate and of the overlying periosteum; the epiphyseal plate becomes swollen, yellowish-white, and the process of ossification irregular. Later the epiphyseal plate softens, and eventually the epiphysis may separate. Separation may occur around the epiphysis without any separation; the pus may extend (1) into the joint—usually more than one joint being thus affected—or (2) into the peri-articular tissues and muscles. The treponema pallidum cannot always be demonstrated in typical cases of epiphyseal disease (Schultz). The upper extremities are more often attacked than the lower, the upper end of the humerus being most often affected. Usually it is symmetrical, though one side may be more markedly affected. Periostitis over the ends of the bones may occur without epiphyseal disease.

CLINICAL ASPECT.—The infant never moves the affected limb voluntarily, and cries when the part is manipulated, though there does not appear to be any pain at other times. The limb is flaccid and flail-like, but the muscles react to electrical stimulation, and there is not any true paralysis. This pseudo-paralysis, which may occur without any evidence of epiphyseal disease, is, in the absence of rickets, characteristic of congenital syphilis. There may be swelling and slight oedema at the site of the lesion. When separation of the epiphysis has occurred, there may be crepitus, but this is far from constant. Skiagraphy shows the irregular ossification and the separation of the epiphysis. The epiphysis may re-unite.

PROGNOSIS is favourable when one or two bones only are affected and when appropriate treatment is adopted. Spontaneous recovery has, indeed, been known to occur. But the outlook is very bad when the lesions are multiple and when other syphilitic manifestations are severe.

DIAGNOSIS.—Signs of syphilis elsewhere, a positive Wassermann reaction, if this is practicable, and to a less degree pseudo-paralysis, make the diagnosis certain. The epiphyseal enlargement might be confused with that of rickets, in which pseudo-paralysis may also occur; the concomitant signs of the two diseases should prevent mistake, but the two diseases may coexist. It must be distinguished from other forms of acute arthritis occurring in infancy, especially of gonococcal and pneumococcal origin. Multiplicity is in favour of syphilis. Infantile scurvy produces a swelling which is not confined to the epiphysis, is accompanied by some other diagnostic signs, and hardly ever occurs before the age of six months. Both pabsies do not implicate the joints, and are present at birth. Acute poly-arthritis also does not involve the joints, and is not painful on movement.

TREATMENT.—*Vide* p. 930.

Fracture of Bones, apart from separation of the epiphysis, is rare in inherited syphilis. It may occur in several members of the same family. Willard records a family in which thirty-two fractures occurred in three generations. In infancy the fractures are usually multiple, and they may be so numerous as to suggest osteogenesis imperfecta. The fracture may be incomplete or "greenstick." In older children fracture of a single bone may be due to a gunna.

The usual signs of fracture are present. A few patients have shown a blue colour of the sclerotics, and the association has been explained by the assumption that there is a deficiency of fibrous tissue in both the sclerotics and the skeleton (J. Rolleston).

The skull in early congenital syphilis is affected in one of two main forms:—(1) Atrophic, in which absorption of bone occurs. In addition to cranial tabes or

Discrete areas of atrophy (vide p. 930), there is in rare instances more widespread absorption of bone (*caries sicca*). The bones of the nose commonly undergo syphilitic ulceration. (2) Hyperplastic, in which there is thickening of the bones of the cranial vault, giving rise to the well-known Parrot's nodes or the natiform or bat-eaten-bun skull. When there is rickets in addition, as is not uncommonly the case, the new bone is soft in consistency (Shattock). In cases of combined syphilis and rickets the bowing of the skull is more marked than in either disease alone. In both craniotabes and bowing of the skull the relative importance of syphilis and rickets has been much discussed. The cranial bowing may be associated with some hydrocephalus and distension of the cutaneous veins.

Syphilitic Dactylitis is not common; in one hundred cases it was noted in two (Still). It usually occurs within the first two years of life, and has been seen a few months after birth. It attacks one or more bones, generally the proximal phalanges of the hands, and may be symmetrical, the swelling being more noticeable on the dorsal than on the ventral surface. Doubt exists as to the morbid anatomy; some describe it as a gummatous periostitis, others as epiphysitis, and others, again, as osteomyelitis. Possibly the morbid change is not the same in all cases. It resembles tuberculous dactylitis, but is less prone to attack the metacarpals, and does not give rise to suppuration. The treatment is the same as in the early stage of congenital syphilis (vide Chapter XXI., p. 1103).

In delayed hereditary syphilis bony changes are frequent. In 132 cases the bones were affected in 39 per cent., the liver being the next most frequent site of lesions—namely, in 34 per cent. (Forbes). The bony changes are most often seen at and shortly after the age of five years, and become less frequent at the age of fifteen. The disease attacks the long bones, and particularly the tibia. Usually more than one bone is affected, and not uncommonly the disease is symmetrical. There may be nodes on the long bones, particularly the tibia, resembling those in acquired syphilis. In a rather special form of the disease the changes consist of hypertrophic osteoperiostitis (see Fig. 163), with extensive production of new bone, or there may be gummatous formations in the medulla and other parts of the bone, or the same bone may show combined osteo-periostitis and gummatous changes. A gumma may cause spontaneous fracture, but this is very rare. The usual change is chronic or subacute osteo-periostitis, and may lead to such hyperostosis that osteitis deformans or a sarcoma is simulated. In this connection it may be mentioned that Lamaze and Fournier regard osteitis deformans as the result of congenital syphilis. The tibia becomes bowed forward (*gibber-blade tibia*), and at first slight bowing in rickets is simulated; but the middle third of the bone, instead of the lower third as in rickets, is mainly affected, and skiagraphy shows absence of the characteristic rickets deformity. As the result of the hyperplastic process, the length of the bone is increased. Necrosis and suppuration, which it seems natural to refer to superadded infection, may occur. Pain, especially at night, may be the earliest manifestation; it may be very severe or may be almost absent. In some instances the patient shows infantilism or progressive mental enfeeblement; occasionally, as the result of syphilitic endarteritis, the pulse may be absent from the extremities.

DIAGNOSIS.—Evidence of syphilis elsewhere or a positive Wassermann reaction may clinch the diagnosis. The condition must be distinguished from the lesions of rickets by the points already mentioned and by other symptoms. Typhoidal

periostitis may occur in children, and will be suggested by the history and by the absence of other signs of syphilis.

TREATMENT.—Atisymphilitic treatment with mercury and iodides should be employed. But the massive osteo-periostitis may be very resistant to treatment. Salzman has been successful when mercurials and iodides have failed, and should be tried.

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CRANIOTABES.

This term has been applied to more than one condition, but this description will be confined to localized spots of thinning of the cranial bones—the craniotabes of Ekström (1843). These may be due to several causes, the relative influence of which has been variously estimated. Most authors agree that they are commonly a manifestation of rickets, or that they are due to dietetic errors which later induce rickets (Castley). Syphilis is a factor of importance. According to Barlow and Lees, 47 per cent. of the cases of craniotabes are syphilitic, and Carpenter found that, out of 258 cases of craniotabes, 204, or 87 per cent., were syphilitic, that 61 per cent. occurred within the first four months of life—that is, before rickets is an active factor—and that about 50 per cent. of syphilitic infants show craniotabes. The most marked examples are seen in children with both rickets and syphilis. Craniotabes may occur in chronic hydrocephalus, and may be met with in infants in whom no obvious cause is forthcoming.

The parietal and occipital bones are affected. In 50 per cent. of Carpenter's cases the parietals alone were affected, and in less than 5 per cent. was the thinning confined to the occipitals. Adams figures craniotabes in the frontal bone. These areas are chiefly seen near the lambdoid suture. The thinning has been thought to be due to the pressure exerted on malnourished bone by the pillow on the outside, and by the brain internally. The process of bony absorption begins in the inner table, and may advance so much that only a layer of membrane is left. These thinned areas, which can be well seen by holding the skull-cap up to the light, yield when pressed by the finger, and feel like tightly stretched parchment. Craniotabes is not uncommonly associated with laryngismus stridulus, both being manifestations of rickets.

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BOSSING OF THE SKULL.

Bosses on the bones of the vault of the skull may be associated with several conditions, especially with rickets and inherited syphilis; in fact, the most marked examples are found in patients with rickets and syphilis combined. These bosses are due to the exuberant formation of new bone around the centres of ossification of the frontal and parietal, and occasionally of the occipital and temporal bones. Usually the bosses are symmetrical. Plagiocephaly, or a prominent boss on one half of the frontal bone, usually the right, with a corresponding prominence on the opposite occipital region, depends on the position of the child's head. As a result of the heaping up of bone over the frontal and parietal eminences, the lines of the sutures appear depressed, and the well-known nutiform or hot-cross-bun skull results. It has been stated that, when due to uncomplicated syphilis, the bosses are nearer to the anterior fontanelle than are the bosses due solely to rickets; but clinically it is unwise to attempt any such distinction purely on the position of the bosses. Other evidences of syphilis or rickets in the head and elsewhere in the body must be sought for. These bosses, specially described by Parrot, are often called "Parrot's aches." As is well known, he considered that rickets was due to inherited syphilis, a view which has never been accepted in this country. Bossing of the skull may occur in cases of anaemia with splenomegaly, and may then be due to associated syphilis or rickets.

Osteo-periostitis over the centres of ossification of the frontal and parietal bones may be due to tuberculosis, and may for a time imitate syphilitic disease. As many as twenty-nine such bosses have been recorded in a child aged four years (Ménard and Bufnoir). They may suppurate, and a diagnosis of their nature may be made by the detection of the tubercle bacillus.

Bosses on the bones of the vault also occur in achondroplasia (vide p. 914), hereditary cleido-cranial dysostosis, secondary metastases of new growth—for example, in malignant hypernephromata and chloremia.

In infantile scurvy bosses on the skull may be due to concomitant rickets *vs.*, in rare instances, to hemorrhages. Bosses due to hemorrhages have been recorded in osteogenesis imperfecta.

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LEONTIASIS OSSEA.

SYNONYMS: Hyperostosis cranii; General hyperostosis; Cephalomegalia.

This condition, formerly regarded as distinct from osteitis deformans, is probably very closely allied to that disease, if it is not a modified form of it. It differs from it in its site of predilection—the bones of the skull—and in occurring earlier in life. But in very rare instances osteitis deformans has begun before the age of three years (Elmer, Sonnenberg), and it has been thought that cranial hyperostosis is the initial stage of osteitis deformans (Priest). The changes may be so much more obvious in the bones of the head than elsewhere that the latter are overlooked.

ETIOLOGY.—The condition is rare: according to Hallé, there are only thirty cases on record. It appears that about a third of the cases start in childhood. Out of twenty-eight cases collected by Karsvel nine were congenital or began in early childhood. Heredity does not exert any influence. The sexes are equally affected.

The cause is quite unknown. It has been suggested that it is a trophoneurosis, but there is no evidence of any primary change in the nervous system.

MORPH. ANATOMY.—All the bones of the head—cranial and facial—are affected. There is great thickening, which leads to obliteration of the various air sinuses, to narrowing of the foramina for nerves and vessels, and to diminution in the size of the orbits. The change is generally described as an osteosclerosis.

SYMPTOMS come on earlier and are more prominent than in ordinary osteitis deformans. The skull, and especially the forehead, increase in size. Headache, neuralgia, blindness, deafness, and paralysis are caused by pressure on nerves; whilst distension of veins, embarrassment of the cerebral circulation, and mental disturbances result from narrowing of the channels for bloodvessels. Enophthalmos and downward displacement of the eyeball are often present. The course of the disease is slow, and may be prolonged for twenty or thirty years.

DIAGNOSIS.—The condition must be distinguished from acromegaly, with which it has in the past been confused. Hyperostosis of the skull and other bones may occur after the arrest of rickets ossium and severe rickets. A form of bone disease resembling osteitis deformans, and chiefly seen in the tibia, may be due to congenital syphilis (see p. 329).

TREATMENT.—Antisyphilitic treatment should be given a trial. If this fails, surgical treatment may occasionally give relief.

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NEW GROWTHS OF BONE.

New growths of bone may be (1) non-malignant or (2) malignant.

1. Non-Malignant New Growths.

Short descriptions will be given of simple cysts, multiple exostoses, and enchondromata.

Simple cysts of bone are not very common, but they chiefly occur during the period of growth and near the ends of the long bones, especially the humerus, tibia, and fibula. Bloodgood collected thirty-six cases under fifteen years of age. They have been thought to be due to injury, but appear to be closely connected with fibrous osteitis, and may be due to softening of a patch of fibrous osseous tissue. Pain is absent, and fracture of the bone may be the first sign, but the subsequent union may be quite satisfactory. These cysts may be confused with central sar-

osarata, especially myeloid tumours, but skiagraphy shows a hollow cavity with a regular outline. The swelling should be explored and the contents examined microscopically. Elasmie recommends curettage with cranking in of portions of the cyst wall. The so-called "myeloid sarcomata," which arise endosteally from the spongy tissue of the shafts of the long bones close to the epiphyses, and most frequently in the upper end of the tibia, are locally destructive, but not truly malignant. They are commoner after the age of twenty years, whereas simple cysts are more frequent under that age.

Multiple Exostoses.—Etiology.—Males are more often affected than females, in the proportion of two to one. This condition is hereditary, and may be transmitted by males or by females. Unaffected females may transmit the affection, and hence it may appear that males more often transmit it. An affected individual who marries a normal person transmits the condition to about half his offspring (Gossage and Carling).

Normal Anatomy.—The exostoses, which are originally cartilaginous, are, while growing, tipped by a layer of hyaline cartilage. Under this there is a layer of compact bone covering cancellous bone, and a central cavity communicating with the medullary canal of the bone. The surface may be covered by a bursa. They arise from bones formed in cartilage, and especially from long bones—such as the humerus, radius, femur, tibia, and fibula—near the epiphyseal plate. They are most frequent around the knee, at the upper end of the humerus and the lower end of the radius, thus corresponding with the favourite sites of myeloma (myeloid osaroma). They vary much in size and in shape.

PATHOGENY.—The condition appears to depend on an inherent vice of development in the epiphyseal cartilage. There is no evidence that inherited syphilis, rheumatism, or rickets in any way influence their production. It has been thought that tuberculosis in the patients or in their parents is an important aetiological factor (Cartillet, Lortat-Jacob; and Sabarbanu).

CLINICAL PICTURE.—The tumours may be few or extremely numerous—even up to 1,000 (Chisci). Though they have been noted at birth, they usually appear in childhood and increase in number and size up to the time when the epiphyses unite—about the twenty-fifth year. They then cease to grow, and may appear to get smaller; this is important from the point of view of prognosis. They may be painless, but during their growth they may cause some discomfort and tenderness; pressure may be exerted on nerves and on vessels, and may even lead to dislocation of joints. Sarcoma may arise in cases of multiple exostoses, just as in multiple fibro-osaromata.

DIAGNOSIS.—As exostoses may occur in myositis ossificans progressiva, the presence of bony growths in the muscles must be looked for. Restriction of movement, which is such a prominent feature in myositis ossificans, is absent or limited to a single joint in multiple exostoses. Skiagrams show the exostosis as a hook-like process which invariably points away from the epiphyseal plate (Shenton). Multiple enchondromata are found in the phalanges of the hands and feet, and grow from the inside of the bone which they expand.

TREATMENT is unnecessary unless pressure is exerted on nerves or bloodvessels. The exostosis should then be removed by a chisel. Removal may be difficult: I have seen sarcoma follow operation.

Multiple Enchondromata.—Multiple enchondromata springing from the centres of the phalanges and bones of the carpus and tarsus occur in the hands and feet of children. Several members of a family may be affected, and, according to Vinograd, rickets, by causing displacement of islands of cartilage, is an aetiological factor. Trauma may also exert a causal influence. The tumours, which usually cease to grow about the time that the epiphyses unite, expand and thin the bone, are prone to degenerate, and from myxomatous change may become cystic. They may fluctuate or give egg-shell cracking, and the skin over them is stretched and shows dilated veins. Considerable deformity results, and the hands may be useless. They must be distinguished from tuberculous dactylitis, in which suppuration is prone to occur, and from syphilitic dactylitis by the presence of concomitant disease.

2. Malignant New Growths.

Primary Malignant New Growths of bones are sarcomata or endotheliomata, and are rare under ten years of age, after which they are more frequent. In 119 cases of sarcoma of the leg and thigh 3 cases occurred under five years of age, 3 between five and ten years, and 35 between ten and twenty years (Batlin and Colby). Primary sarcoma of bone has been seen in infants a few weeks old.

Secondary Malignant New Growths in Bone.—As compared with adults, children rarely suffer from secondary growths in bone; this is probably because metastases in bone in adults are most often secondary to primary carcinoma of the mamma, prostate, thyroid, and malignant hypernephromata, the first three of which are practically unknown in children.

Malignant hypernephromata in children are apt to give rise to bony metastases, especially in the skull bones (see Fig. 69)—(Hutchinson), thus causing proptosis, and in the ribs; this occurs much more often when the growth is on the left side than when it is on the right (Pruv). In the early stages of bony growth there may be pain in the limbs imitating rheumatism. When proptosis appears, the condition must be distinguished from (a) exstoma by the absence of lymphocytosis; (b) infantile scurvy by the absence of other signs and the failure of antiscorbutic remedies; and from (c) the rare condition of plexiform neuroma of the orbit which may occur in children (Parsons).

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DISEASES OF JOINTS.

ACUTE INFECTIVE ARTHRITIS.
 PNEUMOCOCCIC ARTHRITIS.
 STREPTOCOCCIC ARTHRITIS.
 ACUTE TOXIC ARTHRITIS.
 ENTEROCOCCIC ARTHRITIS.
 GONEO-ARTHRITIS.

SYMPLECTIC ARTHRITIS.
 JOINT AFFECTIONS IN NERVOUS DISEASES.
 HEMORRHAGIC JOINTS.
 CLORATED FINGER.
 HYPERTROPHIC OSTEO-ARTHRITIS.

ACUTE INFECTIVE ARTHRITIS.

In addition to the forms of acute arthritis specially described in this article—pneumococcic, streptococcic, toxic—there are numerous conditions in which acute arthritis may occur. In children the vascularity of the epiphysis favours the advent from the blood-stream of micro-organisms, which in suitable circumstances, such as diminished resistance and injury, may lead to suppuration and eventually infect the joint. Micro-organisms in the blood may also reach the synovial membrane directly, as in adults.

It will be convenient to refer briefly to the etiology of these forms of acute infective arthritis under two categories—(1) Non-suppurative, and (2) suppurative.

1. Non-Suppurative Arthritis, often transient and mild, may occur after injury and in many diseases. It is often assumed to be rheumatic—for example, when associated with tonsillitis, erythema nodosum, or purpura; but this conclusion is open to considerable doubt. Arthritis may appear in connection with mumps, measles, diphtheria, Vincent's angina, but the extreme rarity strongly suggests that an additional factor, such as a mild infection, is responsible. Some of these cases may fall in the category of toxic arthritis (vide p. 938). Synovitis of the hip-joint during the course of typhoid fever may so stretch the capsule that spontaneous dislocation follows. Of the cases collected by Keen, a high proportion were in children. Though the effusion may become purulent, this does not usually occur. In extremely rare instances acute synovitis has been noted in the course of generalised tuberculosis. Though probable, it has not been proved to be due to miliary tubercles of the synovial membrane.

2. Suppurative.—The acute arthritis of infants due to penetration into the joint of a small osteo-myelitic abscess near the epiphyseal plate is generally streptococcal or staphylococcal, but may be pneumococcal. It may be excited by injury, but if micro-organisms latent in the bone being thus activated. The clinical condition is very much the same as that of acute infective epiphysitis (see Chapter XX., p. 1685). The prognosis is bad. Out of twenty-seven cases thirteen died (Marsh and Watson).

Closely allied are the suppurative joints seen in pyæmic conditions, such as may complicate scarlet fever, otitis media, occasionally in umbilical and cutaneous infections, enteric and other fevers. In congenital syphilis suppurative arthritis may occur in connection with epiphyseal disease, and also independently.

In rare instances micro-organisms belonging to the influenza (Dudgeon and

Adams) or coliform groups (Nitchley Fletcher) have been found in suppurative arthritis. The occurrence of arthritis due to the *diplococcus intracellularis meningitidis* is referred to on p. 944.

Suppurative arthritis must be treated surgically by incision and washing of the joint.

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PNEUMOCOCCIC ARTHRITIS.

This is one of the most important forms of infective arthritis in early life, especially in infants. In 100 cases Nitch found that 51 were under fourteen years of age, 25 of these being less than two years of age. It is much more often unconnected with pneumonia in children than in adults. Thus, among 69 cases in adults there were 9, or 13 per cent., without pneumonia, whereas in 51 children under fourteen years there were 18 cases, or 35 per cent., without pneumonia (Nitch).

In addition to pneumonia, pneumococcal empyema and broncho-pneumonia, the middle ear, pharynx, tonsils, and possibly the conjunctiva, may be points of entry for pneumococci which eventually reach the joints, and give rise to an arthritis which may appear to be primary. Nattan-Larrier reports arthritis in an infant of three weeks, due to pneumococci derived from a hare-lip operation. Previous injury probably favours infection.

MORBID ANATOMY.—The exudation into the joint varies in character; in mild cases there is thick pus of a greenish-yellow colour, whereas in severe cases it is thin and watery. In some instances the effusion is serous. The changes possible, but are less marked than those in other forms of suppurative arthritis, and except in severe cases the cartilage escape. In a few instances the suppuration has been peri-articular.

SYMPTOMS.—The knee, hip, shoulder, and ankle are the joints most commonly attacked; one or more joints may be affected. The joint is distended, hot, and extremely painful, but there is no local redness. Opinions differ about the presence of local oedema. Hering says that it is present, Eosman that it is absent, and Nitch that it is present in very severe cases only. The swelling may spread widely along the limb. The constitutional disturbance is comparatively slight.

PROGNOSIS is bad, as about 50 per cent. of the cases prove fatal; it has been thought to be less grave in children than in adults. The prognosis is better in cases with thick pus than in cases with a more watery exudation in the joint.

DIAGNOSIS.—The pallor of the skin over the joint is characteristic. The presence of other pneumococcal infections would naturally be of assistance, but acute arthritis occurring shortly after pneumonia may be due to streptococci. In some instances a diagnosis can only be made by bacteriological examination of fluid aspirated from the joint. In suppurative arthritis due to streptococci or staphylococci the constitutional symptoms are more prominent.

TREATMENT.—The affected joint should be open and drained; incision is preferable to aspiration. A vaccine may also be used.

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GONOCOCCIC ARTHRITIS.

HISTORY.—Clement Lucas's observation in 1880 that acute arthritis in the newborn might be secondary to gonococcal conjunctivitis attracted little attention in this country until 1899, when he collected twenty-three cases. Gonococcal arthritis in older children as the result of gonococcal vulvo-vaginitis was recognized later.

ETIOLOGY.—In the newly-born gonococcal conjunctivitis is commonly due to direct maternal inoculation at birth. Gonococcal stomatitis and rhinitis may also be contracted at birth and give rise to arthritis. Schiller has collected four examples of the coexistence of this arthritis in infants and their mothers. In older children gonococcal ophthalmia may be due to the use of infected towels, sponges, or fingers. Gonococcal vulvo-vaginitis may be caused in the same way, especially in institutions, or by sexual contact with an infected person. Gonococcal urethritis in boys is a much rarer cause of arthritis. Gonococcal arthritis has followed inoculation of the conjunctiva with gonococci for the cure of granular lids. In adults gonococcal arthritis is very much commoner in males than in females, but in young children this difference is not so marked; in fifty collected cases thirty-three were males and seventeen females.

MORBID ANATOMY.—The changes vary according to the severity of the attack; in mild cases the inflammation is confined to the synovial membrane; in suppurative cases the cartilages become destroyed and infiltration spreads to the peri-articular tissues.

CLINICAL PICTURE.—In cases secondary to gonococcal conjunctivitis the arthritis usually appears two to three weeks after the onset of the eye affection. The knee, wrist, and ankle joints are most commonly affected; according to Lucas, those on the left side are more often attacked, because, as the infant is usually carried on the nurse's left arm, the left side of its body is more exposed to trauma. Other joints, such as the elbow, hip, small joints of the hand, and sterno-clavicular, may be affected, and the tendon sheaths and bursæ may suffer. More than one joint is usually affected. In twenty-six cases described by Emmett Holt the inflammation was confined to one joint in five. The arthritis varies in severity; suppurative may occur, but usually the inflammation is comparatively mild, and ankylosis is extremely rare. In the suppurative form the disease progresses rapidly, the skin becomes red early, and great pain, acute tenderness, and distension and flexion of the joint are present, with a temperature of 104° F. The milder cases present articular effusion and pain, but no inflammatory redness of the skin. Muscular atrophy occurs early, and is considerable. The disease may last from ten days to eight weeks. Endocarditis is very rare. Arthritis and peritonitis have been known to occur together.

Prognosis.—Death is rare, except in miasmatic and syphilitic children. As regards complete recovery, the outlook appears to be better than in adults; Lucas, in fact, says that complete resolution may be confidently expected. In cases of gonococcal pyemia the outlook is, of course, very grave.

Diagnosis.—The presence or the history of a recent gonococcal conjunctivitis or vulvo-vaginitis is a very important factor. In older children gonococcal arthritis may imitate acute rheumatism, but salicylates usually fail to influence the disease. Pneumococcal arthritis resembles the gonococcal form in some respects, and, like it, may be polyarticular. Diagnosis may only be possible by microscopical examination of the fluid in the joint. Syphilitic epiphyseal disease may imitate it, but the history and concomitant manifestations should prevent a mistake. Mild cases of gonococcal arthritis may be confused with tuberculous arthritis. In cases of doubt von Pirquet's or Moro's tuberculin reaction should be tried.

Treatment.—In the first place the primary gonococcal infection of the conjunctiva or vulva should be treated. The pain should be relieved by aspirin, and the joint should be kept at rest and applications, hot or cold, applied to it. Benefit can be obtained, especially in chronic cases, by the use of a gonococcal vaccine, preferably autogenous, that is cultivated from the patient. An anti-gonococcal serum has also given good results. In suppurative cases the joint should be opened and washed out (see also Chapter XX., p. 1087).

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ACUTE ARTHRITIS OF TOXIC ORIGIN.

In addition to well-established examples of toxic arthritis, such as gout and that of serum disease, some forms of transient arthritis associated with disease elsewhere in the body are probably of this nature. A. E. Garrod argues that the transient lesions of joints accompanying various erythemas, purpura, and urticaria are, like the cutaneous manifestations, toxic; and Schlosinger believes that the interesting condition of intermittent hydrarthrosis, in which attacks of pain and swelling occur so regularly that they can be accurately foretold, is due to arthro-neurotic oedema. Further, the synovitis of acute rheumatism is so transient and fits so rapidly from joint to joint without doing any permanent damage that, in the presence of the conflicting reports as to the sterile or infected condition of the joints, it is quite reasonable to regard it as toxic. McClure considers the so-called rheumatic arthritis of scarlet fever to be toxic on the ground that blood cultures and cultures from the joints were negative in thirty cases. The deduction that an arthritis is toxic and not infective because the aspirated fluid is sterile is open to the fallacy that the causal organisms may have died out or be in the synovial membrane and so not obvious in the fluid. The characters of toxic arthritis are that the manifestations are transient and slight, and that it fits about from joint to joint.

Gout in children is a curiosity. Sir Alfred Garrod refers to two girls of seven and eight years, both with strong family histories of the disease, and Trouessart

gout is a girl of seven years. The cases of gout in sucklings are probably acute infections. Other manifestations ascribed to gout are not so rare as the arthritic form (see Chapter XI, p. 594).

Serum Disease.—In hospital practice pain and effusion into the joints are rare after subcutaneous injections of antitoxic serum, but it is commoner in patients who, having had prophylactic doses, are up and about (McClure). The pain is not relieved by salicylates, but it is worth while to give calcium salts after the injection, as recommended by Wright, to prevent the reaction. Tixier and Trouner have recorded a case of arthritis during attacks of paroxysmal hæmoglobinuria which they compare with serum disease.

Intermittent Hydrarthrosis not uncommonly begins in childhood. In Blanc's case a girl had the first symptoms at the age of seven, and her mother also was affected. Arsenic appears to be the best method of treatment (Garrod).

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RHEUMATOID ARTHRITIS.

This condition is characterized, as von Velkemann and A. E. Garrod have insisted, by peri-articular or capsular changes causing fusiform swellings, and by the absence of any primary changes in the cartilages and bones. The cases correspond to those described as infective arthritis by Goldthwaite and by the Cambridge committee for the study of special diseases, and are included in their pathological group of proliferative arthritis by Nichols and Richardson. Atrophic changes in the bones are seen late in the course of the disease, hence the name "atrophic arthritis" has also been employed. This group includes the cases in children with enlargement of the spleen and lymphatic glands sometimes described as a separate form of joint affection under the name of *Still's disease*. The reasons for regarding these cases as essentially rheumatoid arthritis are—(1) That the articular changes are the same as in rheumatoid arthritis; (2) that enlargement of the lymphatic glands and of the spleen is not very uncommon in adults with rheumatoid arthritis. Thus, from examination of fifty cases, R. Waterhouse concluded that moderate enlargement of the lymphatic glands is almost constant in the multiple rheumatoid arthritis of young women. The greater prominence of the glandular and splenic enlargement in children is due to the readiness with which the lymphoid tissues react to infection and toxæmia in early life, and not to any specific difference in the disease. Moreover, a case which presented enlarged glands in childhood may in adult life, when the glandular swelling has subsided, be indistinguishable from ordinary rheumatoid arthritis (Garrod). (3) That cases of rheumatoid arthritis in children can be seen (a) without any glandular or splenic enlargement, (b) with glandular but no splenic enlargement, and (c) with enlargement both of the lymphatic glands and of the spleen.

From the category of rheumatoid arthritis are excluded joint diseases due to known micro-organisms, such as the tubercle bacillus, the pneumococcus, gonococcus, streptococci, and staphylococci, the toxæmia of arthritis seen in syphilis and after various fevers, toxic arthritis as in gout and after injections of serum, arthropathies, and the joint changes of hæmophilia.

ETIOLOGY.—In adult life rheumatoid arthritis attacks more females than males, and Diamantherger's 35 cases of chronic arthritis in children were made up of 24 girls and 11 boys; but in 59 other cases which I have collected there were 25 females and 24 males. Inheritance does not exert any obvious influence. The age of onset varies; the disease hardly ever attacks infants under one year, but in a considerable proportion of the sufferers it starts before the end of the third year. In 8 out of Sciff's 12 original cases the disease began within the first three years of life.

Although the exact agent is quite unknown, it is generally considered that the disease is infective in origin, on the grounds that there may be fever, glandular and splenic enlargement, and in some instances leucocytosis and albuminuria. Stress can hardly be laid on the occurrence of some infective disease prior to the onset in about half the cases, for this is common in childhood. It has been suggested that it may be a manifestation of attenuated tuberculosis—is, in fact, a form of the tuberculous pseudo-rheumatism of Poncet (*vide p. 527*); but very few cases supply any evidence in favour of this view, and some cases have been proved to be free from tuberculosis. Possibly various micro-organisms of a low grade of virulence may cause the disease.

MORPH ANATOMY.—There is peri-articular fibrosis with thickening of the capsule and ligaments, and the synovial membrane is vascular and swollen with hypertrophy of its villous processes. In the joints of two severe cases opened during life the synovial membrane is described by Whitman as replaced by soft red granulation tissue. The cartilages are normal or present some pitting at the margins, due to pressure exerted by the enlarged synovial fringes. There is an absence of osteophytic outgrowths and of the fibrillation and erosion of the ventral parts of the cartilages characteristic of osteo-arthritis. There may be adhesions in joints affected severely. The adjacent bone shows rarefaction.

The enlarged lymphatic glands show chronic hyperplasia. The glands in the portal fissure may be enlarged. Pericardial and pleural adhesions may be present. Lardaceous disease of the spleen, liver, and other organs, has been reported (Herrington, Whitman).

CLINICAL FEATURES.—The onset is usually acute and febrile; in some cases it is described as gradual.

The course of the disease is prolonged. Exacerbations of arthritis with fever, especially at night, and swelling, are not uncommon. In other instances the temperature is almost continually raised to 102° to 104° F.

The joints usually affected are the knees, wrists, ankles, interphalangeals, especially of the hand, the metacarpo-phalangeal, and the cervical spine; the temporo-maxillary and sterno-clavicular are not attacked so frequently as in adults. In some instances the large articulations suffer to the exclusion of the smaller. The joints are symmetrically affected, and present, especially those of the hand, a characteristic fusiform appearance which is mainly due to peri-articular

flexion and extension, for there is little effusion into the joint. Skiagraphy shows transparency of the bones and absence of osteophytes. On movement crackling may be felt, but there is no bony grating. The joints are comparatively free from pain and tenderness. Muscular wasting is prominent, and late in the disease contraction may occur. Clabbing of the fingers has been observed.

There is no liability to endocarditis, but a hæmic murmur may appear. As the disease advances the pulse becomes rapid. Enlargement of the lymphatic glands is especially seen in young children. The enlarged glands are chiefly related to the affected joints, but the change may be general. The glands increase in size during the febrile periods when the joint changes are progressing, and subside in the intervals. They may reach a very considerable size, but they never suppurate, and the skin over them is natural. Enlargement of the spleen, which is quite moderate, is also more prominent the younger the patient, and is an early symptom which may disappear. The liver may also be increased in size, and this also may disappear as the acute stage of the disease subsides. Albuminuria and casts occur in a few cases; albuminuria may be constant, or only appear during the febrile exacerbations.

In a collection of 50 published cases of rheumatoid arthritis in children, many of which were probably recorded on account of several exacerbations, enlargement was noted in the glands in 39, of the spleen in 25, of the liver in 8, and 5 had albuminuria.

The children are thin, and often show arrest of development. There is usually some anemia, and leucocytosis has been noted during febrile exacerbations. Wassermann's reaction is negative. The skin is pale and may show freckles. The face has often a waxy paler with a malar flush, and pigmentation is occasionally noticed. Slight exophthalmos is not uncommon. In a few instances jaundice has been present, and some grave cases have had purpura. Sweating may be prominent although fever is absent.

Prognosis is probably better than is usually believed, and recovery may certainly occur. Visceral enlargement is an early sign, often disappears, and is not necessarily a bad prognostic. Improvement was noticed by Still after intercurrent attacks of measles, scarlet fever, and jaundice.



FIG. 157.—CHRONIC ARTERITIS, WITH ENLARGEMENT OF THE LYMPHATIC GLANDS AND SPLEEN.

Note the fusiform swelling of the elbow, wrist, and metacarpal joints. The enlargement of the glands in the axilla is well seen.

DIAGNOSIS.—In the acute phase the disease must be distinguished from rheumatic fever by the failure to react to salicylates and the more marked articular changes. Later it must be differentiated from other forms of chronic arthritis. There is a rare condition of chronic peri-articular fibrosis with rheumatic nodules, which may follow acute rheumatism, Jaccoud's chronic fibrous rheumatism. The occurrence of osteo-arthritis in children is referred to elsewhere (*vide supra*). The rigidity of the spine may suggest curies, and the joint changes and spleen enlargement have erroneously been regarded as sickity.

TREATMENT.—Good hygienic and dietetic conditions are important, and residence in a dry warm climate is desirable. Internally cod-liver-oil, guaiac, iodide of iron, arsenic, and thyroid extract may be tried. The joints should be protected by cotton wool, and may be painted with tincture of iodine, or an ointment of 10 per cent. of oleate of mercury applied. Hot-als, rest, radiant-heat, and electric bulbs may give relief, and Kier's hyperæmic treatment is advisable. Massage is useful in maintaining the nutrition of the muscles. Filicollin has also been advocated, and the application of radium suggested. Antistreptococcic serum appears to be useless.

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OSTEO-ARTHRITIS.

This form of joint disease, characterized by primary changes in the cartilages, pitting and charration of the articular surfaces, and osteophytic formations, is extremely rare in children. It appears to be rather the late result of some destructive change in the joints, such as that induced by leucæmia or inherited syphilis, than comparable to the osteo-arthritis of more advanced life. Congenital syphilis may cause multiple osteo-arthritis with Heberden's nodes, which is not necessarily progressive. Eye changes such as keratitis and chorioiritis are apt to be associated with this form; out of four cases of this kind recorded by Still three had interstitial keratitis. Although apparently occurring in syphilitic subjects, the condition is not benefited by mercury and iodides, and in this respect resembles Charcot's arthropathy.

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SYPHILITIC ARTHRITIS.

The following forms of arthritis may occur in congenital syphilis:

1. In young infants syphilitic arthritis is rare, but it may complicate syphilitic epiphyseal disease (*vide p. 528*). Suppuration in connection with syphilitic epiphysitis may extend into the joint. The occurrence of suppuration is generally

assumed to be due to streptococcal or staphylococcal invasion, but the pus has been found to be sterile (Marfan). This form of suppurative arthritis, which is seldom seen after six months of age, is rarely confined to one joint.

2. Primary gummatous disease of the synovial membrane corresponding to that seen in tertiary syphilis in adults.

3. Allied to this is gummatous disease of the synovial membrane associated with osteitis close to the joint.

4. A form of arthritis deformans with bony changes as in osteoarthritis.

5. Effusion associated with osteitis.

6. Acute synovitis like the form seen in the secondary stage of the acquired disease. With the exception of the first, these forms are rare, and will not be described further here.

7. Chronic effusion, nearly always into the knee-joints, and bilateral, though one knee may be more swollen than the other. This form, first recognized by Britten, occurs between the ages of six and fifteen years. It is specially related to the occurrence of interstitial keratitis, and it has been estimated that 75 per cent. of the cases show this eye change (Dunlop), which usually appears after the synovial effusion. The sexes are equally affected, thus differing from interstitial keratitis which is more frequent in girls.

Our knowledge of the morbid changes is very imperfect. In some instances there has been little or no morbid lesion, in others hyperæmia and thickening of the capsule or small gummata or gummatous infiltration of the synovial fringes have been described. Suppuration never occurs.

CLINICAL PICTURE.—The effusion comes on insidiously with little or no pain, and, unless treated, runs a chronic course. The knees are affected in a very high percentage of the cases; von Hippel found this in 41 out of his 43 cases, and in 35 of the cases the knees were the only joints attacked. The mobility of the joints is little interfered with, and local redness and heat are absent. Usually there is no bony enlargement, but some cases present thickening of the synovial membrane. The condition is probably often regarded as tuberculous, and it has been suggested that this is responsible for the long course of such cases. For if treated with mercury and iodides, they usually clear up in about two months, whereas spirits and rest do not effect any improvement. When specific treatment is imperfectly carried out, a relapse may occur. In rare instances, however, tuberculosis appears to supervene in a joint primarily syphilitic. This complication may explain why some cases do not respond to specific treatment.

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JOINT AFFECTIONS IN NERVOUS DISEASES.

Joint affections occurring in connection with nervous diseases may be thus divided.

(4) Associated with acute diseases of the nervous system. In rare instances acute polio-myelitis is accompanied by arthritis in the paralyzed extremity. One joint only, such as the ankle, may be affected either synchronously with the onset of the paralysis or some weeks later. In another form the small joints of the hand suffer; Coats described this form in a brother and sister. It is open to discussion whether the joint affection is due to an ultramicroscopic organism responsible for acute polio-myelitis or to some other infection of the less resistant joints of the affected extremity. From the presence of pain and loss of power acute rheumatism is very likely to be diagnosed, but salicylates fail to relieve the symptoms. Infantile scurvy in an early stage may also imitate polio-myelitis with arthritic manifestations.

In epidemic cerebro-spinal meningitis there is, in rare instances, intra- or extra-articular suppuration due to the meningococcus. Usually meningococcal arthritis subsides without injuring the joint, and may be hardly noticeable, but sometimes it is a prominent feature. It may be associated with the chronic (posterior basilar) form of meningococcal meningitis, and with pseudo-glioma or the intra-cerebral condition due to the meningococcus which imitates new growth (Coates and Forbes). In rare instances, of which Cecil and Soper have collected five, meningococcal septicaemia is not accompanied by meningitis; it is therefore possible that meningococcal arthritis may occur independently of meningitis.

(5) Associated with chronic nervous diseases. Syringomyelia very rarely occurs in childhood, but the occurrence of arthropathies of the Morvan type has been recorded in children (Clarke and Groves, Fletcher).

Hysterical joints in children are usually seen in girls approaching puberty. The affected joint, commonly the hip, which is free from any swelling or bony change when examined by X-rays, is rigidly fixed by muscular contraction, and is intensely painful until the patient's attention is distracted. There may be evidence of definite neurosis and of cutaneous hyperaesthesia. The diagnosis from early tuberculous disease must be made with great care.

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HEMOPHILIC JOINTS.

Arthritic manifestations are extremely common in haemophilia, and contrast with the great rarity of haemorrhages into other serous membranes.

ÆTIOLOGY.—The liability to these articular manifestations is most prominent in early life, the first attack commonly occurring between the fourth and seventh years, and diminishes with advancing years. The joints suddenly swell as the

result of strain or injury, which may be so slight that the onset is described as spontaneous. The attacks, which may closely resemble ordinary synovitis, are now easily induced at one time than another.

MEANS: ANATOMY.—That hemorrhage occurs into the joints is proved by aspiration during life and by examinations of hemophilic joints after death. King described three stages—hemorrhage (hemarthrosis), inflammation, and deformity. After repeated attacks the synovial membrane presents fringes with a scummed appearance of a rusty colour, due to altered blood-pigment; there are adhesions, the cartilages degenerate, and eventually the changes seen in osteoarthritis result. Sir Almonst Wright suggests, on the analogy of the superficial "serous hemorrhages," that the fluid in the joints is often serous, and not hæmorrhagic.

CLINICAL PICTURE.—The knee is most often attacked, in about half the cases; then the elbow, in about a quarter of the cases; and then the ankle and other joints, including those of the fingers. Several articulations may be affected in the same patient. Three stages have been described: At the onset the joint becomes distended and painful, but not tender, and the skin over it is stretched, but not red or hot. The effusion is soon absorbed, and the joint appears little damaged. In the second stage a form of chronic arthritis has resulted from repeated attacks, and thickening of the synovial membrane and some passive effusion are present. In the third stage the joints become permanently deformed and partially ankylosed.

PROGNOSIS.—The vast majority of bleeders eventually suffer from some arthritic crippling, but the liability to hemarthrosis diminishes as age advances, and the prognosis therefore improves as time goes on.

DIAGNOSIS.—In a known "bleeder" any joint affection should be suspected to be of this nature, and all surgical interference forbidden. Unfortunately hemophiles sometimes conceal their diathesis, and the disease may be regarded as tuberculous, rheumatoid arthritis, or even osteomyelitis. In recent attacks of obscure arthritic swelling the possibility of hemophilia should be considered, and inquiry made on this point before active measures are adopted. As hemophilic joints are particularly likely to be regarded as tuberculous, Moro's tuberculin test, which does not involve puncture or incision of the skin, may be employed in a doubtful case.

TREATMENT.—The joint should be kept at rest, if necessary by splints, and cooling applications or an ice-bag applied. The general treatment of hemophiles should of course be carried out (see also Chapter IX., p. 545).

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CLUBBING OF THE FINGERS.

SYNONYM.—Hippocratic fingers.

DEFINITION.—A swollen condition of the terminal phalanges, with curving of the ends of the fingers and toes; it is almost always symmetrical, and is due to changes in the soft parts.

ETIOLOGY.—It is most commonly due to intrathoracic conditions of an infective nature, such as empyema, chronic pneumonia, fatal bronchitis, and chronic pulmonary tuberculosis. It is seen in cases of congenital heart disease with cyanosis, and may then be associated with an analogous change in the nose and ears. It is also occasionally present in a slight degree in advanced valvular disease due to rheumatic endocarditis. It always accompanies osteo-arthropathy. Clubbing of the fingers is not very uncommon in hypertrophic biliary cirrhosis of the liver with chronic jaundice. It is more often seen in this disease in children than in adults. It may also occur in association with chronic jaundice due to other causes. In rare instances clubbing of the fingers has been observed in sarcoma of the lung, hydatid of the lung, lymphadenoma of the mediastinum, amyloid liver, congenital dilatation of the colon (J. Thomson), rheumatoid arthritis, enterogenous cystosis (Stockvis, V. de Borgh), intestinal toxæmia, and pyelonephritis (Marles). Exceptually the condition has been said to be congenital. It has also been seen in delicate children with defective excretion, but without any evidence of definite disease (Thomson), and it has been noted in perfectly healthy individuals.

PATHOGENY.—The factors most probably concerned in the production of clubbed fingers are—(a) The local action of toxins formed elsewhere on the soft tissues of the nail-bed, or (b) the mechanical stagnation of blood in the nail-bed due to circulatory disturbances such as would result from failure of the right side of the heart, or (c) a combination of (a) and (b). Thus, it has been suggested that cyanosis—for example, that due to congenital heart disease—induces a toxic condition by interfering with the excretion of poisons by the lungs. The question of the causation cannot be considered as settled, for many cases in which the conditions suitable for the production of clubbing are present do not show this change. It has been suggested that the condition is due to trophic impulses conveyed through the nervous system; but, except that neuritis and clubbing of the fingers are in rare cases associated, there is no evidence of this.

MORBID ANATOMY.—There is no change in the bones, and the nature of the alteration of the soft tissues has been variously described. In favour of the view that the swelling of the soft parts is due to vascular engorgement and œdema, it has been stated that the bulbosity can be removed by pressure, and that it disappears in fingers preserved in spirit. Hyperplasia of the soft tissues, enlargement of the papillæ of the dermis, and an increased quantity of fat (Emerson), have also been described.

CLINICAL ASPECT.—Clubbing of the fingers usually comes on gradually, but it has been noticed within two weeks from the onset of the causal disease (West). The fingers are more often and more rapidly affected than the toes; usually all the fingers share in the change, but sometimes the index finger and thumb are chiefly affected, and in rare instances the clubbing is confined to one or two fingers. The terminal phalanges are much enlarged, and their tips are cyanotic. The nails

are curved longitudinally and transversely, are often longitudinally striated, and may be easily brittle. Their growth is more rapid than in health, and they are also peculiar in that the root of the nail is raised and unusually movable. The terminal phalanges have been compared to the bowl of a spoon, to a drumstick, and, when seen in profile, to a parrot's beak. The bulbous fingers are usually free from pain, and there is not any alteration in sensation. When the cause of the condition—for example, an empyema—is removed, the clubbing disappears.

The TREATMENT is that of the cause.

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HYPERTROPHIC OSTEO-ARTHROPATHY.

SYNONYM.—Hypertrophic pulmonary osteo-arthropathy.

DESCRIPTION.—Symmetrical enlargement of the hands and feet, with clubbing of the fingers and toes, periostitic thickening of the long bones, especially in the neighbourhood of the joints, and articular inflammation. It is a secondary manifestation of morbid processes elsewhere, usually in the lungs.

PREVALENCE.—In adults it is about five times more frequent in males than in females, but in children this proportion is not maintained.

P. Marie, who described the condition in 1890, considered that it was always secondary to disease of the lungs, and due to toxins absorbed from lesions in these organs; he therefore called it "hypertrophic pulmonary osteo-arthropathy." Although it is now recognized that it may occur in cases in which the lungs are healthy, Marie's statement holds good in the majority of cases. The intrathoracic conditions with which it is associated are bronchiectasis, chronic pneumonia, chronic pulmonary tuberculosis, empyema, and malignant disease of the mediastinum or lung. In rare instances it has been noted in hypertrophic biliary (Hanot's) cirrhosis, and still more rarely in tuberculous disease of the spine, and intrathoracic lymphadenoma. In some cases the cause has not been apparent.

PATHEMNENY.—It was thought by Marie that the osteo-arthritic changes were due to toxins absorbed from the lungs, and Bamberger (1889), who described the condition before Marie, though less fully, considered that its onset was specially related to the sputum becoming foetid. This explains to some extent why bronchiectasis and pulmonary tuberculosis may be present for years without any associated osteo-arthropathy. In 1896 Thorburn and Westmacott put forward the view that the changes were due to tuberculosis, and Poncet and his followers regard it as a form of tuberculous pseudo-rheumatism—namely, due to toxins produced in tuberculous foci elsewhere.

MORBID ANATOMY.—There is a deposit of new periosteal bone on the shafts of the long bones, especially the lower ends of the radius, ulna, metacarpals, and first two rows of phalanges; less often the lower ends of the humeri, the upper extremi-

tion of the radius and ulna, and the bones of the lower extremities, particularly the tibia and fibula near the ankle-joint, are affected. The shafts may show a diffuse sheath of periostitic bone, but the sheath is thicker near the joints. In some instances this change is seen in other parts of the skeleton. These changes are symmetrical on the two sides of the body. The joints usually contain fluid, and may show definite evidence of inflammation, such as gelatinous thickening of the synovial membrane. There is no bony change in the terminal phalanges, which are in the same condition as in clubbed fingers. The ductless glands are normal. The newly-formed bone is friable, and, like that of ordinary periostitis, is composed of trabeculae at right angles to the long axis of the bone. Rarefaction of the underlying shaft has been described as a distinguishing feature from ordinary osteo-periostitis. Microscopically there is no evidence of tuberculosis.

SYMPTOMS.—Clubbing of the fingers is always present, and in addition there is enlargement of the ends of the bones and prominence of joints, especially the wrists and ankles, and sometimes the knees, elbows, and phalangeal joints. The bony thickening and irregularity may be felt, and the enlargement of the ends of the bones is exaggerated by the swelling of the overlying subcutaneous tissues and by the atrophy of the muscles of the limbs. The joints may be tender, often contain fluid, and their movements are clumsy and somewhat impaired. The course of the disease varies; acute and chronic cases have been described, and not uncommonly there are distinct exacerbations, during which the articulations become painful and swollen, the skin over them red, and the temperature of the body somewhat raised. Excessive perspiration is common. Kyphosis in the dorsi-lumbar region may occur, but is due to pulmonary or other lesions. When the causal condition, such as empyema, is cured, the bony lesions undergo slow resolution.

DIAGNOSIS.—Acroostealgia is very rare in children; it is characterized by elongation of the face and prognathism, and is not accompanied by clubbing of the fingers. From rheumatoid arthritis an X-ray examination will at once distinguish it.

TREATMENT.—It is essential to treat the underlying cause. Pain is not often troublesome, but if so relief may be obtained from hot applications or aspirin.

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CHAPTER XVIII

RHEUMATISM AND CHOREA

ARTHUR P. YOELCKER

RHEUMATISM.

INTRODUCTION.—Of all the diseases to which children are liable, none is more potent in aspect than rheumatism. The varied possession of its multiple manifestations leads us to think of rheumatism as a state rather than as a disease. It is by the study of the condition as met with in children rather than in adults that we can gradually fill in our conception of the rheumatic state. Its manifestations, while of the same nature in childhood and in adult life, present some striking differences in their frequency, distribution, and severity, which will be referred to in the course of this chapter. So widespread and varied are the results of rheumatism in childhood that it is doubtful if an adequate conception of the condition can be gained outside the walls of a children's hospital. The difficulties which present themselves in attaining to anything like a comprehensive view of the pathology, the varied and numerous manifestations of the disease, the limitations of our power to control it, and, finally, our appreciation of the immense amount of injury done both to the individual and to the community by rheumatism, lend to its study in childhood a peculiar interest.

At the present time we have no specific reaction by which we can decide whether a given manifestation is or is not a rheumatic one, and consequently, in the consideration of the symptomatology and treatment, reference will have to be made to the points which appear to justify us in regarding certain conditions as expressions of rheumatism. Rheumatism in childhood runs such a prolonged course that inflammations of certain organs which occur at one time as isolated phenomena, at another appearing in an avalanche of overwhelming and unmistakable manifestations of the rheumatic state, make us prepared on the one hand for the absence of corroboratory evidence at the moment, and on the other hand reluctant to designate as rheumatic a lesion simply because it occurs in association with other manifestations which we already regard as rheumatic. It will be pointed out that there is hardly any isolated phenomenon which can be accepted as undoubtedly rheumatic; and to prove a bacteriological origin for rheumatism, we ought to be able to detect the organism or its products in the lesions. Although this may be possible or long, at the present time there is no method by which we can determine with scientific accuracy the specific (rheumatic) nature of a given lesion. Thus, it happens that the rheumatic nature of some lesions is a matter of controversy, while in the case of others their rheumatic title is based on an almost universal consensus of opinion. At the present time the evidence we can advance as to the rheumatic nature of a lesion is derived from the previous and the family history of

the child, the recognition of coexisting lesions tacitly accepted as rheumatic, and, lastly, from noting the effect of treatment on these lesions.

In the case of children, a knowledge of the previous illnesses, and to a certain extent a knowledge of the family history, will give us valuable help; but it is regrettable that hospital records, from which so much of our statistical information is drawn, should, from the nature of the conditions under which such records are obtained, be of such unequal and doubtful value. This drawback is intensified by the looseness with which the unfortunate term *rheumatic* is so often used as a designation of disorders which have nothing whatever to do with those which are now under consideration. Unless the inquirer has had experience of the manifold and varied aspects in which rheumatism may present itself in childhood, the percentage of cases in which a "rheumatic" previous history will be traced will fall far short of the actual truth. The last method by which we may judge of the rheumatic nature of a lesion—that afforded by the result of treatment—is perhaps the most fallacious; and it will be pointed out that, while certain drugs, such as the salicylates, are invaluable in the treatment of some lesions, they are quite inefficacious in others of an undoubtedly rheumatic nature. The characteristics presented by an adult patient the subject of acute rheumatism are well known, but an attack of acute rheumatism in a child usually presents features which differ widely in the extent to which certain structures are involved, differ in the existence of certain manifestations which are peculiar to children, and differ in the relative liability of certain structures to be attacked at different periods of life, and yet there is no reason to believe that the cause of rheumatism is in any way different at these several periods of life.

Rheumatism in childhood is often a long-drawn-out disease in the course of which many lesions develop, often with so little evidence of active mischief that we are sometimes appalled by the recognition of some grave consequences of the disease which has been developing insidiously and unsuspected. The smoldering form is characteristic of rheumatism in childhood. While in adult life it is the acute joint pain and swelling, with fever and sweating, which characterise an attack of rheumatism, in children these symptoms may be so slight that their very existence is often overlooked; indeed, there may be no local affection of the joints at all.

Ætiology.—Heredity.—Rheumatism is a family disease. The liability to it may be inherited from either or both parents, and when the latter is the case this liability is distinctly increased. With the exception of the case of congenital heart disease—not infrequently the result of an intra-uterine endocarditis—we are not aware of any congenital manifestation of rheumatism.

Age.—Rheumatic manifestations rarely show themselves before the third year of life, though undoubted cases of articular rheumatism have been met with in the second year. Between the ages of four and nine years nearly 80 per cent. of the cases in children first show themselves, three-fifths of these cases occurring between the fourth and sixth years of life, and two-fifths between the seventh and ninth years.

Sex.—The more frequent incidence of chorea in females causes the proportion of female cases over eight years of age to be higher than that of the males; otherwise the two sexes are equally affected.

Race.—Rheumatism is ubiquitous. In England it is met with most commonly among the blonde types—the descendants of Teutonic or Nordic ancestors—

than among the brunette, dark, or Iberian type. Mackintosh has pointed out that this proclivity of the blonde type to rheumatism may be associated with the fact that the evolution of the Nordic races took place under climatic conditions of great dryness, so that in England we find that, while these types preponderate in the southern and eastern districts, yet there are certain damp areas, such as the Fens, the Sussex Weald, and Romney Marsh, where the blonde type is conspicuously absent. Church has called attention to the marked frequency of rheumatic affections at Whitehaven and in the Isle of Man, localities where the men are the most purely Norwegian, the fairest, and almost the tallest, in England. It is here that we find a heavy rainfall—a climatic condition unfavourable to the race—and this reaction to unfavourable climatic conditions is expressed in the prevalence of rheumatism among the blondes of these districts.

Localities.—No locality can claim to be exempt from the incidence of rheumatism; but at the same time it would appear that in the London district rheumatism in children is specially prevalent and frequently of a severe type.

Season.—Rheumatic manifestations are most commonly met with in spring and autumn, May and November being in London the months in which rheumatic manifestations of an articular nature are most frequent.

Exposure to damp does not seem in children to play such a definite part in the production of an attack of rheumatism as it does in the adult; but in childhood the probabilities of exposure to damp beds is naturally less than in adult life, while a wetting by day is less likely to attract attention in a child than in the case in an adult.

Rheumatism undoubtedly may follow as a sequel to scarlet fever, and has been described as a sequel both of measles and of influenza.

Pathology.—The manifestations of rheumatism are generally clear, but the cause is as yet undetermined. All recent investigation tends to include it among the infective diseases, and to attribute to it a microbial origin. The occurrence of pyrexia in the rheumatic state is an argument in favour of a microbial origin, and many of the manifestations of rheumatism are typically inflammatory in nature, and it is to the chemical products of bacterial activity that we look as the chief source of inflammatory lesions. The microbial origin of rheumatism, though it has not yet earned universal acceptance, seems to the writer to be becoming more and more probable, and the claim of Poynton and Paine to have recognized a specific organism—the rheumococcus—derives additional force from the results of the inoculation of animals with the material derived from the effusions of patients suffering from acute rheumatic arthritis. Against the view of a specific organism it has been urged that the disease is the result of the invasion of the tissues by different members of the streptococcal groups. Rheumatism has by many been regarded as a mild form of pyrexia; but, as has been pointed out by Poynton, it is very rarely that rheumatism in the true sense of the word arises in the course of a true pyrexia, though acute rheumatism is a relatively common disease. The occurrence of very severe and often fatal cases of rheumatism is not in keeping with the phenomena which we should expect as the result of the invasion of the body by an attenuated non-protecing organism.

The existence of a specific organism is not yet accepted as proven, but in spite of the variety of organisms, each of which has been claimed as specific, the *Diplococcus rheumaticus*, described by Poynton and Paine in 1900, seems to have fulfilled Koch's postulates more nearly than any of its competitors. It has been obtained

from the blood, from the pericardial effusion, and from the effusions into the joints, in cases of rheumatic fever in the human subject, and when injected into rabbits and monkeys, after cultivation outside the body, has produced in them endo- and pericarditis and multiple arthritis. From these lesions the organism has been recovered and proved identical with that originally isolated from the rheumatic patient.

The organism is widely distributed throughout the tissues in the course of an attack of acute rheumatism, and is found more abundantly in the tissues themselves than in the inflammatory exudations in the joints and serous cavities—a fact which may explain the frequent failure of investigators to recover an organism from the pleural, joint, or even pericardial effusions in acute rheumatism. The effect of the presence of the organism and its products is to cause a reaction on the part of the tissues in which they are situated, and thus we find the chief lesions to be inflammatory exudations, degenerative changes in the tissues, and proliferative changes leading to the formation of new connective tissue.

The nature of the changes in the tissues will depend on the violence of the organism, the time for which it acts, and the resistance possessed by the individual. Where resistance is high, the micro-organisms are rapidly destroyed after producing effusion around the bloodvessels and into the serous cavities. The effusions are accompanied by degenerative changes in the connective tissue, and these may either resolve without cell necrosis or the cells may be replaced by some form of connective tissue. The minute changes observed as the result of rheumatic infection will be described under the various lesions produced by rheumatism (e.g., pericarditis, endocarditis, myocarditis, chorea), but a description of the histology of the rheumatic nodule is here given as affording a type of the changes which are commonly met with.

The subcutaneous nodule is an undoubted manifestation of rheumatism, and is rare apart from this condition. As pointed out by Chenebise, the structure of the nodule is practically identical with that of the structure of a valve of the heart when it is the seat of a rheumatic valvulitis. A rheumatic nodule consists of a central portion composed of fibrin surrounded by a zone of cells, and this in turn is succeeded by definite fibrous tissue with swollen connective-tissue cells, showing bloodvessels which are often tortuous and distended with blood. Poynton has demonstrated the presence of the *diplococcus rheumaticus* in the nodule. Poynton and Still contend that the formation of fibrous tissue in a nodule is a late phenomenon, and that the fibrinous and cellular exudations precede the formation of actual fibrous tissue—a fact which must be borne in mind when visualizing the changes which are occurring in the valves of the heart during a rheumatic valvulitis. Bolloch denies the fibrinous nature of the centre of the nodule, which he regards as a form of coagulation necrosis.

While it is true that Poynton and Paine have succeeded in demonstrating the presence of the *diplococcus rheumaticus* in the tissues of patients suffering from acute rheumatism, it must be borne in mind that other capable observers have altogether failed to confirm these observations. Bolloch and Thorpe, in twenty cases of acute rheumatism which they examined carefully, some during life and others after death, failed altogether to find any organisms, even when rheumatic nodules removed during life were the tissues examined. It is difficult to assume that the organism is present in some cases only, and that one observer should find the organism in thirty-two cases, while another should fail to find it even once in twenty cases.

SYMPTOMATOLOGY.—Pyrexia.—Rheumatism, like other diseases due to an invasive organism, is a pyrexial disease, but the degree of pyrexia varies with the particular organs which happen to be the seat of the rheumatic process. Most commonly we find that cases characterized by inflammation of the joints, by tonsillitis, and by serous-membrane inflammations, are accompanied by a considerable elevation of temperature; while in the other instances a much lower degree of pyrexia may be the only discoverable evidence of the activity of the rheumatic poison. In such cases the range of temperature is usually only about $1\frac{1}{2}^{\circ}$ (98° to 99.5° F.), which on the chart shows a remarkably regular saw-like form. Such cases of protracted pyrexia are sometimes accompanied by marked anemia.

In what is usually recognized as an attack of rheumatic fever, in which we have joint pains and joint effusions, the temperature is as a rule high (102° to 103.5° F.), but with rest in bed and treatment with salicylates it generally falls, so that by the third day it has reached 99° F. or lower. Pyrexia, however, is not an invariable accompaniment of the rheumatic state, for the occurrence of various skin manifestations of rheumatism, the onset and presence of chorea, and even the appearance of a crop of rheumatic nodules, may be unaccompanied by any rise of temperature. As a rare occurrence, hyperpyrexia may be met with in children in the course of rheumatic fever, generally in association with pericarditis or with chorea.

While the pyrexial period in a case of articular rheumatism is short, it must be remembered that the rheumatic state is, generally speaking, a pyrexial state, and that the pyrexia may be prolonged through weeks, and even months, in spite of rest and what is termed "anti-rheumatic treatment." This condition is frequently associated with anemia, nodule formation, and an established endocarditis, though it may be impossible to trace any progress in the cardiac lesions.

See Throat.—This, as was pointed out by Trousseau, and later by Kingston Fowler, is commonly the earliest manifestation of rheumatism. The tonsils and pharynx are red and tender. The inflammation is parenchymatous rather than exudative, and abscess formation is rare. Follicular tonsillitis may, however, be met with. After two or three days the throat symptoms subside, but pains about the joints and in the limbs are replaced by definite swelling and tenderness in the joints.

Attacks of tonsillitis are by no means confined to the onset of an attack of rheumatism, but may occur during the course of the disease, when as a rule they last for four or five days.

Epigastric Pains.—Among the early symptoms of a rheumatic attack in a child may be mentioned the occurrence of severe pain in the epigastrium. This may precede the sore throat, and may be unaccompanied by any evidence of cardiac dilatation, pericarditis, or pleurisy, and may be met with when no drugs have been administered. The pain is definitely epigastric, but not attended by as much tenderness as usually accompanies a rheumatic fibrositis or myalgia, nor is it associated with that intolerance of food which we should expect if acute gastritis were present.

Joint Pains and Effusions.—Pain in the joints, which is often so conspicuous a sign in the rheumatism of adults, is of variable severity; in fact, it is possible that acute rheumatism may run its course without there being any appreciable involvement of the joints. The pains may be mentioned by the child or its parents as merely "growing pains," and are referred most commonly to the knees or to the thighs, or we may meet with the most exquisite tenderness of the joints, so that the child dreads the approach of anyone to the bedside. The joints most

commonly affected are the wrists, knuckles, ankles, knees, shoulders, and less commonly, though more often than in adults, the hips. When the knees are affected, the pain is referred either to the front of the joint over the middle of the patella, or else to the back of the knee at the insertion of the hamstring tendons. When the hips are affected, the child usually lies with the thighs flexed, and the pain is generally severe.

The joints are swollen; sometimes they have a pinkish blush over them, look puffy, and are very tender. When the knuckles are affected, the digits are kept flexed. The knees when affected are kept extended. The amount of effusion varies partly with the severity of the attack, and also largely with the amount of movement to which the affected joints have been subjected. The amount of fluid is rarely so great as to call for its removal by aspiration. The joint effusions are usually multiple, and, although present to varying degrees in several joints, the statement that effusion in one joint may rapidly disappear, to be replaced by the fresh involvement of another joint, does not hold good for the majority of children. Effusions are found, not only in the joints and the surrounding soft tissues, but also into the bursæ in the neighbourhood of joints, especially in the case of the knuckles, and into the sheaths of the tendons, especially the flexor tendons of the fingers and wrists. Effusions into joints tend under rest and treatment to subside rapidly, so that within three days no fluid can be detected. Sometimes, however, the fluid persists in spite of rest and treatment with salicylates, and then it will generally be found that there has been some traumatism, such as may have been brought about by the child getting up and using the inflamed joint, or that there is an additional infection, such as gonorrhœa. Effusions into the peri-articular tissues, the bursæ, and tendon sheaths, undergo absorption more slowly than those into the joints.

The significance to be attached to the existence of growing pains has given rise to much difference of opinion, some actually regarding them as normal occurrences, others seeing in them unmistakable evidences of rheumatism. They are certainly of more common occurrence in children who develop other rheumatic manifestations than in those in whom these signs are wanting. They are quite distinct from the true arthritic pains, and are usually referred to the fronts of the thighs and to the shins. They are not attended by any recognisable effusion or periosteal thickenings.

Skin.—Rheumatic children have, in the absence of anaemia, a delicate, clear skin, with a pink flush on the cheeks, which, while not pathognomonic of rheumatism, is certainly suggestive.

Free perspiration, which is a marked and constant occurrence in the acute rheumatism of adults, is in children rare, but there is often a cold clamminess of the palms and soles, accompanied by a distinct dryness of the rest of the surface of the body. Profuse sweating during an acute attack of rheumatism is met with only in older children.

Rheumatism in childhood has more numerous and frequent skin manifestations than in adult life. The cutaneous eruptions are erythematous and purpuric. The erythematæ may be erythema simplex, urticaria, erythema papulatum, erythema marginatum, erythema multiforme, and erythema nodosum. These eruptions are variable in their occurrence, and it is not unusual to come across quite a number of cases in a short space of time, and then to see none for a prolonged period. This suggests that there is a seasonal variation in their oc-

erance. Some children are more prone than others to exhibit the cutaneous manifestation of rheumatism, and it is not uncommon to meet with cases which have been repeatedly suspected of scarlet fever on account of the similarity which a rheumatic rash may bear to the exanthema of scarlet fever. In my own experience rheumatic skin manifestations are less common than they were twenty years ago—a fact which cannot be explained as being due to the influence of salicylate treatment.

Erythema nodosum is the most common form of rheumatic erythema. In such cases we may find a simple erythema over the face, associated with a marginate erythema which often commences at the inner edge of the palms and soles and spreads over the limbs, or may be situated over the cheeks and trunk. Associated with this there may be a papular erythema, often best marked in the neighbourhood of the elbows and knees. These rashes may persist for days or be quite transient. When the former is the case, there is often a well-marked brown staining left. Urticaria is not uncommon, and the wheals may be large, but as a rule there is not as much irritation as in the case of urticarial eruptions of a gastro-intestinal origin.

The relation of *erythema nodosum* to rheumatism is much debated. It is true that *erythema nodosum* is rarely seen during an attack of acute articular rheumatism, and it is also true that a very large number of cases in which it occurs do not present any evidence of endocarditis; yet in a large proportion of the cases it is easy to trace a strong rheumatic history either in the patient or in near relatives. *Erythema nodosum* occurs in children chiefly over the fronts of the shins, sometimes reaching up to the thighs, but rarely appearing on the trunk or face. On the upper limbs it is usually confined to the backs of the forearms and of the arms, just above and below the elbows. The patches are in the early stages bright pinkish-red in colour, raised, and frequently, though by no means always, exquisitely tender to touch. As they get older they become a dusky red, then a purple, and gradually a greenish-brown colour. The outbreak of the eruption may be heralded by malaise, sore throat, joint pains, and pyrexia; but in other cases attention may only be called to its existence by the local eruption. *Erythema nodosum* appears in crops, with frequently an interval of five to seven days between the individual outbreaks. Such crops may come out over a period of several weeks, or the whole attack may be comprised in a single outbreak.

In addition to the various erythemata, purpuric eruptions are met with in the course of acute rheumatism, as well as apart from other simultaneous rheumatic manifestations. Although not uncommon in rheumatic children, it must be remembered that purpura is an accompaniment of other conditions which are not rheumatic. Thus we find a "symptomatic" purpura occurring in malarial, in the acute infective fevers, scarlet fever, measles, and smallpox; in pyæmia, in septicæmia, as well as in renal disease, and in various forms of anemia; and also we cannot regard these as different varieties of purpura, we are hardly justified in recognising a special form in association with rheumatism. The association of purpura with rheumatism may be partly explained by the not infrequent association of purpura with malignant endocarditis; but even excluding such cases, there still remain those in which purpura is ushered in by an attack of sore throat, joint and limb pains, and slight pyrexia. But something more is required before we can accept such a purpuric rash as a rheumatic manifestation. An examination of the fatal cases of purpura occurring in the Hospital for Sick

Children (excluding those cases where purpura was only a terminal incident) showed that in those fatal cases there were no evidences of any endocarditis—an indication, though no proof, that rheumatism was not present. Until, however, we have some means of testing the rheumatic or non-rheumatic nature of a case, it is open to the individual to form his own views as to the rheumatic nature of purpura. The writer's views are against its recognition as a distinctly rheumatic manifestation.

As a rare occurrence, herpes, and also a bullous erythema, have been met with in the course of rheumatism in children.

Cricker regarded rheumatism as a predisposing cause of psoriasis, but the two conditions have not, in my experience, been at all frequently associated in children.

Nodules.—The true rheumatic nodule described by Barlow and Warner in 1881 is an undoubted rheumatic manifestation. Not only is it pathognomonic of rheumatism, but it also indicates a grave form of it, since it is found almost



FIG. 128.—RHEUMATIC NODULES ON THE KNEE.

invariably associated with evidence of peri- or endocarditis. Out of hundreds of cases which have been under observation at the Hospital for Sick Children, the writer knows of only one in which peri- or endocarditis was not present.

The nodules are found beneath the skin in connection with the fibrous tissue, but are also present in some cases in the deeper structures, such as the pericardium. The favourite sites are the backs of the elbows, the fronts of the knees, the knuckles, over the styloid processes of the radius and ulna, along the tendon of the palmaris longus, along the tendons of the flexors and extensor tendons of the fingers. In the lower limbs they are found chiefly over the knees, over the malleoli, the extensor tendons of the toes, over the tendons of the tibialis anticus and the peroneal tendons, along the tendo Achillis, and over the fascia lata of the thigh. On the trunk they are seen over the vertebral spines, over the spine and the vertebral border of the scapula, over the acromion, and occasionally over the clavicle. They are also seen over the iliac crests. In rare cases they are to be felt in the axilla

origin of the pectoralis major and in the tendinous intersections of the rectus abdominis. In the head they are frequently present, though often overlooked, in the aponeurosis of the occipito-frontalis, and most commonly over the attachment of the occipito-frontalis to the occiput. The helix of the ear at its outer margin and posterior and median aspect is also sometimes the seat of rheumatic nodules. On three occasions the writer has demonstrated their presence in the pericardium.

The nodules vary in size from that of a hemp-seed to that of a walnut. They are irregularly spherical in shape, and can sometimes be felt to have lateral prolongations. They are movable under the skin and over the bones. They are generally quite painless, though exceptionally somewhat tender. They may appear within twelve hours, and disappear within twenty-four, or may persist for weeks, or even months, and then disappear quite suddenly. They appear in crops, and their appearance may or may not be accompanied by pyrexia. Exceptionally the nodules may be surrounded by a hemorrhagic extravasation. To the naked eye a nodule appears as a pinkish-white mass, having whitish prolongations like



FIG. 159.—RHEUMATIC NODULES ON THE ELBOW.

lateral rimming from the central mass, and gradually lost in the surrounding connective tissue. The nodule is jelly, and an attempt to dissect it out often leads to its gradual disappearance.

The prevalence of nodules among children as compared to adults is one of the striking differences between the rheumatism of the child and that of the adult.

In addition to the actual formation of nodules in the connective tissue, rheumatism affects the fibrous tissues about the joints and the fascia, giving rise to the stiff neck which is so frequent in children.

Muscle wasting, especially in the forearm and small muscles of the hand, is not infrequent as the result of an attack of rheumatism and after severe attacks of chorea. The wasted muscles on the front of the forearm give that part a scooped-out appearance, and the wasting of the dorsal interossei, especially of the first, is often conspicuous.

Anemia is a marked result of nearly every severe rheumatic attack. The blood counts in acute rheumatism show a distinct diminution in the number of the red cells, but leucocytosis is but little marked. Cases in which nodules appear,

and those in which there is prolonged, though possibly but slight, pyrexia, are characterized by marked anemia.

Epistaxis is not infrequent in rheumatic children, and is met with chiefly in cases in which there is rheumatic endocarditis. Epistaxis is rarely severe, and is more common in girls than boys. Its presence should always lead to a careful examination of the heart for evidences of endocarditis.

Thrombosis of the veins has been shown by Chaddock, Lees, and Garrod, to occur as one of the rarer manifestations of rheumatism, and Gistay has ascribed this to a rheumatic phlebitis, though the alterations in the blood and in the circulation may largely contribute without the intervention of a specific phlebitis. The thrombosis occurs chiefly in the veins of the lower limbs and in those of the neck. When the latter veins are the seat of thrombosis, there is oedema of the face and neck, with cyanosis, a condition of mental apathy, and tenderness extending the head. The thrombosed jugulars may be sometimes felt as cords. The writer has recently had under his care a very severe case of chorea in a boy of thirteen, who developed thrombosis in both his femoral and saphena veins. He had no endocarditis, and made a complete recovery.

Pericardium.—The pericardium is liable to inflammation in the course of an attack of rheumatism, and the exudation may be serous, hæmorrhagic, fibrinous, or purulent. The serous and fibrinous are the commonest forms. The onset of pericarditis may be marked by a rise in temperature, vomiting, distress culminating in delirium, dyspnoea, restlessness, precordial or epigastric pain, and a rise in the pulse-rate. A posture very suggestive of the occurrence of pericarditis is one in which the child sits up in bed, leaning forward over the bed-board. Pericarditis may, however, not give rise to any marked symptoms, and friction may be discovered merely in the course of a routine examination of the heart. Pericarditis is more commonly met with in the rheumatism of children than in that of adults. It is frequently recurrent. Resolution of the inflammatory products may occur, but complete adhesion of the layers of the pericardium (strangled heart) is not infrequent. Nodules may be found in the pericardial walls without the presence of active pericardial inflammation. Speaking generally, pericarditis is a complication less immediately grave in children than in adults.

Myocardium.—The rheumatic poison acts markedly on the myocardium, causing a carditis which is evidenced clinically by dilatation of the heart, often of very rapid onset, an increased rate of the heart-beats, and a diminished power of the ventricular contraction. This carditis may terminate fatally or may lead to fatty and fibroid changes in the heart muscle.

Endocarditis.—The rheumatic poison has a special tendency to cause inflammatory changes in the endocardium, both of the valves and of the cavities of the heart; of these the valves are the more liable to damage, and the valves of the left side of the heart more than those of the right. Affections of the heart valves occurring in intra-uterine life are frequently right-sided, and it is found that a considerable number of cases of congenital heart disease occur in families in which a history of rheumatism can be traced.

Rheumatic valvulitis is most common in the mitral valve. Various murmurs are heard as the result of mitral valvulitis. A systolic murmur due to mitral regurgitation is by far the most common. A mid or early diastolic murmur is usually attributed to mitral stenosis, but is in the writer's opinion due to thickening and rigidity of the mitral cusps without stenosis of the mitral orifice. Preystale

murmurs are sometimes due to true stenosis, but sometimes are heard where there is actual dilatation of the mitral orifice. Various combinations of these murmurs may be heard, and in children we must always be prepared to find a dilated mitral orifice in cases in which we have felt a presystolic thrill and heard a presystolic, a mid-diastolic, or even a late diastolic, murmur. Systolic thrills due to mitral regurgitation are more commonly met with in children than in adults, but pure presystolic thrills are much rarer than in adults.

The aortic valve is by no means exempt from rheumatic endocarditis, but is usually affected in association with the mitral valve, rarely alone. As a general rule, the older the child the greater is the risk of involvement of the aortic valve. While mitral stenosis is not uncommon in childhood, aortic stenosis is rare, sclerosing endocarditis leading rather to insufficiency than to obstruction.

When an infective endocarditis is grafted on to a simple rheumatic endocarditis, we may find the tricuspid and pulmonary valves involved, and in the terminal stages of an acute rheumatic endocarditis recent vegetations are often found on all the valves of the heart in addition to the changes produced by previous attacks. Evidence of endocarditis in children constitutes one of the most frequent and weighty justifications for the diagnosis of the existence of rheumatism.

Pleuræ.—Attacks of pleurisy are not uncommon in the course of rheumatism, and may or may not be accompanied by a recognizable amount of fluid effusion. If present, the exudation is serous rather than purulent. Attacks of pleurisy are more common on the left side, and may be associated with peri- and endocarditis. A rheumatic pleurisy is rarely attended by much rise of temperature, but the pain may be severe at the onset of the attack. Friction is rarely heard over a large area in the attacks. The amount of fluid is seldom large. The diplococci of Poynton and Paine have been found in the exudation.

Lungs.—The rarity of manifestations of rheumatism in the lungs presents a marked contrast to their frequency in the heart. Some cases of pneumonia are attributed to rheumatism, but these have generally occurred in the subjects of sub- or pericarditis, in whom we are not unprepared to find a general lowering of resistance to invasion by infective agents. Poynton and Paine have isolated a diplo-streptococcus from the lungs and pleura, in a fatal case of broncho-pneumonia, which morphologically and in its behaviour on injection was different to the pneumococcus, and have found that this organism produces in animals broncho-pneumonia as well as arthritic, endo- and pericardial manifestations similar to those found in rheumatism.

Acute oedema of the lungs may occur in the course of peri- or endocarditis, but its purely rheumatic nature is questionable. It is certainly a rare phenomenon.

Abdomen.—Reference has already been made to the abdominal pain met with at the onset of a rheumatic attack. Vomiting is not infrequently met with in the course of an attack of rheumatism, and may be due (1) to the rheumatic poison; (2) to peritonitis or toxicilitis; or (3) to drugs, especially the salicylates or calomel used in the treatment of the disease.

Peritonitis is so rare a manifestation of rheumatism in children that it is a practically negligible event, except when it occurs as a result of extension from the pericardium or pleura, from the appendix, or from an infarct in the spleen, kidney, or mesentery. The vermiform appendix is supposed to be a not infrequent seat of rheumatic inflammation. In some cases the diagnosis of the rheumatic nature of an attack has been based on the alleviation of the symptoms brought about by

the administration of the salicylates—a very fallacious piece of evidence; in others the history has pointed to a rheumatic inheritance. It is rarely, indeed, that an acute attack of appendicitis is met with in the course of acute rheumatism. It is interesting to note that quite recently Poynton and Paine have produced in rabbits an experimental appendicitis as the sequel to the intravenous injection of the diplococcus rheumaticus, and that in the lesions in the appendix this organism has been demonstrated.

Nephritis may be a sequel to an attack of acute rheumatism, though it is an uncommon event. An acute nephritis occurring in the course of an attack of rheumatism should always give rise to the question of the attack being in reality one of scarlet fever. Apart, however, from such cases, a certain number of cases of acute nephritis do occur in children with joint pains, joint effusions, and endocarditis or chorea, and the attacks are accompanied by oliguria, edema, a considerable amount of blood in the urine, with epithelial and blood casts, which would negative the view that these signs were only the result of renal infarction.

Peritonitis occurs much more rarely in children than in adults, and is generally met with in association with erythema nodosum or with outbreaks of rheumatic nodules.

Meningitis has occasionally been met with, either unilaterally or bilaterally, even before the time of puberty in rheumatic children, but is a rare occurrence.

Nervous System.—Reference to the mental peculiarities of the rheumatic child will be made in the section on Chorea.

DIAGNOSIS.—Under the head of diagnosis we must consider first the diagnosis of acute rheumatism, and secondly the diagnosis of the rheumatic state.

Acute Rheumatism.—As a rule there is not much difficulty in recognizing an acute attack when articular signs are present, but, as we have seen, these may be very little in evidence. When articular symptoms are present, the condition may be simulated by an acute anterior poliomyelitis, for in some such cases there is, in addition to the pyrexia, sore throat, and sickness, a very marked tenderness of the joints, especially of the knees, so that the child dreads being moved or touched. It is, naturally, no easy matter to investigate the condition of the knee-jerks under these circumstances, nor is the absence of a knee-jerk by any means an essential sign of anterior poliomyelitis. The chief differences between the two conditions are that in poliomyelitis there is no effusion into the joints, in spite of the great tenderness; that the pain is not spontaneous but caused by movement; that the knee-jerks are frequently abolished if the lower limbs are affected; and perhaps the most useful point is that the tenderness is not appreciably affected by the administration of salicylates. The absence of a history of previous attacks of rheumatism or of any evidences of endo- or pericarditis may all contribute to establish a correct diagnosis.

Acute osteomyelitis may simulate acute rheumatism, especially in younger children. Here, however, the constitutional symptoms are more severe, the tenderness is great and is extra-articular, and rigors or convulsions may occur. The severity of the child's illness, associated with a local swelling and tenderness, should arouse suspicion, and the failure to obtain relief from the administration of salicylates should warn us of the true nature of the disease. Expectancy and the tentative administration of drugs will not justify the omission to examine the affected part with a mind open to the possibility of the existence of an infective osteomyelitis, and the existence of a cardiac murmur must not be allowed to prejudice us entirely

in favor of rheumatism. Rheumatic periostitis is so rare in children that an exploratory incision would be preferable to an unincised periosteum in a case of infective osteomyelitis.

Acute epiphysitis, due either to syphilis or to some septic infection, or occurring in the course of an attack of scurvy, may give rise to conditions which are liable to be mistaken for rheumatism. In the case of syphilis the temperature is not much raised, and the tenderness is evoked on movement, the child otherwise lying quite still (pseudo-paralysis of Parrot); there is no effusion into the joint; the child may present other evidences of syphilis, and may give a positive Wassermann's reaction. In an acute septic epiphysitis the constitutional disturbance is greater and the temperature is higher.

Scurvy is a condition not likely to be confounded with rheumatism, for the condition occurs at an earlier age than that at which rheumatism generally makes its appearance. The warty appearance, tender limbs rather than tender joints, the swelling of the gums, and the slight degree of pyrexia, all indicate the true nature of the attack.

Acute pyrexia with suppurative arthritis is a rare condition, in which the swollen joints bear some resemblance to the swollen joints in a rheumatic attack; but here the constitutional conditions are much more severe, and there is usually some obvious source of pyæmic infection. Rigors, which are rare in children, are met with in pyrexia, but not in rheumatism. It is, of course, conceivable that any lesion to a joint causing effusion might be mistaken for rheumatism, but practically the above-mentioned conditions are the only ones of real moment. While this is true of the joint condition only, we may experience some difficulty in deciding between an attack of scarlet fever—accompanied, as it often is, by joint pains—and the onset of a simple rheumatic attack; and the same difficulty may confront us with the differential diagnosis of tonsillitis at its outset. Reference has already been made to the relation of these two diseases to rheumatism, and a correct diagnosis may be possible only by waiting.

It must be borne in mind that sometimes an attack of acute appendicitis may be associated with severe tenderness in the region of the right hip-joint, a possibility which has only to be mentioned to put the physician on his guard against it.

The diagnosis of the Rheumatic State opens up such a vista of possibilities that one is tempted to suspect that a claim will be advanced for "rheumatism" in childhood which shall occupy the place and command the same satisfaction as the diagnosis of "gonorrhea" commands in the adult, especially among the laity. The recognition of the rheumatic state will be determined from the family and previous personal history of the child, and from the irregular manifestation of now one, and now another, facet of that many-sided body we designate "rheumatic." Clammy hands and feet, a liability to sore throats, growing pains, stiff necks and joints, the occurrence of one or more of the rheumatic rashes—all these met with in bright, sharp, intelligent children lead one to apply to them the unenviable distinction of being rheumatic. Apart from nodules, I do not think that we can regard any one of the manifestations described above, when taken alone, as an undoubted evidence of rheumatism; but the general tendency of the rheumatic mosaic to piece itself bit by bit, and so to complete the picture, often enables us to recognize under the guise of a simple ailment a portion of the rheumatic state. No one piece of the mosaic taken alone may bear the mark of its true nature, but when

pieced together the whole presents an unmistakable portrait of one of the most serious affections to which the period of childhood is liable.

Endocarditis alone need not be rheumatic; sore throat also need not be rheumatic; but if we find the two together the likelihood of their being both rheumatic manifestations is enormously increased. It is on this account that particular attention must be paid to the investigation of the history of rheumatic cases. Under the head of Treatment it will be shown that, while some rheumatic affections respond promptly to the salicylates, yet there are others of undoubtedly rheumatic nature which are quite uninfluenced by these drugs, so that we cannot accept the response to salicylic treatment in any way as proof of the rheumatic nature of a symptom or sign.

PROGNOSIS.—An attack of acute rheumatism in a child is always an event of grave potentiality. If, fortunately, it does not itself leave residual damage, yet it is an indication of a susceptibility to subsequent attacks, or, what is much more often seen in children, to subsequent manifestations of rheumatic invasion. It is frequently the apparently trivial nature of some of these attacks which leads to the ending of the patient, for early evidences of cardiac disease are not sought for, and are consequently unrecognized until well established, and by this time they have as a rule become incurable. No rheumatic manifestation in a child can afford to be neglected, and a careful watch must be kept on the heart and pericardium, these being the parts where mischief is most liable to be permanent, serious, and established insidiously. Paradoxical as it may sound, there is greater safety in a definite acute rheumatic attack, when carefully treated, than in many of the so-called "slight" attacks, and perhaps the most dangerous of all are the sneaking attacks in which there is evidence of some general infection, shown by continued, though slight, pyrexia, anemia, and transient joint-pains and sore throat. It is in the course of these that we too often discover that endocarditis has been developing, or that an old endocardial lesion which had been stationary has become active again. The immunity of some cases to the sequelæ of rheumatism is very striking, some children passing through four or five attacks of acute rheumatism and yet escaping without any cardiac involvement. The severity of an attack, if judged only by the articular involvement, the degree of pyrexia, and the discomfort of the patient, affords no true measure of the real liability to permanent mischief, and we must explain the too frequent occurrence of an established endocarditis by the fact that its onset was attended by symptoms insufficiently prominent to call serious attention to the possibility of its existence. From these remarks it will be evident that one of the factors in the prognosis of a rheumatic attack is the recognition of the possible complications, and the adoption of methods of treatment calculated to diminish the liability of their occurrence.

Hyperpyrexia is so rare in children that it is practically a negligible factor in prognosis. Although persistent peri-articular damage may result, yet the other manifestations of rheumatism all tend to clear up without leaving permanent damage behind. The influence of the occurrence of chorea will be treated of under Chorea.

The prognosis of rheumatism is to be expressed in terms of the liability to heart mischief and the nature of that mischief. These in turn depend on (1) the age, (2) the inheritance, (3) the surroundings, and (4) the treatment, of the patient.

Age.—The younger the age at which definite rheumatic manifestations appear, the worse is the prognosis, and the greater the liability to heart involvement.

Pericardial inflammation is more frequent in children than in adults, but less commonly met with under the age of seven than above it. Myocarditis leading to acute dilatation, as has been pointed out by Lees, is a cause of a certain number of deaths in the course of an acute rheumatic attack, sometimes even in the first attack. Endocarditis is rare under four years of age, though by no means unknown. The existence of a valvular lesion is serious, partly in relation to the age at which it appears, and partly to the seat of the lesion. The earlier in life a valvular lesion becomes established, the worse is the outlook. With regard to the influence of the site and nature of the lesion, reference must be made to the chapter on Diseases of the Heart (see Chapter VIII., p. 451).

Inheritance.—The question of heredity has a bearing on prognosis in that the liability to rheumatic invasion is one which is distinctly inherited; or, to put it in another way, resistance to rheumatic invasion is lowered if one of the parents is rheumatic, and markedly so if both are. The surroundings of a patient also enter into the question of prognosis. The terrible prevalence of endocarditis among the children of the poor, compared to its relative infrequency among the children of the well-to-do, points to the influence which surroundings, and especially the absence of adequate rest during convalescence—or, for that matter, actually during an attack—exert on the occurrence of heart complications in rheumatism. The influence of climate has been pointed out under the head of aetiology, but it should be remembered that in the course of an attack of endocarditis the existence of unhealthy surroundings, such as bad drains, escaping gas, as well as those sources of infection in the patient himself, such as bad teeth, unhealthy gums, chronic pharyngeal catarrh, otitis, or vaginitis, may cause an ep infection which may possibly have a fatal termination.

Apart from these general considerations as to prognosis, we shall do well to remember the significance which must be attached to the presence or history of the existence of nodules. No sign of rheumatism could be less propitious than the appearance of nodules. Nodules are almost always accompanied by evidence of endo- or pericarditis; and if these are fortunately absent, yet the prognosis in such a case should be none the less grave, since the probabilities of heart disease developing are enormous. Rheumatism with nodules is not immediately, but remotely, the worst form in which rheumatism presents itself.

Apart from the existence of nodules, those sneaking forms of rheumatism which are attended by involvement of the fibrous tissues, especially about the neck, are cases in which the outlook is not good. The more the stress of rheumatism falls on the joints, the less serious is the prognosis.

The existence of skin eruptions does not aid us in a prognosis, though the writer would again call attention to the relative infrequency of heart affections with erythema nodosum.

TREATMENT.—The treatment of rheumatism may be considered under two heads—(1) that of the general rheumatic state and (2) that of the individual manifestations.

If a child is known to be of rheumatic stock, it is well to take special precautions before any manifestations have shown themselves, for we are by no means possessed of an antidote for the rheumatic poison.

Clothing.—Rheumatic children should wear flannel in some form next to the skin. The underclothing should extend from the ankles to the clavicles, and down the upper limbs as low as the middle of the forearms. Bare legs should be taboo.

There is no advantage, but a positive disadvantage, in heaping layer on layer of garments on a child because it is "rheumatic." Some children experience very positive discomfort from the presence of woollen garments next to their skin, and in them silk or a mixture of silk and wool may be adopted. As has been pointed out, the soles of the feet are usually clammy, so that woollen socks or stockings must be worn.

Residence.—A clay soil is recognized as a favourable condition for the production of rheumatism; it should certainly be avoided. River valleys, low-lying situations, and places where there is a low level of ground water, are also held to be favourable to the development of rheumatism, and consequently situations to be avoided. On the other hand, it cannot be said that cold and high places are free from rheumatic cases.

Exposure.—The history of exposure to wet is so frequently obtained in the history of rheumatic cases that it is difficult to deny its influence as a definite factor in the production of a rheumatic attack. On this account especial care must be taken that children who get wet should be overhauled and made to change clothing which is damp. Too often the child's statement that its things are not wet is accepted without any confirmation by actual examination. In connection with this subject, it is important to insist on greater care being taken of children who often go short distances out of London to get to their playing-grounds, and who are then allowed to sit in trains or other conveyances in clothing wet from rain or from perspiration. If a child can keep on the move in wet things, little or no harm will result; but if he sits about in wet things, a child predisposed to rheumatism is being exposed to a grave and unnecessary risk. It is in this way that hot weather may be quite as risky to a rheumatic child as wet weather. A child should not be debarred participation in strenuous games just because it is rheumatic, provided there is no evidence that the heart is in any way affected. In connection with this matter, it is well to see that after a bath a child is thoroughly dried, especially behind the knees and in the axillæ.

Diet.—Rheumatic children should not indulge in much sweetstuff. While it is unnecessary to forbid jam and sugar, it is at the same time advantageous to the child not to accustom it to the continuous use of these substances. Red meat, which is certainly bad during active rheumatism, is not to be forbidden to the rheumatic child, and the popular belief that meat is bad for the rheumatic is certainly false.

Of equal importance with the attention to the above points is the recognition and prompt treatment of apparently trifling manifestations, more particularly slight sore throats, stiffness about the limbs, stiff necks, and various myalgias. All of these should be treated by the administration of some preparation of salicylic acid or its salts, for while it is impossible to prove that the administration of any drug has been the cause of the non-development of further symptoms, yet we must acknowledge that the untreated cases are those in which we find that sequelæ most commonly occur. When we remember that many cases of acute rheumatism are ushered in by a sore throat, it seems more rational to treat this at the outset than to wait for developments, consoling ourselves with the statement that many of these sore throats get well without treatment.

It is the writer's belief that much rheumatic trouble might be avoided by the early administration of salicylates, and for this purpose solid in doses of from 3 to 10 grains three times a day is the most useful, least disagreeable, and least irritating, of the salicylic compounds.

The Treatment of an Acute Rheumatic Attack.—The recumbent position in bed is essential. The child should be put into flannel night-clothes and placed between blankets. The mattress should be firm and rest on springs. If there is much joint pain, a cradle should support the weight of the bed-clothes. A liberal supply of change night-clothes should be provided, so that the child should not be exposed to any chill should profuse perspiration occur, though this is exceptional in children. The clothes should open in front, so as to allow of ready access to the precordia for the purpose of examination; and if it is desired to make local applications to the joints, these should be applied to the parts, not by roller bandages, as is too often done, but by many-tailed bandages or by pieces of muslin tied or pinned so that they can be undone without disturbing the joint at all.

The diet should be liquid, and consist of milk diluted with soda-water, barley-water, or lime-water, or with some of the alkaline waters, such as Vichy, Ober-saïen, or Apollinaris. The use of sodium citrate added in the proportion of 2 grains to the ounce of milk will be found useful. Ewart recommends the addition of 20 to 30 grains of salt to each pint of milk. Veal broth, mutton broth, and beef-tee may be allowed in conjunction with the milk, but must not be allowed to replace the milk as the chief food. Lemon or orange jellies will often prove welcome additions to the diet, and additional variation may be obtained by the use of some of the proprietary foods.

Thirst is not as marked a symptom in children as in adults, probably because sweating is much less marked. It is best treated by allowing the patient to have barley-water in which slices of fresh lemon are floating. Imperial drink, made by adding a drachm of potassium bitartrate to a pint of barley-water to which some sugar rubbed on a fresh lemon has been added, will be agreeable to most children. Another grateful drink can be prepared by tearing a fresh lemon up and boiling it in a pint of water, straining it off, and adding 20 grains of bicarbonate of potash.

When the joint pains, joint effusion, and pyrexia, have subsided, we can gradually diminish the fluid and increase the solid articles of diet; fish, chicken, and finally meat being allowed; but with each change of diet a watch must be kept on the temperature, and a rise should mean the prohibition of solid food for at least three days. One of the commonest causes of a rise in temperature during convalescence from an attack of acute rheumatism is a premature allowance of solid food.

Apéritive.—Calomel $\frac{1}{2}$ grain given every half-hour for four doses, will be found to be the best apéritive in a rheumatic attack.

Drugs.—No one who has seen the striking effects produced by the administration of the salicylates in acute rheumatism will hesitate to place them fast and foremost in the list of efficacious drugs. So striking are the effects on some of the most obtrusive phenomena of a rheumatic attack that salicylates have been considered as specific, and every rheumatic manifestation has been treated with salicylates in the firm belief that it must do good. While some rheumatic affections are eminently amenable to salicylate treatment, there are a considerable number which are not appreciably benefited by it. The manifestations responsive to salicylate treatment are—joint pains, joint effusions, sore throat, and the pyrexia in association with the foregoing symptoms; but skin manifestations, including erythema nodosum, rheumatic nodules, peri- and endocarditis, anæmia and the pyrexia which accompanies it, and chorea, in spite of all that has been claimed for the salicylates as effective agents in their treatment, are not, in the writer's opinion,

controlled by them. It is an interesting point, however, to note that some of the rheumatic manifestations which do not respond to the internal administration of salicylates yet are definitely relieved by the local application of preparations containing salicylates. The term "salicylates" has been used above to indicate the various preparations, salts, and derivatives of salicylic acid. The preparations in most common use are the salicylate of soda, salicin (natural and synthetic), aspirin (acetyl salicylic acid), salol (salicylate of phenyl). Of these, the salicylate of soda is most frequently used, but some prefer the use of natural salicin. It must be remembered that salicin only yields 43 per cent. of its weight of salicylic acid, so that the dose is nearly double that of salicylate of soda. The use of sodium salicylate is attended by risks of gastric irritation and by the production of a condition of acidosis. This latter complication can be prevented by the simultaneous administration of bicarbonate of soda in doses double that of the salicylate given. Aspirin will sometimes give relief where salicylate of soda has failed to give relief. It also causes the skin to act more freely, and has a distinctly hypnotic effect. Salol is much less irritating to the gastric mucous membrane, but the action of the phenyl on the kidneys must be borne in mind. It is insoluble in water, and only splits up and is absorbed in presence of an alkali, so that its action is not rapid. Salicin is more soluble than salicylate of soda; it is bitter, and does not readily cause vomiting. It is consequently useful in children who show an intolerance of salicylate of soda. It must be borne in mind that, while salicylate of soda is advantageously prescribed with an alkali, aspirin is incompatible with an alkali. Sulphate of quinine is also incompatible with salicylate of soda, as are ammonia, carbonate of ammonia, and aromatic spirits of ammonia. The oil of wintergreen, which consists of 99 per cent. of salicylate of methyl, can be used internally in the form of capsules, but is more commonly employed as an external application. As most of the forms in which the salicylates are administered internally are irritating to the gastric mucous membrane, it is found best to administer them directly after food. As a general rule it will be found that children bear the salicylates well, though in some cases there is intolerance. In such cases the various compounds of salicylic acid which have just been referred to must be tried. When large doses are being given, it will be found that intolerance can be avoided by the occasional omission of a dose. Lees has pointed out the necessity for keeping the bowels opened daily while large doses of salicylates are being given, and also the advantage of the simultaneous administration of an alkali, such as bicarbonate of soda, as a means by which the production of acidosis can be prevented.

Dosage.—When we have to deal with an uncomplicated attack of acute rheumatism, there is little difference of opinion as to the dosage to be employed; but when we come to the treatment of complications, or rather of manifestations of rheumatism, apart from the temperature, joint pains and effusions, and the more throat, there is a very wide difference in the practice of experienced physicians. Lees has advocated enthusiastically the employment of large doses of salicylate of soda, giving to children as much as 600 grains of salicylate of soda in the twenty-four hours; others have failed to find such doses really instrumental in preventing, modifying, or still less curing, many of the rheumatic manifestations.

Joint Pains.—These are eminently susceptible to salicylates. At the outset 10 grains every two hours should be given for three or four doses, and then the same dose every four hours till the pains are relieved, and there should be no pain left after twenty-four or at most thirty-six hours' treatment in bed. Should the

poor patient, the frequency of the dose should be increased to every two or three hours, the diet should be diluted milk only; or it should be recognized that we are dealing with a case in which salicylate of soda is not going to do good, and try salicin or, preferably, aspirin. Cases which do not respond to salicylates are more commonly met with in adults than in children.

When the joint pain is very severe at the outset, it is well to wrap wool around the painful joints. An ointment consisting of 1 drachm of oil of wintergreen to an ounce of lanolin, if spread on a piece of linen and placed over the joint, which is then wrapped in wool, will afford rapid relief to the pain. When there is much restlessness associated with the pain, a few drops of tincture of opium may be sprinkled on the affected joints before wrapping them in wool.

Joint Effusion.—These as a rule readily subside under salicylic treatment. The application of the oil of wintergreen ointment aids absorption. Pain disappears before the fluid effusion in joints. If the fluid persists, the application of a blister over the joint will often hasten absorption, but in some cases it may be necessary to aspirate the fluid from the joint. When this is necessary it will be found that the joint has been the subject of undue movement at the onset of the rheumatic attack.

See Throat.—This condition is well treated by the salicylates, which are more efficacious than chlorate of potash or formalin. Should the tonsils be enlarged and exhibit follicular exudation, nothing will cause more rapid improvement than the local application of a saturated alcoholic solution of salol (salol, $\frac{1}{2}$ drachm; glycerine, 2 drachms; rectified spirits of wine, to 1 ounce). This should be painted with a camel-hair brush every three or four hours on to the tonsils and soft palate. The first application is often painful, but the subsequent ones much less so, so that children rarely object after one or two applications have been made.

Pyrexia.—The pyrexia of acute rheumatism is readily controlled by the salicylates. The temperature in the usual run of cases subsides gradually under rest and treatment, so that after the third day it should be not higher than 99° F. If the temperature has not come down by the third day of treatment with salicylates, it is because there is some complication present, such as pericarditis or endocarditis. Occasionally patients resistant to salicylates respond at once to aspirin. While the treatment of the pyrexia at the outset of an attack of rheumatism is as a rule quite satisfactory, the same cannot be said of the continued moderate pyrexia referred to as existing in the rheumatic state. In the majority of such cases salicylates have little or no influence upon the fever. The dose of salicylate may be increased without producing any alteration in the chart, though best again a change from sodium salicylate to aspirin or, when this fails to salol may be followed by the disappearance of the pyrexia.

Hyperpyrexia is very rare in children, and requires to be treated by cold baths and stimulants. To administer salicylates in such cases is to waste valuable time and lose the patient.

Skin Manifestations of Rheumatism.—With the exception of purpura and erythema nodosum, the other skin manifestations of rheumatism neither call for anti-rheumatic treatment nor do they respond to it. Purpura has in the writer's experience been quite uninfluenced by the salicylates, sometimes, indeed, developing in patients who are taking large doses. Rest in bed is essential, and its influence can be seen in the recurrence of purpura about the ankles when convalescent patients get up for the first time. Chloride of calcium, calcium lactate, ergot, turpentine, capsicum extract, and the astringent preparations of iron, have all been used in

the treatment of a condition which tends to get well spontaneously. Erythema nodosum is said to be favourably influenced by the salicylates, but probably on insufficient evidence; it certainly sometimes develops in children taking large doses of these drugs. The local treatment of the erythematous patches is much more important and successful. When the pain is severe, the patches may be painted with glycerine of belladonna and have warm fomentations applied; but even more successful is the application of an ointment containing 1 drachm of oil of gaultheria to 1 ounce of lanolin on a piece of lint. When there is much exudation and pain, the local application of lead lotion or calamine lotion will often give marked relief. Here, as in purpura, rest is an important factor in successful treatment. It might be expected that the exudative erythematosa would respond to treatment with vacuum salta, but there is little if any direct evidence that they do. A knowledge of the natural history of untreated conditions often damps therapeutic optimism.

Nodules.—As such the rheumatic nodules do not call for treatment, but the discovery of any form of treatment which will cause the disappearance of the nodules will rob rheumatism of most of its terrors. Unfortunately, in spite of the claims made for the efficacy of the treatment by doses, often heroic, of salicylates, the writer is still of opinion that the nodules are uninfluenced by these drugs. Nodules not infrequently appear, or, when present, increase in size or in number, both when patients are taking salicylates and when they are not, and under similar conditions they disappear. Reference has been made to the anatomical identity of the nodules with the valve changes in endocarditis, and clinical experience has confirmed the view held by the writer that both conditions are uninfluenced by the administration of the salicylates. It has been asserted that the incidence of endocarditis of rheumatic origin has actually increased since the introduction of the salicylate treatment. This is in all probability due to the lack of care taken in preventing the patient from getting about too much and too soon after an attack of acute or subacute rheumatism. The writer has tried, in addition to the internal administration of the salicylates, the local application of salicylate of methyl and of menthol, but the only case in which the nodules diminished directly as the result of treatment was one in which the application produced an inflammatory dermatitis of the arm, necessitating treatment with lead lotion, after which the nodules on the treated arm underwent a rapid diminution in size. The employment of Bier's method of inducing passive hyperemia in the part affected produced no diminution in the size of the nodules.

For the treatment of the rheumatic affections of the heart, pleura, and pericardium, the reader is referred to the chapters on Diseases of the Heart, Pericarditis, and Pleurisy. The writer would, however, like to express his opinion that the salicylates are impotent in the treatment of these conditions, though in the case of pericarditis and of pleurisy the local application of oil of gaultheria (1 drachm to the ounce of lanolin) is nearly always followed by marked relief of the pain. It is largely due to the presence of salicylate of methyl that antiphlogistic proves so useful an application.

Jacura, as has been pointed out, is a frequent sequel as well as accompaniment of rheumatism, and for its treatment a combination of iron and arsenic is valuable. These drugs should not be given until the temperature has been down at the normal level for a fortnight, and, when given, it is well not to omit the salicylates, but rather to substitute for them 5 to 10 grains of salol every night and

morning. When there is much debility, and especially if the heart is well dilated, it is well to combine the iron and arsenic with strychnine.

Convalescence is best established at dry and high elevations, and the seaside is not always beneficial. In the treatment of the stiff neck and of the stiffness occasionally left about the joints in children after an acute attack of rheumatism, benefit will be found to follow the use of a liniment containing 1 drachm of menthol to 1 ounce of chloroform liniment, or to the use of the gaultheria ointment. Salol or aspirin will often be found more useful here than salicylate of soda.

Two methods of treatment of the rheumatic condition have yet to be mentioned: the alkaline method advocated by Fuller. This consists in the administration of citrate or bicarbonate of potash, as advocated by Sir Alfred Garrod. The relief obtained was distinctly less than that afforded by the salicylates, though there seems to have been rather less liability to endo- and pericarditis than with salicylates. In the alkaline method of treatment the alkalies are pushed till the urine is steadily alkaline in reaction at the time it is passed.

The other method is serum treatment. This is at present in an experimental stage. In the few cases which the writer has seen treated with rheumecoccic serum, he has failed to detect the least evidence of benefit. Meiner has prepared a serum, which has been employed in the treatment of acute rheumatism. The serum is derived from horses injected with living cultures of organisms obtained from the joints or pericardial effusion of patients suffering from acute rheumatism. The results of this treatment are not very encouraging, for, judging from the temperature charts, there is a longer period of pyrexia, there is the disadvantage and discomfort of subcutaneous administration, the relief of pain is less rapid, and a rash commonly appears after some days. Against these drawbacks it is claimed that there is no disturbance of digestion and that the sweating, which, however, we have seen, is an inconspicuous symptom in children, is much less. The reaction which takes place after injection is seen only in the joints which have been the seat of pain and effusion, and is not general. It is said that the treatment by Meiner's serum is more satisfactory in the case of chronic rheumatism than in acute cases.

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CHOREA.

SYNONYMS.—Sydenham's chorea; St. Vitus's dance; Chorea minor; Chorea angulorum.

Chorea was described in 1686 by Sydenham, who first clearly differentiated it from the hysterical form of oscillatory spasm.

DEFINITION.—Chorea is a rheumatic affection of the central nervous system, characterized by the temporary withdrawal of restraint (the highest function of the central nervous system), and manifested by inco-ordinate movements, loss of power, and by psychical and emotional instability, but tending to complete recovery.

Chorea has been described as an "insanity of the muscles," but this is far too limited a description, for it leaves out of account the muscular weakness, and also the psychical manifestations.

Sturges has pointed out that the movements in chorea are merely an exaggeration of the natural fidgetiness of a child, and, indeed, no sharp line can be drawn between slight cases of choreic movement and marked fidgetiness.

ÆTIOLOGY.—The question of the rheumatic nature of chorea has, since the early part of the nineteenth century, engaged the attention of the profession. The experience of most of those who have worked at London hospitals has been strongly in favour of regarding chorea as a manifestation of rheumatism, a view held by the writer. The statistics gathered from the written reports on cases admitted into the wards of a hospital naturally vary so much with the experience of the recorder, and the conception, often very inadequate, of what is rheumatic possessed by the laity, that we can hardly expect to find anything like uniformity in the results of such investigations. In spite of the obvious limitations which must attend the inquiries into the family and personal histories of children admitted to hospital, there is a general agreement that an appreciable proportion of the cases of chorea admitted as such give a definite history either of personal rheumatism or a history of rheumatism in the parents, brothers, or sisters. The Collective Investigation Committee of the British Medical Association in 1887 found that a definite history of rheumatic fever was present in 26 per cent. of the cases of chorea. Morley Fletcher found that in 327 in-patients with chorea 25·2 per cent. had a personal history of rheumatic fever. Frazer, from an analysis of 300 cases of chorea occurring at the Paddington Green Children's Hospital between 1890 and 1910, found that in 50 per cent. the chorea had been preceded by definite articular rheumatism. Batten found that in 115 cases of chorea, in which no rheumatic history was obtained, at least 25 per cent. of these cases developed rheumatism within six years of the attack of chorea. The frequent association of chorea with evidences of endo- and pericarditis compels us either to accept the chorea and the endo- and pericarditis as being all three expressions of rheumatism, or to attribute the chorea to the endo- or pericarditis, or these latter conditions to the chorea. No one who has worked in the wards of a children's hospital can have failed to have met with cases of chorea which have developed during the course of an attack of articular rheumatism, or of peri- or endocarditis, and often associated with the appearance of rheumatic nodules. It may be said that, apart from associations with definite

rheumatic manifestations, an attack of chorea is unknown to develop during a child's stay in hospital. Chorea is the only transient nervous disease known to be associated with endocarditis. When it is remembered that rheumatism may, at a given time, have only a single manifestation, we need not be surprised if we fail to get a history of rheumatism in every case of chorea. Chorea, in fact, may be the first evidence of rheumatism. In fatal cases of chorea a diplococcus which produced arthritis in rabbits was isolated by Westphal, Wassermann, and Malkoff; and Poynton and Paine have found the diplococcus rheumaticus both in the sinusses and in the blood in cases of chorea.

In opposition to the rheumatic view above maintained, it is urged that there are a certain number of cases of chorea in which there is no history nor any subsequent development of symptoms which can be designated rheumatic, and this, though it is by no means a fatal objection to the rheumatic view, is certainly true. It has been urged that, as chorea not infrequently develops as the result of a fright or from overstudy, there is a chorea independent of rheumatism. To look on rheumatism as the only factor to be considered in the production of chorea is to shut one's eyes to certain important factors in the etiology of the disease.

Sex.—The disease is certainly nearly three times as common in girls as in boys, and age also plays an important part in its occurrence. It is still held by most men that the nervous system of the female is less stable, even in youth, than that of the male. In investigating the influence of sex we may bear in mind that chorea is a disease which affects the sharp, bright, active-minded children; that in school the prospective choreic is to be sought for at the top end of the class and not among the dullards. Fright is often said to be the cause of chorea, and it certainly may be the spark that sets fire to the train already laid by rheumatism. A child, as the result of early chorea, is inattentive, is punished for this inattention, and an attack of chorea, expressed by inco-ordinate movements, is precipitated. The chorea is said to be due to the punishment, whereas the punishment is due to the chorea. The frequency with which fright acts in bringing out an attack of chorea is probably understated in hospital records, for often, when the child's confidence is gained, it will tell the story of some fright; black dogs, drunken men, and policemen being among the most frequently assigned causes for fright, even when the history, as obtained from the parent or friend, has been silent on this point.

Imitation has been asserted to be a cause of chorea. The writer has never seen a case which could be ascribed to this cause. Apart from personal experience in hospital, it is a striking fact that although many members of a family may at different times develop chorea, yet it is very unusual to find two cases of chorea existing at the same time in one family. If imitation were a cause of true chorea, it should occur fairly commonly in two members of a family simultaneously.

Age.—Chorea may occur under two years of age. At the Hospital for Sick Children the writer has seen two cases under three years of age. In boys the disease occurs at an earlier age than in girls, but in both attains its maximum incidence between six and nine years of age. It occurs much less frequently in boys than in girls over ten years of age.

PATHEOLOGY.—The earliest explanation offered of the phenomena of chorea was that of Scudamore, who attributed them to the debilitating effects of acute rheumatism. In 1839 Bright attributed chorea to a reflex stimulation from the inflamed pericardium. In 1863 Kirkes explained chorea as due to showers of minute

thrombi projected into the cerebral vessels and causing minute emboli. This view was also held by Hughlings Jackson. Money and Mott succeeded in producing in dogs, as the result of the introduction of minute particles of carbon, movements in the limbs, but these movements were not truly choreic, nor is canine chorea the same condition in dogs as chorea in man.

Recently Poynton and Gordon Holmes have shown that distinct changes are found in the bloodvessels of the brain and meninges. The changes in the nerve cells are secondary to the vascular changes, and are evidenced by various degrees of chromatolysis in the large pyramidal cells and in the Betz cells. Diplococci identical with those found in the blood, endocardium, pericardium, and in the joints, were also found by them both in the cerebral vessels and in the meninges, as well as in the cerebro-spinal fluid in cases of chorea.

The complete recovery which occurs in every case of chorea when not fatal makes the embolic view very improbable; and the difficulty of discovering a source of emboli in the absence of endocarditis, and the very different picture which results from a visible embolism in the brain, are further difficulties. On the other hand, we can conceive that a poison circulating in the blood may have some especial affinity for the cortical cells of the cerebrum and cerebellum, and may cause in them irregular discharges of nerve energy, and deprive movements of their co-ordinated character. The association of movement and weakness, or even absolute, though temporary, paralysis of the muscles of the limbs, is quite in keeping with the slight structural changes which have been described by Poynton and Gordon Holmes.

The cessation of movements during sleep, the existence of choreic movements in which one half of the body is markedly, and the other hardly appreciably affected, the influence of fright in evoking choreic movements, and the mental changes, all point to the cerebral and cerebellar cortex as being the seat of the changes.

Chorea may thus be regarded as the result of the action of the rheumatic toxin or toxins on the cortical cells of the cerebrum and cerebellum.

SYMPTOMATOLOGY.—Although the earliest symptoms to attract attention are the movements, yet it will be found that not infrequently these are preceded by moderately severe headache, although as a rule this is not spontaneously complained of. Irritability, quarrelsomeness, and peevishness are often noticed for some little time before any movements attract attention. At school the child is inattentive, is unable to concentrate its attention; the handwriting deteriorates, and these departures from a previously high standard of conduct and attainment bring in their train moral or physical punishments in the form of reproof, loss of place in class, extra tasks, or corporal punishment. Any or all of these may precipitate an attack of inco-ordinated movements.

Motor Phenomena.—These are varied both in extent and in degree. In some cases the facial muscles are most affected. The irregular contractions of groups of muscles cause the facial expression to indicate now fear, now amusement, now pain, now disgust. The movements are irregular and unlike habit spasms; they are not the recurrent performance of a single act such as a jerk of the head, a blinking of the eyelids, a twitch of the nose, but an irregular affection of different groups of muscles. There is no grinding of the teeth. There is frequently a sudden depression of the tongue in the floor of the mouth, which results in the production of the "choreic duck." The tongue is moved about in all directions, but in spite

of the irregular and uncontrolled action of the muscles of mastication, it is almost unknown that the tongue should be bitten. The eye muscles are as a rule but little affected. Sometimes the eyeballs are rotated, but squints are exceptional. The pupils are nearly always dilated, and Langmead has noted that they are often eccentric, and show a condition of slow alternate contraction and relaxation.

The neck is almost invariably bent, the head hanging forwards. There may be violent shragging movements of the shoulders, spreading to the upper limbs. This is especially well seen in what is known as "lightning chorea." The trunk muscles may be affected, the child's body being jerked here and there irregularly. The respiratory muscles are also affected, the respirations being often grouped in rhythm, and sighing is frequently noted. Hiccough is rare. In the upper limbs we may meet all varieties of movement, from a simple twitching of the fingers to violent contractions which necessitate restraint. The slight movements in the fingers may often be best appreciated by making the child place his hands on the upturned palms of the observer, who will thus be able to detect fine twitchings which might readily escape notice. When a choreic child with movements in the upper limbs walks, it will be noticed that he generally adducts the arm and forearm, which is strongly pronated, and keeps it applied to the side of the trunk. The hand is usually flexed at the wrist, and the hand, with fingers extended, waves about behind something like an oar when a boat is being "sculled" from the stern.

There is a very interesting and common position of the hand, which the writer first noted in 1889. This consists in the over-extension of the wrist and of the metacarpophalangeal joints, when the child attempts to hold the hands up straight above the head. The sign is of interest, because frequently, after nearly all choreic movements have ceased, the sign remains, and is most marked on the side on which the movements have been most marked. Sometimes, instead of the over-extension of the wrists and digits, there is much muscular weakness, and if the hands can be held up, the wrists drop into the flexed posture, and the child is unable to hold them up straight.

In the lower limbs the movements are as a rule less than in the upper limbs. In walking, one or other limb may be dragged along; the gait is very uncertain, and the bruised shins are ample evidence of unintentional encounters with hindrances to progression. When lying in bed, the movements of the lower limbs and trunk may throw the child out of bed. When walking, sometimes a leg will give way, with the result that the child falls to the ground.

Muscular weakness is always present in chorea. The sphincters are not generally affected, though in the severer cases there may be temporary incontinence of urine or of feces. The act of deglutition is difficult on account of the inco-ordinated movements of the lips, cheeks, tongue, and other muscles of mastication, though when once food can be got to the back of the pharynx, the rest of the act of deglutition takes place co-ordinately. Vomiting is rare in uncomplicated chorea, except as the result of drugs or of too rapid feeding. The heart may be affected in several different ways in chorea. There may be peri-, myo-, or endocarditis, the result of the rheumatic poison, and Lees has drawn attention to the risk of acute dilatation of the heart in this condition. Apart from these rheumatic affections, there is frequently irregularity of the action of the heart, and with, or without this, a systolic murmur is heard at, and internal to, the apex of the heart, and also up the left edge of the sternum, as high as the pulmonary cartilage. This murmur is probably due to a dilatation of the aorta arteriosa.

In the severer cases of chorea, sordes may be met with on the lips, even in the absence of any heart or lung complication. Choreic children sometimes burst out into a sudden, piercing cry, which is quite characteristic of the disease. The speech is often affected. Most remarkably there is hesitation and deliberation in the speech, but true stammering is unknown. In the severer cases there may be complete aphasia, a condition which has been known to persist for more than six months. The mental condition of the choreic child is practically always abnormal. The children are emotional, and liable to outbursts of crying, or less frequently of laughter. In severe attacks a child may become delirious and imagine that it sees all sorts of spectres—a point referred to under the head of treatment. The choreic child does not as a rule in hospital growse or grizzle after the first few days, nor constantly want to be taken home. Sleep is often disturbed, and many of the children complain of having bad dreams.

Reflexes.—The chief alterations in the reflexes in chorea are seen in the variations which may occur in the knee-jerks. These may be natural, but more commonly are found to be unduly active. The knee-jerks may be reduplicated or even triplicated, one tap on the patella causing double or treble jerks. Another variation which has been demonstrated for over twenty-five years at Great Ormond Street is the "hang-up" knee-jerk. In this condition a tap on the ligamentum patellæ is followed by a contraction of the quadriceps muscle which lasts for a longer period than usual, so that the leg may be held extended for as long as three seconds, and then suddenly drops. Yet another modification may be found is that the knee-jerk may be very difficult to elicit; it may only be evoked as reinforcement; finally, the knee-jerk may not be obtainable at all. All these modifications may be met with at different times in the same case, the exaggerated and hang-up jerks being met with in the earlier stages, when the movements are active, the loss of jerks being usually accompanied by aphasic troubles and the paralytic manifestations of chorea. As convalescence progresses, the knee-jerks gradually return to their healthy condition. The superficial reflexes are not altered in chorea, though they may be incapable of demonstration on account of the choreic movements. Ankle clonus is not met with even when the knee-jerks are extremely brisk.

Wasting.—A severe attack of chorea is almost always accompanied by very marked wasting, which may be shown in the forearm and hand muscles, as described under Rheumatism; but apart from this more local, there is often extreme general, muscular wasting, the child being reduced almost to a skeleton. It is in these cases, which are generally associated with the paralytic form of chorea, with aphasia and with loss of knee-jerks, that the greatest care has to be taken to prevent the formation of bedsores. The wasting is associated with very marked loss of power, so that if, when the child is lying in bed, the arm is raised and held over the face, and then let drop, it will fall on to the face—an occurrence which does not take place in functional paralysis of the limb. The power of the child to prevent the limb falling on the face may be used as a useful measure of the child's progress. Sensory phenomena are, as a rule, undetected in chorea.

Anæmia accompanies chorea, as it does many other manifestations of rheumatism.

Owing to the involuntary movements which are met with in the early stage of chorea, little domestic accidents, such as the upsetting of the teacup, the dropping of spoons or forks, or of the jug, are apt to occur—accidents which, from the fact

consequences, materially accelerate the evolution of choreic movements. Choreic movements, though described as involuntary when not severe, can be largely controlled by the will.

Varieties of Chorea.—Cases of chorea may be divided into (1) those in which the movements are slight—mild chorea, for which the name of "latent chorea" has been proposed by Miller; (2) ordinary or moderately severe cases, in which the movements are well marked; (3) severe chorea, in which the movements are severe, and call for some mechanical provision for the prevention of injury; (4) the lightning form of chorea, in which the movements are characterized by their sudden and shock-like nature; (5) the paralytic variety of chorea, in which there is loss of power and of movement, often associated with aphasia; (6) there is a variety of chorea characterized by the maniacal condition of the patient.

It must be understood that there is no sharp line of demarcation between these varieties, and that one may pass into the other. It is not uncommon to observe severe motor chorea give place to the paralytic variety, and mild cases may become severe or even maniacal.

When the movements are well marked on one side and hardly appreciable on the other, the name "hemichorea" is often used; but careful investigation will always show that true hemichorea—a condition in which one side of the body entirely escapes—does not exist. The movements in chorea rarely affect the two sides of the body equally, and this difference is one which during an attack remains constant, unless the onset of paralytic conditions leaves the side which originally showed more movement now the quieter of the two.

The temperature of a patient in an attack of chorea is not raised in the absence of complications, such as sore throat, endo- or, more commonly, pericarditis, pleurisy, or in rare cases hyperpyrexia. The pyrexial conditions in chorea are the expression of rheumatic complications or are due to infection of abrasions or bedsores with pyogenic organisms.

Complications.—The complications of chorea are the other expressions of rheumatic poisoning. Tonsillitis is one of the commonest. Arthritis is not common, and is rarely seen in severe motor chorea. Pericarditis, myocarditis, and endocarditis are fairly common. Rheumatic nodules are not at all infrequently met with. Paps are uncommon, though the rheumatic erythematata are not infrequent. Erythema nodosum is rarely met with in the course of a choreic attack. Dilatation of the heart is very common in the early part of a choreic attack, and may give rise to murmurs, which are not infrequently misinterpreted as being caused by organic changes in the valves. Hyperpyrexia, with or without pericarditis, may very rarely occur in the course of a choreic attack. Pyæmia, resulting from infection of some injury to the skin, has been recorded. Delirium or acute mania may arise as complications of chorea. In rare cases an attack of chorea may be followed by insanity.

Differential Diagnosis.—The diagnosis of chorea rarely presents any difficulty. As Sturges pointed out, there is no sharp line between the fidgetiness of the nervous child and the movements of slight chorea. Children suffering from various forms of *habitus spasmus* are frequently brought to a medical man for treatment as cases of St. Vitus's dance, but the recurrent character of a habit spasm differs from the typical irregular muscular movements which characterize a choreic attack. The history of other rheumatic manifestations may enable the physician to detect, in the fidgetiness of

a child, the early motor phenomena of a choreic attack. In addition to the collateral evidences of rheumatism, attention must be paid to those changes in disposition and temperament which are so truly part of the early manifestations of chorea.

In the paralytic form of chorea the diagnosis may be difficult, and the difficulty is increased if the weakness on one side is much more than on the other. Such cases, when they arise in children who have signs of endocarditis, may simulate a *hemiplegia* resulting from cerebral embolism. In the latter case the onset is sudden, the paralyzed areas do not correspond to those in which the movements have been most marked, as is the case in the choreic paralysis, and the deep reflexes in paralytic chorea are diminished or lost, while in the embolic cases they are exaggerated, and there may be an extensor phenomenon present in the toes. The severe paralytic form has been mistaken for a toxic paralysis, such as is met with after diphtheria; but here the eye muscles, which escape in chorea, are almost always affected, and the respiratory muscles, which escape in chorea, are usually profoundly affected in post-diphtheritic paralysis.

The movements of chorea are sometimes roughly simulated by those of *hysteria*, but the latter are without that inco-ordinate and unpurposeful character seen in chorea, and are increased rather than diminished by the conscious exercise of the will. The absence of sensory alterations in chorea may assist the diagnosis.

PROGNOSIS.—The prognosis in a case of chorea is favorable as regards eventual recovery. The dangers are those of rheumatism (*q.v.*). Death is a rare event in chorea, except from pericarditis, heart failure in cases of endocarditis, or very rarely from exhaustion after very violent movements. Both hyperpyrexia and pyrexia have been recorded as rare causes of death. In the absence of involvement of the heart a fairly confident prospect of recovery may be held out in every case of chorea in a child, however led the movements, and even when the paralytic symptoms are marked. The patient may take a long time to recover, but recovery is the rule.

The duration of an attack is variable. Few are of less than six weeks' duration; some may last for twelve months, or even longer. Moderately severe movements in the less intelligent children, either boys or girls, are the most apt to be protracted on account of their liability to recurrent relapses, so that a child may be in a state of mild chorea for as long as three years. The disease is one which is prone to recur, many children having two, three, or as many as six or seven attacks of chorea. The duration of an attack of chorea is not materially shortened by the administration of drugs, though their use may be often of the greatest benefit to the patient.

Owing to the rheumatic nature of the disease, repeated attacks of chorea bring with them increased risks of inflammatory conditions of the heart and its coverings. The severity of an attack does not depend on its occurrence as a first or as a recurrent attack.

Speaking generally, we may say that, however appalling to the friends the phenomena of chorea may be, they rarely cause serious anxiety to the physician in charge of the case.

TREATMENT.—The treatment of a case of chorea may be considered under three heads: (1) General; (2) Symptomatic; (3) Specific.

1. Under the head of *General* treatment we must first attend to the surroundings of the patient. If the child is at school, it should be at once removed, and all

house stopped. Taken at its onset, the judicious removal of a child from home to the rest and quiet of the country, apart from other children, with a sensible relative or nurse who can exercise a wise restraint with kindness and tact, will prevent many cases of chorea from developing; but it must be remembered that to send such patients among strangers or to convalescent homes is only to precipitate an attack. If treatment is to be carried out at home, it is essential that the other children should be kept away or only allowed to be with the patient for a short time each day. Choreic children should never be left alone. Parents and relatives are rarely the best nurses for choreic patients, and nurses in attendance require to be careful, patient, and experienced, and congenial to the patient.

When the movements are but slight, there is no necessity to confine the child to bed, though a certain amount of rest in the recumbent position must be insisted on. These periods of rest must be made as little irksome to the child as possible, though exciting stories, games of skill, or competition, must be avoided. The practice of acquired arts, such as reading, sewing, knitting, painting, or drawing, may be allowed, but no new acquisitions should be attempted. Gentle walking may be permitted, provided the surroundings are not such as to subject the child to risk of fright or excitement. If there is any difficulty in the gait, the child is best kept to the house.

Rest, sleep, and food are three essentials for the successful treatment of a case of chorea. It is well to remember that to the choreic child darkness may cause a great terror, and therefore it is advisable in all cases to have a light burning all night in the room. This precaution is apt to be forgotten in the case of children who are being treated in a ward by isolation from other cases by means of a screen.

Sleep in the early stages, when the movements are but little marked, can be largely aided by the use of a warm bath at night-time. Should there be much sleeplessness, it is well to give a small dose (5 grains) of triazol at bedtime.

Food, in the absence of evidences of other active rheumatic processes, should be liberal, easily digested, and not exclusively liquid.

For the successful treatment of early cases of chorea it is essential that the mental alterations should be borne in mind, so that peculiarities of temper, inattention, and similar aberrations, are treated as part of a disease, and not corrected as in the case of a healthy child. The bowels should be moved once daily with *massena* or *senna*. Should the movements become more marked, it will be necessary to keep the child in bed, and treat other symptoms as they arise.

2. Symptomatic Treatment.—The patient must be put to bed. The bed should be placed with one side against the wall to diminish the liability of the child to fall out; or if the child is not too big it may be put into a cot. Should the movements be at all violent, it will be necessary to pad the sides of the cot. In cases of very severe movement it may be advisable to make up a bed on the floor, but this adds very materially to the difficulties of nursing, and exposes the child to draughts. In some cases where the child is wasting and the skin is tender, a water-cushion may be used, but it will be found that the discomfort of the patient is often increased by the mobility of the bed. A certain amount of mechanical restraint to the movements is welcome to the patient, so that it is often a good practice to wrap a blanket firmly around the trunk, including the limbs, and to pin the blanket firmly round the child. This must be done without the suggestion of punishment. A child who has been wrestling with a chaos of blankets, sheets, and nightdress

often experiences a grateful sense of relief by this mode of restraint. Even the tight lacing in of the blankets will often serve to diminish the movements.

Should these measures prove unavailing to restrain the movements, it is best to place the child in a hot-pack. Blankets, well wrung out in hot water and allowed to cool sufficiently, are wrapped around the naked child, who is then enveloped in a mackintosh sheet, and this in turn wrapped around with another dry blanket. In such a pack the child may be left for several hours, a careful watch being kept in the pulse and colour. In most cases the hot-pack acts like a charm, but sometimes it is ill-borne. It is, however, the most effective agent, short of actual narcosis, in diminishing the movements of a violent chorea. Warm baths may be tried as a substitute for the hot-pack, but are less easy to administer and less useful in restraining movements. Cold applications, douches or icebags, applied to the spine, may succeed where the hot-pack has failed, but will be found to be of more use in the treatment of chronic cases of chorea or during convalescence. The cold-pack may be used when the chorea is complicated by severe pyrexia.

Internal Medication.—Whatever views may be held as to the efficacy of drugs in the treatment of chorea, few cases escape drug treatment; and there is no doubt but that, while drug treatment may not appreciably shorten the duration of an attack of chorea, a great deal may be effected by drugs in giving relief to the patient. Foremost among the internal remedies are the sedatives. These cause a diminution in the movements, and induce sleep, and so contribute to the restoration of the nervous system. In the writer's experience, trienal has proved the safest and most efficacious sedative. With its use the movements certainly diminish, sleep is secured, and the mental condition improves. There are practically no ill effects following its use. It is given in doses of 5 grains three times a day for twenty-four hours to ascertain that there is no idiosyncrasy on the part of the patient, and then in the same dose every six, four, or in severe cases every three hours. The occurrence of marked drowsiness is the signal to diminish the frequency. The drug can be given as a powder, or better suspended in mullage with peppermint, cinnamon, or similar flavouring agents. It is practically insoluble in water. Trienal does not cause any gastric irritation, nor has it any depressant action on the heart.

Chloroform has been strongly recommended by Wynter, who claims that its administration materially shortens the course of the attack. It has occasionally succeeded where trienal has failed, but it is neither so safe nor so useful a drug as trienal. It certainly causes more drowsiness, and may give rise to a puffly appearance about the face, and occasionally to an erythematous rash. Neither veronal nor bromural have proved as efficacious as trienal. Chloral hydrate and the bromides of potash, soda, ammonium, or strontium, are recommended by some; and although chloral is well borne by children, yet the not infrequent coexistence of endocarditis with chorea makes chloral undesirable for prolonged use.

Should the movements be very severe and otherwise uncontrollable by the above-mentioned drugs in combination with external applications, it may be necessary to resort to continuous inhalation of chloroform.

When there is marked excitement, a hypodermic injection of morphia or of hyosine hydrobromide ($\frac{1}{10}$ grain) may be tried, should the hot or cold pack have failed to give relief.

3. *Specific Treatment.*—Apart from the administration of sedatives, certain other drugs have been claimed to have a specific action in chorea. These, again,

are divided into those which may be spoken of as anti-rheumatic and those which act as nerve tonics.

The recognition of the rheumatic nature of chorea has led some observers to look to the salicylates as the antidotes to all rheumatic affections. Less has advocated the free administration of salicylates in chorea, but in the writer's experience the salicylates have proved inefficacious. He has repeatedly observed chorea develop in patients already well under the influence of salicylates, and has repeatedly demonstrated the benefits of sedatives where the salicylates have failed to give relief. Aspirin sometimes is useful in affording sleep and relieving headache when the salicylate of soda has failed, but its influence on the movements is scarcely less than is that of the sedatives.

Of the second group of drugs—the nerve tonics—arsenic is the one most frequently employed. It may be administered in gradually increasing doses till 10 to 15 minims of the liquor arsenicalis are being taken three times a day, after which the dose is gradually decreased; or it may be given in moderate doses (3 to 5 minims) three times a day; or, lastly, large doses of arsenic may be given in order to bring patients rapidly under its action. In cases with much movement arsenic is of very uncertain utility, though in increasing and then diminishing doses it enables a toleration to be established, and serves to pass the time; but in large doses it is very liable to cause great gastric irritation and to produce peripheral neuritis, which may quiet the patient's movements in an undesirable manner. In the treatment of chronic cases of chorea and during convalescence arsenic is a most valuable drug.

Estace Smith speaks warmly of the beneficial effects of a combination of ergot andstrychnia, but neither this combination, nor the use of ergot alone have satisfied the writer. In the treatment of cases of chorea which are hanging fire there is sometimes distinct benefit from the administration of a combination of antipyrin in 3-grain doses with 2 to 3 minims of liquor arsenicalis.

For the headache which sometimes accompanies chorea, aspirin or a combination of phenacetin with citrate of caffeine will be found most useful.

In the paralytic forms of chorea, as might be expected, the administration of sedatives is not indicated unless there is much mental upset associated with sleeplessness, when the administration of small doses (5 grains) of trional will be found useful. In paralytic chorea there is the greatest need for careful feeding with concentrated forms of food, the avoidance of long intervals between the meals, and the exercise of a great amount of patience on the part of the nurse. The compound syrup of the hypophosphites, or the compound syrup of the phosphate of iron, or Estace's Syrup, or a combination of all three, will be found of most use in the paralytic cases, and along with these, should there be much wasting, cod-liver-oil with or without maltine may advantageously be given. Massage to the limbs, which is invaluable in the period of convalescence, is not to be entered in the earlier stages of paralytic chorea, nor should douches be given at this stage.

When movements have become very little marked, or when they reach a stage in which they are neither getting better nor worse, it is best to stop the administration of the sedatives, and to give tonics containing iron; but it is in this stage that a course of warm baths followed by a cold or tepid douche will be found most useful. Massage to the limbs may supplement this form of treatment.

Movements designed to re-educate the co-ordination of movements are recommended by some authorities, but the writer is of opinion that the co-ordination

should be regarded as an indication of the need of rest rather than of exercise; and as the natural tendency of the disease is to recovery, it is more reasonable to allow altered nerve cells to recover their physical integrity by rest rather than to call on them to exercise some of their latest, and therefore least stable, acquisitions before they are fit for it. The improvement which takes place as the result of re-education of co-ordination of movements in organic diseases of the central nervous system, such as tabes, is not to be expected in the case of excretion of nerve cells such as is seen in chorea. Change of air, having regard to the choice of a suitable place from a rheumatic point of view, exercise, and a liberal diet, will complete the restoration to health.

During convalescence, when the pulse in the absence of endo- or pericarditis is rapid or feeble or irregular, the administration of alcohol in the form of port wine for a limited period—that is to say, till these symptoms have disappeared—will often be found to be of great value, though it must clearly be understood that there is no occasion for its administration as a routine line of treatment.

The complications of chorea, which are of a rheumatic nature, are to be treated without especial regard to the chorea. If pericarditis arises, the application of an ointment of oil of gaultheria over the præcordia, the administration of trional if the movements are marked, will, with occasional doses of Dover's powder, usually suffice to relieve the child and control the disease. If sore throat or articular pains complicate a case of chorea, they are best treated by the administration of salol in 5 to 10 grain doses three or four times a day.

Bedsore are best prevented by early care of the back, by bandaging with alcohol, by the use of starch-powder or of a combination of oxide of zinc, starch, and castor-oil. If the skin is broken, Friar's balsam may be used; but if there is an indication that the wounds are unhealthy, an application of iodoform to the wound will often bring about healing. The judicious use of ring pads or water-pillows in such cases is assumed, and it is advisable to fix a ring pad or circular air-matress to the margin of the bolser.

Seeing that feeding is an essential factor in recovery, and recognizing that both in severe motor and in paralytic chorea the child is practically unable to feed itself, care must be taken to insure that the child gets sufficient food. In order to secure this, solid food requires to be finely minced up, covered with good gravy, and administered patiently. Twenty minutes to half an hour is not too long to allow for each feed. Should the movements be too great, or should the child merely retain the food in the mouth or be distressed by the feeding, nasal feeding should be resorted to. It will be found that it rarely requires to be continued for any length of time.

In the absence of any cardiac complication, the question of the time for which a case of chorea should be kept in bed requires consideration. Should the movements be either so slight that the hands can be held fairly steadily above the patient's head, or should they have reached a stage in which they are neither getting better nor worse, the child may be up for a couple of days on a couch, and gradually allowed to walk, first with and then without assistance. The child should be able to feed himself before being allowed to get up. If after a couple of days the movements are increased, he should be sent back to bed. Another point to be borne in mind is the condition of the knee-jerks. As long as these are absent it is not advisable to get the child up out of bed. Needless to say, the existence of dilatation of the heart is an imperative indication for rest in bed.

It will be gathered from what has been said that the treatment of a case of chorea involves attention to many details, the exercise of much skill and patience in wiring, the judicious administration of drugs, and that the element of time is not without its effects in procuring restoration to health.

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CHAPTER XIX

INFECTIOUS DISEASES

E. W. GOODALL.

SMALLPOX.
VACCINE.
SCARLET FEVER.
CHICKENPOX.
DIPHTHERIA.

MEASLES.
RUBEOLA.
"FOURTH DISEASE."
GLANDULAR FEVER.
MUMPS.

SMALLPOX.

SYNONYM.—Variola.

INTRODUCTION.—There can be no doubt that smallpox attracted the attention of mankind in times very remote. It is, indeed, claimed that it was recognized by the Chinese so far back as 1122 B.C., and tradition gives it a considerable antiquity amongst the inhabitants of India. But no unequivocal account of the disease has survived that was written before the Christian era was several centuries old. The first complete and certain description is that of Rhazes of Bagdad, at the beginning of the tenth century, though before his day several authors had employed in their writings terms which suggest smallpox. The word "*variola*" is first met with in an account given by Marius, Bishop of Lausanne, in 570. He mentions it as a symptom of a very fatal epidemic which was prevalent in Italy and France at about that date.

Smallpox was probably introduced into Europe from the East by the Saracens towards the close of the seventh century, and reached England perhaps about the tenth, though it has not been absolutely proved to have existed there before the sixteenth. From Europe it was carried to the New World at the beginning of the sixteenth century.

The disease was called "small pox" to distinguish it from the "great pox" or syphilis.

ÆTIOLOGY.—Smallpox is an extremely infectious disease. In most instances it is caught directly from a person who is suffering from it, either by actual contact or by the transmission of the infection through a short space of air. But the virus can certainly be conveyed by, and stored up in, infected articles, especially clothing, and is readily carried by the hands and garments of third persons. It is also believed by several leading epidemiologists that under certain conditions it can be disseminated for a considerable distance, up to about three-quarters of a mile, through the air, from a hospital in which smallpox patients are being treated ("aerial convection"). This is, however, by no means a universally

scepted view. While the capability of a smallpox hospital to act as a focus of infection is admitted by nearly all, there are not a few who attribute this conveyance of infection to such agencies as members of the staff, visitors to patients, and infected articles; so that, though a good case has been made out for "social restriction," the question cannot be regarded as settled.

History shows that no race is immune to smallpox, and no climate affords protection against it. In some countries it is still more or less endemic; in others it occurs in epidemics, usually local. Occasionally it has been pandemic over a continent. It spares neither age nor sex. Even the unborn infant may be infected and attacked by the disease, while still in the womb of its variculous parent, but this event does not occur during the first three months of pregnancy. The child may be born at any period of its disease. It may also go through the various stages in the uterus and be born scarred, or it may not show any symptoms till a few days after birth. Cases, too, are known in which the child has been born with the eruption out upon it several weeks after the mother has undergone an attack of smallpox.

The influence of vaccination on the age incidence of smallpox is pointed out in the section on Vaccinia.

Left to itself smallpox behaves somewhat like measles, which is comparatively seldom met with in adults because the majority of persons go through an attack in childhood, and one attack almost invariably protects against another. In pre-vaccination days smallpox was almost as much a children's disease as measles and chickenpox are to-day; and even to-day, in smallpox amongst the unvaccinated, the proportion of affected children is large. Thus, of 2,365 patients suffering from smallpox and unvaccinated, admitted to the smallpox hospitals of the Metropolitan Asylums Board during the epidemic of 1901 to 1903, 1,719, or 72.6 per cent., were under fifteen years of age. The proportion of children attacked varies according to the frequency of epidemics, a fact which there are reasons for believing to be true of other infectious diseases.

Taking all ages, males are more liable to attack than females, but the difference is least marked in young children.

In this country epidemics of smallpox usually prevail from the beginning of the winter to midsummer, and generally it may be said that their occurrence is favoured more by cold than hot weather. The incubation period is about twelve days. The extremes are six and eighteen; but only very exceptionally is it longer than fourteen or shorter than ten.

SYMPTOMATOLOGY.—An attack of smallpox begins suddenly, with intense headache, most commonly frontal, pains in the limbs, shivering, pyrexia, and, in rather more than half the cases, pain in the lumbar region. Pain in the epigastrium and frequent vomiting are very constant in children. The patient is thirsty, but refuses food. Suffusion and slight injection of the eyes are not infrequently present. As the attack advances, the child often becomes drowsy, and there are muscular twitchings and grinding of the teeth. Convulsions, which may be frequently repeated, are not uncommon, and are accompanied or exceeded by coma. Or there are delirium and sleeplessness, and the patient is disturbed by frightful dreams. The pulse and respiration rates are increased in frequency, the former often to over 150 per minute. When there is coma the breathing is stertorous. The temperature rises considerably in the course of two or three days, commonly to 103° F., and even up to 106° F. The tongue is thickly furled

and the breath offensive; the bowels are confined, the urine febrile and diminished in quantity.

The Rash.—These symptoms last in most instances till after the characteristic eruption comes out, an event which usually happens on the third day, though it may occur at the end of the first or be put off till the fourth or fifth.

Were this account not confined to smallpox in children, it would be necessary to write at some length on the initial rashes which are to be seen in some adults. But, fortunately for the diagnosis, they are decidedly uncommon in children. A diffuse erythema may on the second day, sometimes on the first, appear on the trunk and limbs. It is prone to consist of irregularly shaped and sized patches of uniform redness, but may also occur in the form of either the punctate rash of scarlet fever or the macular rash of measles. The face and neck are seldom involved. Thomson and Brucelee describe under the term "capitoid" an erythema in which the region of the lower abdomen is of a pale sepia hue, "the affected skin being slightly darker and more livid than that in the neighbourhood." The colour disappears on pressure, leaving a faint yellowish staining. MacCormick has met with an initial rash which he states occurs usually in children, and consists of small dark violet spots, sparsely and irregularly scattered over the trunk and limbs. There are no other hemorrhages accompanying this rash.

All these rashes are uncommon in children, but in hemorrhagic smallpox there is a petechial eruption during the initial stage. This will be described later.

The eruption which is characteristic of the disease shows itself first on the face and wrists as red specks, impalpable and delible on pressure. In the course of a few hours they increase in size and become papular, and by the end of twenty-four hours can be felt as small firm bodies, as it were shot let into the surface of the skin. The next stage is that of vesiculation. According to Bassotti, this begins with the formation of a ring of vesiculation within the macule or papule, enclosing a central area free from vesiculation. Later, vesiculation takes place in the central area. Hence, as the process goes on, the centre of the resulting vesicle, having developed later than the periphery, is depressed or umbilicated. Umbilication, however, is by no means constant. At the end of two or three days the vesicle is fully formed. It is circular, projects from the skin with a sloping edge, as if it had been thrust up from below, and is, if small, dome-shaped; if large, flat-topped. Most of the vesicles are about a quarter of an inch in diameter. They are opalescent and filled with clear or slightly turbid serum, which is contained in several separate spaces, so that in order to empty a vesicle completely of its contents several pricks must be made into it with a pin or needle. The vesicle is surrounded with a red inflammatory area, often of considerable extent. Now comes pustulation. This begins at the periphery of the vesicle, so that in the transition from vesicle to pustule the pock presents a yellowish ring surrounding a grey translucent centre. For the pustule to mature takes about two days. It is then three-eighths of an inch across, hemispherical, full of thin pus, but has lost the umbilication of the vesicle. The surrounding areola has faded. After a day or two the pustule begins to dry up. Often it ruptures, and the contents escape to form a yellow or brownish-yellow crust. A scab results, which is disc-shaped and of a brownish-yellow, or even a reddish-brown, colour. The scab commences to come off on about the fourteenth day of the eruption. The process of separation is of variable duration according to the depth to which the inflammation has penetrated. According to the depth also is the cutaneous lesion left by the shedding

of the scab. It may be a raised or flat stain or a depressed ulcer. On the palms and soles, where the skin is thick, the scabs are somewhat deeply embedded, forming the so-called "scabs," which may not separate for weeks, and are usually removed artificially. The separation of the scabs is attended with unbearable itching.

Round each stain or scar a little desquamation takes place, which is usually slight, but may be considerable when the eruption has been crowded. The stains and scars are pink at first, but later take on a brown hue, and may be very conspicuous and enduring. In severe cases, in which the cutaneous lesion has been deep, the discoloration is followed by permanent white scarring. There may be extensive loss of hair.

The eruption affects also certain mucous membranes. On them the vesicles form more quickly than on the skin; they are smaller, are flattish, and have a white appearance. They rupture before pustulation takes place, and an ulcerated surface is revealed, more or less extensive according to the amount of the eruption. The discharge from this consists of pus and debris of mucous membrane, and is extremely offensive.

It is of the highest importance to note the order of invasion of the skin by the eruption, and the distribution of the poeks, because these points, especially the latter, are of extreme value in diagnosis. The eruption comes out first on the forehead and face, especially about the mouth and nose, and on the wrists. The next day the trunk and the rest of the upper extremities become affected, and lastly the lower extremities. In two to three days the whole of the eruption is out. Inasmuch as the face and wrist are earliest, and the legs last involved, the poeks are always more developed, by a day or two, on the former than on the latter. If any given area of the skin be taken, it will be found that the eruption situated upon it consists, on the whole, of poeks all in the same stage, though here and there a poek may have hurried or lagged in its development. A few of the poeks are smaller than the rest.

The distribution of the eruption, when fully out, is as follows: On the forehead and face, especially the nose; the distal portions of the limbs rather than the proximal; the back of the trunk more than the front; the chest more than the abdomen. Poeks are also present on the scalp, and in all but slight cases on the mucous membranes of the palate, pharynx, and nasal passages, and occasionally on the tongue and sides of the mouth and in the larynx, trachea, and bronchi. Very rarely are they found on the inner surface of the eyelids. In children the sparseness of the eruption on the proximal as compared with the distal parts of the lower limbs is not so pronounced as in adults. Again, in children the eruption is prone to be nearly as profuse on the legs as on the arms; in them, too, the poeks are usually numerous on the buttocks.

Eckstein has pointed out that, besides this broad preference of the eruption for certain regions, it will be found that it has a nicer liking for those parts which are the subjects of stimulation or irritation. Thus, exposure to sun and wind accounts for its density on the face and hands. On parts protected by clothing, the friction to which the skin over prominent bones and tendons is exposed determines the presence of the poeks. On the other hand, places protected from friction, as the armpits, are almost void of poeks. Abnormal stimulation will produce abnormal distribution. For instance, an irritated wound or an inflamed patch of skin will be the seat of many poeks, even though it may be in a position well sheltered from pressure or friction.

Where the eruption is at all abundant the skin becomes swollen and painful, and the degree of swelling is in proportion to the number of poeks. Therefore the face, forearms, and hands, are most affected in this way. The painful swelling of the skin may be so intense as temporarily to cripple the patient. The puffed-out eyelids, swollen to such a degree as to deprive the patient of the use of his eyes, the thickened lips, and the bloated nose, render the features so hideous and distorted that the individual is quite unrecognizable even by those who know him most intimately. There is oedema also of the nasal bones, pharynx, and larynx giving rise to difficulty in swallowing and breathing.

Confluent Smallpox.—The description given above of the evolution of the individual poek has been confined to that form of the disease in which each poek is distinctly separated from its neighbour. But so crowded may be the poeks that to a greater or less extent they merge into one another. The degree of density has partly determined the nomenclature of the disease. Thus, when every poek is distinct from its fellows the attack is called *discrete*; if the poeks have mostly run together it is *confluent*. It should be mentioned that confluence of the poeks is usually confined to the face, arms, and hands, and only in very severe cases occurs elsewhere, more particularly on the back. In severe confluent cases the face seems to be covered with a mask of vesiculation or pustulation, but the dimpling of the mask more than hints at its composition from many separate elements. Intermediate cases, in which the confluence is partial, are often termed *semi-confluent* or *coherent*.

Confluence is usually noticeable towards the end of the vesicular and in the pustular stage, but it may be seen in the papular, in which case the "shotty" character may be absent and the eruption strongly suggests measles. When it is extreme, the cuticle, even in the vesicular stage, may separate extensively and leave raw weeping surfaces.

The symptoms of the initial stage attain a climax a day or two after the appearance of the eruption. As the rash develops the patient becomes easier, and the temperature falls, to reach the normal on the fourth to sixth day of the eruption. This fall occurs even in the most confluent cases, though in them the drop may not be to normal. In some discrete cases the fall is very abrupt. But as the poeks separate the patient exhibits symptoms which are due partly to septic absorption from the pustules, partly to the exquisite pain caused by the cutaneous lesions. The temperature rises again, and may attain a considerable elevation, though it does not as a rule run so high as in the initial fever. This secondary fever, or fever of maturation, lasts from three or four to seven or eight days. When desiccation begins the temperature drops. Besides pyrexia there are sleeplessness, restlessness, and delirium. In severe cases the patient becomes very prostrate, and may be unconscious. A fatal event supervenes more often during this stage than at any other period.

In the pustular stage blebs may form in connection with and about the poeks. They are somewhat painful. They are circular and contain a little thin turbid fluid. When a bleb breaks, its base is found to be formed of excoriated skin.

In mild cases of discrete smallpox, even unmodified, there may be hardly any secondary fever.

In confluent smallpox the initial symptoms are usually severe; so also they may be in discrete cases. A discrete attack may be, and often is, preceded by a mild initial illness; but very rarely is this the case in confluent smallpox.

Again, when the initial illness has been severe and there has been much prostration, the vesicles may not develop in the usual way, but are flat and grey, and contain but little fluid. They are then very different in appearance from the hemispherical vesicles of the usual form of the eruption.

There are two other forms of the disease which remain to be described—the hæmorrhagic and the modified.

Hæmorrhagic Smallpox.—No little confusion has been caused by the adoption of a classification of this variety of the disease which is based upon such accidental circumstances as the sites of the hæmorrhages and the dates of their occurrence. *See*, according to Ricketts, the symptoms which have been described above as constituting the initial fever, the illness which precedes the eruption, are due to an intoxication; hence he calls this the "toxic fever." In this sense every case in which there is an initial fever is toxic, and there are all degrees of toxæmia. Hæmorrhagic symptoms often are the expression of a profound toxæmia, but not every toxic case is hæmorrhagic. At the same time the presence of hæmorrhages does not necessarily connote toxæmia; they may be due to some other cause, such as injury to the pocks or their destructive character. But Ricketts, while he believes the initial fever to be of toxic origin—that is, while he holds nearly every case to be more or less toxic—places a limitation on the word "toxic" when he states that "a patient may be said to suffer from toxic or hæmorrhagic smallpox when his life is menaced by toxæmia."

The constitutional symptoms of hæmorrhagic smallpox are usually very severe, but they are not always so at the beginning of the attack; and even in children it may not be till after a day or two that the gravity of the case is manifest. The pain, whether of the head or back, may be agonizing and persistent. Sleeplessness and delirium are common. There may be profound prostration and enfeeblement of the heart's action. The temperature is variable. In some cases it is high, but in others, and those the worst, it may never rise above 100° F. Just before death there may be either hyperpyrexia or a subnormal temperature. The odour of the breath is particularly offensive, even though there is no faucial lesion. The liver is enlarged. In children two early rashes are occasionally seen, the one consisting of petechiæ scattered irregularly in small numbers over the skin, especially of the trunk and limbs; the other of small, roundish hæmorrhages, up to the size of a split pea, and of a dark violet, almost black, colour. They have the same distribution as the petechiæ, and may be associated with them.

Besides these hæmorrhages there may be larger extravasations into and beneath the skin. They are of a purple, blue-black, or indigo colour, and are of various sizes, sometimes occupying extensive areas, and are irregularly distributed. If the eruption of pocks shows itself during the toxic stage, blood may be effused into the areola round the pock or beneath its base (subvesicular). Bleeding may occur from any mucous surface, so that there may be epistaxis, bleeding from the gums, hæmoptoe, and lungs, hæmæma, and hæmaturia. Subconjunctival ecchymosis is not uncommon. Severe toxic cases, in which the patient survives to the late eruptive stage, will even then for the first time not infrequently present hæmorrhagic lesions of the skin.

Some patients die with hæmorrhagic symptoms before the eruption proper shows itself; and in a few cases, especially of young children, a fatal result from collapse may take place before the eruptive stage and without any hæmorrhage.

Death is seldom due to hemorrhage; usually it is caused by heart failure or pulmonary oedema.

Modified Smallpox, or Varioloid.—In the account of smallpox given above, the eruption is described as going through certain definite stages of papules, vesicles, pustules, and scales. Each individual pock passes through these stages, and the severity of the attack is determined very largely by the number of pocks. These definite stages are regarded as constituting the natural or normal course either of the single pock or of the multiple eruption. But the course run by each pock, and therefore by the eruption as a whole, may be so altered in the way of mitigation as considerably to lessen the severity of the attack. When such is the case, the patient is said to be suffering from modified smallpox. This lessening of severity of attack is met with in a few persons who have neither been vaccinated nor suffered previously from smallpox; indeed, there are a very few individuals who are insusceptible to smallpox altogether. From this absolute insusceptibility can be traced all degrees of immunity, as evidenced by the character of attack, up to the most malignant hemorrhagic form. As Ricketts has pointed out, "there is no distinct dividing line between modified and natural smallpox." But though these modified attacks, which differ from the natural attack in being milder, may be met with amongst the unvaccinated, they are very much more often seen in the vaccinated. The power of resisting, completely or partially, an attack of smallpox is either congenital or acquired by vaccination. According to Ricketts, congenital immunity is shown by sparseness of eruption rather than by alteration in the character of the pocks, though even in the unvaccinated the eruption may be modified in this latter respect. In immunity conferred by vaccination the character of the pocks is altered, but the eruption may or may not be copious.

The well-vaccinated infant, immediately after vaccination, possesses an acquired immunity to smallpox in addition to any degree of immunity he may have been born with. As time goes on he gradually loses his acquired immunity, and finally parts with it altogether; but he may still go on retaining his congenital degree of insusceptibility. We have seen that acquired immunity is shown by both sparseness of eruption and alteration of pocks. The first of the two factors to be lost is that which makes for a scanty eruption, but even when this is lost the other factor may for some time remain more or less influential.

The evidence that an attack of smallpox is modified may become manifest at an early stage of the disease. The initial period may be shortened and the symptoms very mild. There may be no eruption, even though the initial symptoms have been of a sharp character (*variola sine variolis* or *variolous fever*). When the eruption comes out it may be unusually sparse. As a rule the papules in a case of modified smallpox do not differ markedly from those of the natural disease; it is when vesiculation is reached that differences in character appear. The vesicles are smaller and more superficial. They may dry up quickly without pustulation. If pustules are formed, they too are small and superficial. Umbilication is wanting from the vesicles. There is less inflammation around and beneath the pocks, and less swelling of the skin. On the face the larger pocks are prone to have fleshy bases surmounted with small vesicles and pustules. Ricketts says that such lesions may leave wart-like excrescences which persist for a long time. While in natural smallpox an insignificant minority of the pocks are smaller than the rest, in the modified disease there is much wider variety of size and character.

The constitutional symptoms of the eruptive stage are less severe than in *variola* smallpox. If there is little or no pustulation, there is no secondary fever; and even though there may be many poeks which suppurate, yet the lesions, being superficial, do not give rise to the same severe symptoms of septic absorption as occurs in the unmodified form of the disease.

It must not be forgotten, when discussing modification, that smallpox is a disease which varies much in its virulence in different epidemics; so that mildness of attack may be due to weakness of virulence on the part of the disease as well as to inaccessibility, congenital or acquired, on the part of the patient. Some ten or twelve years ago there was prevalent in certain of the United States and in the West Indian Islands epidemic smallpox which was hardly at all fatal even to the unvaccinated. A disease which has been prevalent from time to time in South Africa, and is there known as *owsoor*, would appear to be a mitigated variety of smallpox.

COMPLICATIONS AND SEQUELÆ.—*Boils and abscesses* are common. They occur during the scabbing stage and later, and are most often found on the scalp and extremities, but may affect any part of the body. They may be present in large numbers and be of varying size. Sometimes they follow one another in rapid succession and delay recovery for several weeks. Usually they are superficial; but occasionally an abscess will be deep, as the result of a severe cellulitis which may be very extensive, even to the implication of the whole of a limb. Such cases may be fatal.

During the stage of desiccation secondary rashes are not uncommon; they take the form of an erythema, like that of scarlet fever or measles, or of an impetiginous eruption, which in some cases seems to spread from the poeks. The latter may be accompanied by considerable febrile disturbance, and prove fatal from septicæmia.

The eye and its appendages are frequently the seat of lesions more or less serious. Ophthalmia is common, especially in confluent and severe discrete cases. It sets in during the pustular, sometimes even during the vesicular, stage. In severe cases not only the conjunctive, but also the eyelids, are inflamed, and may be so swollen that they cannot be separated. From this follow, on the one hand acute keratitis, with ulceration, sloughing, and even panophthalmitis, and on the other side inflammation of the Meibomian glands, and scarring and eversion of the lids. The cornea may become inflamed without there being any general conjunctivitis. In very confluent cases it may become quite opaque within thirty-six hours; then rapid sloughing takes place, and this is followed by prolapse of the iris, escape of the lens, and panophthalmitis. All this may occur without signs of inflammation and without pain and photophobia. Even if the eye is not destroyed as a result of these complications, sight may be impaired by the persistence of such lesions as corneal opacities. MacCombie describes a form of corneal ulceration which, starting at one margin of the cornea, creeps over the front of the eye to the other margin. As the ulceration progresses, healing takes place in its wake. There is a good deal of injection, with pain and intolerance of light. *Iritis* is not common. In hæmorrhagic smallpox there may be hæmorrhage into the retina.

Inflammation of the middle ear with its sequels is not infrequent. The respiratory tract is very often involved. *Laryngitis*, set up by the poeks, may be sufficiently severe to necessitate tracheotomy. Very occasionally this complication comes on during early convalescence. *Bronchitis* and *lobular pneumonia* are extremely common, and are a frequent cause of death. They supervene during

the pustular stage of confluent cases. *Lobar pneumonia*, *pleurisy*, and *empyema*, may also occur.

Welch and Schramberg state that *arthritis* is occasionally met with, especially in little children. *Nephritis* is rare, but *albuminuria* is often present in the early stages. *Nervous complications* are uncommon in children. *Septicæmia* is frequent, *pyæmia* less so. Inflammation and suppuration of the cervical and axillary glands are often met with. *Relapses* and *second attacks* are very rare.

MORBID ANATOMY.—Pocks are found after death, not only on the skin and mucous membranes, but often in the larynx, trachea, and bronchi, very occasionally in the upper part of the œsophagus, about the anus and lower part of the rectum, and just within the orifice of the vagina. On mucous membranes they usually appear as small ulcers, sometimes with blood upon them. The liver is enlarged, and, in cases which are early fatal, the spleen also. In the testes, even of children, there is degeneration of the spermatogenic cells, and areas of cell infiltration with necrosis are scattered amongst the tubules.

The Blood.—There is considerable leucocytosis, more especially of the small and medium-sized mononuclear cells, the polymorphonuclear elements being diminished in number. In severe cases there is marked destruction of the red corpuscles, and transitional forms of cells are found. In hemorrhagic cases the leucocytosis may be slight or absent. In the early stages of the disease the eosinophils are increased in numbers. In fatal cases the leucocytosis disappears before death.

The pock begins as a localized hypæmia. Then swelling and degeneration of the epithelial cells set in, and result in the formation of intracellular spaces. In the cells of the deepest layers the spaces are more or less angle and fill out the cells; in the cells above they are reticulated. This degeneration is more widely spread amongst the upper than the lower layer of cells, so that a vertical section of the pock shows it to be somewhat mushroom-shaped, with the stalk below. Serum exudes from the vessels of the corium, especially at the periphery of the lesion; and this early peripheral exudation is the principal cause of the umbilication of the vesicle. The fluid escapes upwards between the cells, many of which rupture, and the remains of the cells and their trabeculae form the septa of the localized vesicle. At first the fluid is clear and without leucocytes; later, probably through bacterial infection, leucocytes pour out into the vesicle, and a pustule is formed. Lastly, the contents of the pustule dry up by absorption or evaporation of its moisture, and a crust or scab is left. If the inflammation in the pock has not gone right through the epithelial layers down to the corium, healing takes place without scarring; but if all the layers of epithelium are destroyed, scarring results. In hemorrhagic smallpox, fatal before the pocks have appeared, degeneration of the cells, such as has been described above, can be made out, but there is no exudation from the corium. In these cases streptococci abound in the vessels and perivascular spaces of the corium.

PATHOLOGY.—Analogy leads us to suppose that smallpox is due to a specific micro-organism. But so far, in spite of prolonged research on the part of many observers, no such organism has been definitely identified. Of late years attention has been drawn to peculiar changes in the epithelial cells of the skin which are to be found in smallpox and varicella; and there are those who are of the opinion that

these changes are due to the presence of organisms of a protozoic nature. On the other hand, some observers, without questioning the reality of the changes, refuse to admit that they are anything but the expression of a cellular degeneration peculiar to smallpox. The changes are to be found not only in smallpox, whether acquired naturally by a human being or given by inoculation to a monkey, but in vaccinia. They suggest the penetration of the body of the cell by a spore-like but minute organism which grows till it assumes an amoeboid form. In it small round bodies, called "gemmules," arise, which, escaping into the cell substance, penetrate the nucleus. Here it is supposed that from the gemmules two bodies are formed (sexual forms, gametocytes), which unite into a single body, the zygote. The zygote undergoes various changes, which result finally in the formation of minute spore-like bodies; and these escape from the cell and penetrate other cells in the immediate neighbourhood, or are carried to other parts by the blood, or are thrown off in the serum or pus of ruptured poeks. The organism which goes through these changes was named twenty years ago, by Guarnieri, the "*cytocytoes variolæ*." How it obtains an entrance into the body so as to set up an attack of smallpox is not known, but it is thought that it finds its way in at some point in the respiratory tract.

The supuration of the poek is believed to be due chiefly to secondary infection with other organisms, especially streptococci. Smallpox is very seldom met with in animals in the same guise as in the human subject. Epidemics have been described amongst monkeys. But Councilman and others, experimenting with certain macaques and orangutans in the Philippine Islands, failed to infect these animals save by inoculation with variolous matter. If the monkeys were kept in wards with smallpox patients they did not catch the disease.

Smallpox can be given to a human being by *inoculation*; after two or three days a local lesion, which finally becomes a pustule, is found, followed in eight to ten days after inoculation by an attack of smallpox with a generalized eruption. From smallpox induced by inoculation the infection can be conveyed in the ordinary way to susceptible persons. As we shall learn when discussing vaccinia, smallpox in some animals, especially the cow, takes the form of a local lesion without any general infection.

DIAGNOSIS.—So variable are the manifestations of smallpox that it is not surprising that errors of diagnosis are by no means infrequent.

Is the Pre-Eruptive Stage.—These errors may be made at almost any stage. Before the characteristic eruption comes out, smallpox presents symptoms which are common to all acute febrile diseases, and hence difficulties of diagnosis arise. The initial symptoms of this disease and of influenza are extremely similar in some cases. But, fortunately, the eruption in smallpox is seldom delayed beyond the third day, so that the period of suspense is short. Smallpox, if suspected, should not be put out of court because there is no lumbar pain; in nearly half the cases this symptom is absent. Fortunately, too, the initial rash, mentioned above, which is not infrequently met with in adults, are decidedly uncommon in children under ten, and even in those under fifteen, years of age. Hence a scarlatiniform or morbilliform erythema in a child is not likely to be an initial smallpox rash. The difficulties really begin with the eruption proper.

From Measles.—This, as has been already pointed out, may be mistaken for measles, especially when it is confluent and lacks its shotty feel. The resemblance to measles may be enhanced by injection and suffusion of the eyes. But in small-

pox there are no Koplik's spots, for which search should be made in every child who is ill with doubtful acute symptoms, and there are not the frequent sneezing and short suffocative coughs and the increased respiratory rate that are present in measles. If, as is sometimes the case, the smallpox eruption comes out on the mucous membrane of the mouth before it appears on the skin, it affects chiefly the palate and fauces, whereas Koplik's spots are situated on the sides of the mouth.

From Enteric Fever.—The poeks of smallpox, while still hardly papular, may be mistaken for the spots of enteric fever. But the rash of this disease rarely comes out till after at least four days' illness, very seldom affects, and never begins on, the face.

From Erythema Multiforme.—One or other of the varieties of erythema multiforme may be mistaken for the early stage of the variolous papules. The chief distinctions are that these rashes, if they show any preference in distribution, choose the extremities, and at any rate do not select the distribution taken by the smallpox eruption, and that the elements of which the rash is composed vary more in size and character, and are more superficial, than those of smallpox. These erythemata are due to various causes, though often the cause is not obvious. Febrile symptoms are frequently present with the erythema.

From Varicella.—In the vesicular stage smallpox is most often confounded with chickenpox. In fact, of the mistaken cases that are sent to the smallpox hospitals of the Metropolitan Asylums Board, more than half are chickenpox. Now, is the diagnosis between smallpox on the one hand and chickenpox and any other vesicular or pustular eruption on the other, the most important point for consideration is the distribution of the poeks. That of smallpox has already been described. In chickenpox the poeks affect mostly the trunk, and not one part of it more than another. The face and scalp may or may not be equally or more or less implicated; the parts of the limbs adjoining the trunk may be invaded, but the distal portions, and often the entire limb, are free. The eruption does not show a preference for those parts of the skin which are exposed to stimulation or irritation. A few poeks may be seen on the palate. Next to the distribution the character of the lesions is of importance. In varicella these are superficial, with little inflammation either around or beneath them. Oval poeks can usually be seen. As the poeks begin to dry up they become flat, and present an irregular, crenated edge. The scabs are small and yellowish-brown, and are usually but slightly attached to the surface of the skin. Over a small area, if the eruption be plentiful, lesions of different ages may be present together—papules, vesicles, pustules, and scabs. The vesicles are rarely umbilicated, and are multicellular. In chickenpox the eruption is in most cases amongst the first signs of the disease, whereas in smallpox it is rare for the initial period to be entirely wanting. In modified smallpox, with which chickenpox is most likely to be confused, the eruption, even though scanty, very seldom departs from its usual distribution; and the poeks, even though they may not develop as in the natural disease, never assume the positive characters, mentioned above, of the chickenpox lesions.

From Certain Other Skin Eruptions.—Other papular, vesicular, and pustular eruptions that have been misdiagnosed as smallpox are acute eczema, vesicular and pustular dermatitis, impetigo, scabies, erythema bullosum, dermatitis herpetiformis, and bromide and iodide eruptions. But a careful attention to the following points in the order mentioned should obviate error: distribution, characters of the elements of which the eruption is made up, and history.

From Typhus Fever and Purpura.—I have more than once known hæmorrhagic smallpox to be mistaken for typhus fever and for purpura hæmorrhagica. In these cases mistakes can be made only before the pox has appeared. In typhus the rash does not come out before the fourth day, is erythematous for three or four days before it becomes petechial, and does not affect the face. Fulminating hæmorrhagic purpura is not always easy to distinguish from hæmorrhagic smallpox. Fortunately it is rare. The hæmorrhages in purpura do not often assume the indigo colour seen in many cases of smallpox. Most cases even of fatal but not fulminating purpura live much longer than does the hæmorrhagic smallpox case.

If a child who is the subject of a doubtful eruption be vaccinated, and the vaccination be successful, then the eruption cannot be that of smallpox. This is the experience of most observers. But there is some evidence to show that, if vaccination be performed early in the eruptive stage of smallpox, it may very occasionally prove successful.

PROGNOSIS.—There are four chief points governing the prognosis: (1) The vaccinal condition and (2) the age of the patient; (3) the character of the attack and certain of the symptoms; (4) the character of the epidemic.

1. This is the most important of the four factors. It may be illustrated by figures derived from the records, which have been referred to already, of the cases treated in Metropolitan Asylums Board hospitals during the years 1901 to 1903. Of the 1,719 unvaccinated children under fifteen years of age 533 died, or 31·0 per cent.; while of the 479 vaccinated children admitted during the same period 5 died, or 1·2 per cent. The better vaccinated a child is—that is, as judged by the number and area of his vaccination marks—the less likely is he to get smallpox badly if he takes it at all.

2. Amongst the unvaccinated, the younger the child the more likely is the attack to be fatal. Amongst the vaccinated the reverse is the case; the younger the patient the higher probability is there of his recovery. It is, in fact, very unusual for vaccinated children under ten years of age to die from smallpox.

3. The more profuse the eruption, the greater chance is there that the patient will die. But the influence of vaccination must be taken into account, and this justifies a more favourable prognosis in the vaccinated. The eruption even in a confluent case will be deprived of most of its power of doing harm in the suppurative stage by the influence of vaccination. Hæmorrhagic cases are invariably fatal. Severe involvement of the fauces, and even a moderate implication of the larynx, are unfavourable occurrences; so also is absence of swelling of the face with a profuse eruption. A severe initial stage does not necessarily herald a severe eruptive stage, but a mild initial stage never precedes a severe eruptive one. Broncho-pneumonia is a grave complication.

4. As has been mentioned above, in some epidemics the power of the virus seems to be weak, so that the bulk of the cases, even amongst the unvaccinated, are mild or of only a moderate severity.

TREATMENT.—Isolation.—The patient should be isolated and confined to bed directly the nature of his illness is even suspected of being smallpox. Isolation is best carried out in a hospital set aside for the purpose. The infection of the disease is so readily carried about by third persons that the restrictions necessary to be maintained amongst the attendants upon the patient could be insisted upon in a private dwelling only with very great inconvenience to the members of the household generally.

Vaccination of Contacts.—Every effort should be made to persuade those who have been brought into contact with the patient to be vaccinated or revaccinated, as the case may be. If a person has been infected, the subsequent attack may be mitigated to a degree dependent upon the time of vaccination. The earlier during the incubation period that vaccination has been successfully accomplished, the more profoundly modified will be the attack of smallpox.

Diet.—The patient should be put upon a fluid diet if the attack is at all severe—milk, beef-tea, etc., with plenty of water or lemonade. In very mild attacks the diet may be more substantial.

Symptomatic Treatment.—The vomiting of the initial stage will be relieved by giving substitute of bismuth, peptonizing the milk, or allowing the patient to suck ice. For headache, sleeplessness, and delirium, and also for convulsions, chloral hydrate and potassium bromide are most useful. If necessary, they should be given by the rectum. Warm baths are also very soothing for these conditions as well as for the headache, for which salicylate of sodium should also be prescribed. Delirious patients require most careful watching, as they are very prone to get out of bed and escape from the ward. When the eruption comes out, the hair should be cut short—at any rate, if the poeks are at all numerous. In the papular and vesicular stage compresses of cold (even iced) water, or of glycerine and water, a drachm to an ounce, applied to the skin, will afford great relief. The face should be covered with a mask dipped in the liquid. Welch and Schanberg speak favourably of painting the skin with pure tincture of iodine twice a day for eight or nine days, at the end of which period some antiseptic ointment should be applied. In some cases the pure tincture is too painful, and then a diluted solution should be used. If the patient cannot tolerate a mask, his face should be dusted over with starch and oxide of zinc or boracic powder, or vasoline, to which 3 or 4 per cent. of carbolic acid has been added, should be lightly painted on. When the poeks suppurate, iodoform should be mixed with the vasoline. Crusts and scale should be removed by the application of a linseed-meal or starch poultice, smeared over with vasoline and iodoform, and renewed every two hours. The ulcerated and excoriated surface left by the removal of the crusts should be treated with some antiseptic ointment. Boracic fomentations will also remove crusts.

Warm baths are extremely grateful in cases in which the eruption is profuse. Hebra and others have spoken highly of the continuous warm bath. This is somewhat difficult to manage except in a room specially fitted up, but the patient may be kept for some hours in a warm bath with very little trouble.

The deep-seated poeks on the palms and soles should be opened with the point of a fine scalpel, and the contents expressed.

It will be necessary to place the patient on a water bed and pillow when the eruption is profuse, and the nurse must exercise extreme gentleness when moving him, in order to avoid giving pain.

During the stage of the separation of the crusts the almost intolerable itching will be relieved by warm baths or by sponging with warm water slightly acidulated with acetic acid.

In severe cases, when the eruption is coming out upon the fauces and larynx, dysphagia may necessitate feeding by nutrient enemata for two or three days, and the dyspnoea due to laryngeal obstruction may require tracheotomy; but an operation may be averted by placing the child in a steam-tem.

In most cases the mouth will require frequent attention, and should be washed

or gently scrubbed out with a solution of chloride of potash, glycerine and borax, or chlorinated soda. When suppuration and sloughing have taken place, an antiseptic may with advantage be added to the mouth-wash.

From the very first the eyes should be bathed frequently with boric lotion, and vaseline may be applied to the lids to prevent them from sticking together. Other complications should be treated according to their nature.

Stimulants are indicated only in certain cases where there is heart failure during the secondary fever, and in hemorrhagic cases, but the treatment of the latter is very hopeless and unsatisfactory. Since there is every reason to believe that suppuration is due to secondary infection by streptococci, it seems reasonable to recommend the use of an anti-streptococcal vaccine during the vesicular stage; but I am not aware that any cases illustrative of the trial of this method have been published. Anti-streptococcal serum has been used in a few cases, with apparently favorable results.

The treatment by red light, secured by keeping the patient in a room from which all rays but the red are rigorously excluded, which was advocated some years ago by the late Professor Finsen, has not met with success in the hands of authorities in this country and the United States. It is said to have a most depressing effect on the patient and those who are in attendance upon him.

Treatment of Convalescents.—The management of the convalescent stage should be on general principles. As regards the length of infectivity, no patient should be looked upon as free from infection till all scabs have completely separated and all ulcers and abscesses have healed.

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VACCINIA.

SYNOPSIS.—Variola vaccinae; Cowpox.

INTRODUCTION.—It bears witness to the dread in which smallpox was held by mankind that, for centuries before the introduction of vaccination, methods, based upon the observation that one attack of smallpox protects against another, had been in vogue, chiefly amongst Eastern nations, to avert an attack, possibly severe, of the disease by submitting the individual to a mild attack under chosen favourable circumstances. One of these methods—inoculation—was introduced into England in 1721. In this method superficial scarifications were made in the skin, and matter from a smallpox pustule was inserted into them. In a day or two a small papule appeared at the site, and successively became a vesicle and a pustule. The pustule was at its height on the tenth day after inoculation, and was then sur-

rounded by an inflamed areola. In this areola several secondary pustules appeared. The pustules then dried up and the crust separated, to leave an ulcer, which resulted in a cicatrix. On about the ninth day from inoculation the patient experienced the initial symptoms of smallpox, and the eruption came out as usual on the third day. In consequence of careful selection, the attack was in most instances mild, but occasionally it was severe, and even fatal. Moreover, the disease was infectious, just as it is when it is acquired naturally. After the introduction of vaccination inoculation was made, and it still remains, illegal.

During the latter half of the eighteenth century the notion prevailed amongst the dairyfolk—in the West and South-West of England, at any rate—that any of them who contracted sores on their hands from certain ulcers which occurred occasionally on the teats of cows were protected from smallpox; and there is evidence that some of them practised to a limited extent the inoculation of human beings with the virus of this disease, which they knew as “cowpox,” with the view of affording protection against smallpox. Thus, Benjamin Jesty, a farmer of Yarnham in Dorset, deliberately inoculated his wife and two children in 1774. The medical practitioners of those parts were acquainted with these ideas and facts, but appear to have lacked the faith of the vulgar, and it was reserved for Edward Jenner to test scientifically the popular belief and place it on a firm foundation.

Cowpox, or *vaccinia*, is a natural disease of the cow, and it may at once be stated that it is now held to be, as Jenner believed, smallpox of the cow. In the animal it shows itself as inflamed papules upon which vesiculation takes place. The vesicles are ruptured, and indurated ulcers are exposed, from which exude pus and blood. Upon the ulcers dark brown or black crusts form. After a time the ulcers heal, and result in irregular, depressed cicatrices. In the human subject the disease, when acquired straight from the cow, differs little from the form it assumes in the animal.

Natural cowpox is not common at the present day either in the animal or in man. It is probable that it was more often met with when smallpox was more prevalent than it is now, and when inoculation was in vogue.

Symptomatology.—If the virus of cowpox, animal or human, be inoculated into a human being, there results at the end of about three days a papule. In another two or three days a vesicle will have formed, the edge of which is raised and the centre depressed. During the following two or three days the vesicle enlarges and becomes distended with clear lymph. Towards the end of this period inflammation takes place round the vesicle, and a red, indurated areola is formed. As this increases in size the contents of the vesicle become purulent, and by the eleventh day the inflammation begins to subside and the pustule to dry up. A brownish-red scab forms, which separates in another ten or twelve days. A pink scar is left, which after a time becomes white and depressed, and presents a dotted or forested appearance. In some instances the local inflammation is extensive and the nearest glands are enlarged. At the same time there may be febrile symptoms.

Complications.—While in the vast majority of cases the results of vaccination are those which have just been described, in a few instances certain complications may arise. These may be due to vaccinia itself or may be accidentally acquired.

1. *Complications due to Vaccinia itself*—(a) *Generalized Vaccinia*.—At a variable time after vaccination, usually from the fourth to the seventh day, there appears

widely scattered over the skin, as eruption of dark red papules, which in the course of two or three days go through the processes of vesiculation, pustulation, and maturation. They then very closely resemble the pustules which are the local result of vaccination, but in some instances they are much like the pox of variola. The papules continue to come out in successive crops for several days, so that on any given day all stages of the development of the pox may be seen. There may be some febrile disturbance while the rash is coming out.

[b] *Auto-inoculation*.—By this is meant the production of vaccine lesions in various parts of the body by accidental inoculation with matter from the original vaccine pox. The most common way in which it is brought about is by scratching some slight wound or raw surface with fingers infected with the vaccine virus. Several separate pox may be produced in this way. Usually they are of little moment, but if situated on an eyelid or near an eye serious inflammation of the organ may be set up.

[c] *Various Eruptions* (Urticaria, Erythema Multiforme, and papular, vesicular, and pustular Rashes).—These may appear at any time during the week after vaccination. They are comparable to the rashes which follow the use of antitoxins, and are to be seen in several of the infective diseases.

2. *Complications accidentally acquired*.—These are chiefly erysipelas and septic infections of various kinds which may lead to ulceration or gangrene. They are due to accidental contamination of the pox. But the virus of erysipelas may be inoculated (inactivated) along with the vaccine lymph. In a very few cases erysipelas has been invaccinated.

METHOD OF VACCINATION.—It is usual to select the skin over the insertion of the left deltoid for vaccination, as it may be necessary to keep the arm at rest while the pustules are forming. The area chosen is well washed with soap and water. The instruments to be used—a slightly blunt lancet or one of the arrangements of needles that have been invented for the purpose—should be sterilized by boiling. The lymph, which is usually contained in a capillary tube, is blown from the tube as to four small spots about three-quarters of an inch from one another. There are tubes in the market which are furnished with an india-rubber squeezer for this purpose. The places on which the lymph has been deposited are then washed just sufficiently to expose the tops of the papilla, but not to draw more than a minute drop or two of blood. By this process the lymph will be inserted into the skin. The places should be allowed to dry, and should be protected with a pad of sterilized gauze. It is rarely necessary to do more than keep the arm at rest until the scabs have separated; but if the inflammation attending pustulation is severe, boracic, or zinc and starch powder should be dusted upon the affected area. It is important to allow the pustules to dry up without rupture.

The result of a primary successful vaccination should be feveated scars having a total area of not less than half a square inch.

At the present day calf lymph is nearly always used for vaccinating, and public vaccinators are forbidden to use any other. After its introduction, and till quite recently, vaccinia was induced with lymph from another human case, which had been inoculated from a similar case, and so on. As the inoculation was always made on the arm, the practice was known as "arm to arm" vaccination; but the first case of any series had been inoculated from some case of the disease occurring naturally in a cow.

The calf or bovine lymph in use at the present time was originally obtained

from several sources on the Continent. The strain is kept up by inoculation from one calf to another, with now and then the interposition of a human being. The site selected for the vaccination of a calf is the skin of the abdomen, which is shaved for the purpose. Glycerinated lymph is used. The resulting vesicles are removed with a steel scoop or spoon, weighed, and mixed with four times their weight of equal parts of glycerine and sterilized water. Glycerine has the effect of destroying any extraneous organisms—usually various staphylococci—that have found their way into the lymph. The mixture of lymph, glycerine, and water, is drawn into capillary tubes or stored in a cold dark place. It will remain efficacious for eight months.

MORBID ANATOMY.—The changes in the epithelial cells of the skin are very similar to those found in smallpox. They begin in the middle layer of the cells of the rete Malpighii, and consist of swelling followed by vacuolar degeneration, and there are the usual appearances of inflammation in the subjacent corium. The supposed protozoön—the *Cytoryctes vaccinæ*—is like that of variola, but is said to attack the body of the epithelial cell only, and not its nucleus, and not to go through a sexual phase (see p. 991).

PATHOLOGY.—It has already been stated that the virus of cowpox of the cow, when inoculated accidentally or designedly into a human being, produces lesions similar to those seen in the animal. By inoculation these lesions can be transmitted from one person to another, or from one calf to another, or from a calf to a human being, or from a human being to a calf, in series of cases. Very rarely indeed is a generalized eruption produced, and the disease is not communicable except by inoculation. The proof which Jenner accepted that cowpox was a modified form of smallpox was that persons who had had cowpox, whether acquired naturally from the cow or given by vaccination, did not catch smallpox, and were inaseptible to inoculation with smallpox virus. Soon after the introduction of vaccination observers set themselves to produce cowpox in the cow by inoculating that animal with smallpox virus. Though certain experiments were unsuccessful, others were not; and the misinterpretation of these led to much confusion, which veiled the subject in obscurity for a considerable time. But it has now been established that, if a calf is inoculated with smallpox virus, a local lesion is produced just like that which is obtained by inoculation with the virus of cowpox; and if, after the virus has been passed through one or more calves, a human being is inoculated with it, the lesions of vaccinia, and not of smallpox, are produced. That is to say, the virus of smallpox has been modified by its passage through the calves. We must conclude, therefore, that cowpox is smallpox of the cow, and that the manifestations of the latter disease have become changed so remarkably by passage of its virus through the animal, that although they are restricted to a local lesion which is not infectious except by inoculation, yet the power of protection against ordinary human smallpox is retained.

Until lately the only animal known to be capable of suffering from smallpox in a form resembling the disease in man was the monkey; and even in this case the disease must be conveyed by inoculation, because the animal will not catch it in the same way that one human being catches it from another. Inoculation of a calf with virus from a pustule of a case of smallpox in a monkey produces vaccinia.

But quite recently (July, 1912) W. J. Simpson has published certain observations made by him in India during 1894 and 1895. According to him, there exists

amongst cattle in Bengal a disease known by the natives as "gotee," and by Europeans as cattle plague and rinderpest. In one form of this disease there is a generalized cutaneous pustular eruption, like that of human smallpox, and it is the opinion of Simpson and others that the disease is indeed bovine smallpox. From a case of this nature Simpson isolated a diplo-bacterium. From subcultures of this organism he inoculated a series of calves, and in some instances produced localized vesicles like those of ordinary vaccinia. With the contents of these vesicles unvaccinated children were inoculated, and vaccinia was the result. These children were refractory to ordinary vaccine lymph. In a few instances, however, inoculation with the same subcultures produced a more or less generalized papular and vesicular eruption, not only in calves, but also in children—a result which, as Simpson points out, was familiar to Burton in the eighteenth century as following inoculation with varicellous lymph of a mild type.

REVACCINATION.—By this is meant vaccination of a person who has already been successfully vaccinated. In the early days of vaccination it was believed that if the operation had been successfully performed the individual was protected against smallpox for the rest of his life. Subsequent experience has shown that this view is wrong. The immunity conferred by vaccination gradually wanes, and after a time—usually several years—is wholly lost. Vaccination confers immunity against vaccinia as well as against smallpox, and the immunity to vaccinia is lost before that to smallpox. If a person be revaccinated, the result may be the same as occurs in successful primary vaccination; but often a modified lesion is produced. The papule may not vesiculate, or pustulation may be absent, or the stages may be gone through very quickly, or the appearance of the papule may be retarded. In some cases the local inflammation is very severe and accompanied by marked constitutional symptoms.

Every infant should be vaccinated within a few weeks of its birth. The operation should, however, be postponed if the child is in an unhealthy condition, unless smallpox happens to be prevalent and the child is likely to be exposed to infection. I am personally acquainted with several instances in which children convalescent from one or other of the acute infectious diseases of this country have been submitted to vaccination, and in none was there any untoward result. It is advisable to revaccinate every child at the age of ten years, because by that age the immunity induced by the vaccination in infancy will have been very considerably diminished.

VALUE OF VACCINATION.—This is shown by the following facts:

1. *The reduction in the number of deaths from smallpox, both relatively and absolutely, since the introduction of vaccination.* Thus, in London the average annual number of deaths per million persons living from all causes has fallen from approximately 50,000 in 1771-1780 to 15,100 in 1901-1910, and the average annual number of deaths from smallpox per million persons living has sunk from 5,020 in 1771-1780 to 34 in 1901-1910. Vaccination was introduced generally during the years 1801-1810, and the figures for that period are 29,200 and 2,040. The proportion of smallpox deaths to deaths from all causes was 1 to 9 in 1771-1780, 1 to 14 in 1801-1810, 1 to 120 in 1872-1890, and 1 to 444 in 1901-1910. Similar figures may be given for every country in which vaccination has been systematically practiced. Further, it has been shown that these results are not due, or are due only to a limited degree, to the amelioration of insanitary conditions.

2. *The alteration in the age incidence of the disease.* It has been pointed out in the

article on Smallpox that in pre-vaccination times smallpox was a disease of children to such an extent that most of the cases and deaths occurred amongst them, and that the same remark is true of the unvaccinated to-day. For instance, the proportion of deaths under ten amongst the total smallpox deaths in Geneva during the years 1580 to 1700 was 96 per cent., and in Kilmarnock during 1728 to 1764 98 per cent. In London in 1884 it was 34 per cent. If the smallpox deaths in London during this year are divided according to their vaccinal condition, the percentage is under 2 for the vaccinated, and 61 for the unvaccinated. As with deaths, so with attacks. The close investigation of epidemic after epidemic during the last twenty-three years has proved that smallpox attacks the unvaccinated child much more frequently than it does the vaccinated. Thus, in Gloucester in 1886 the attack-rate in children under ten amongst the vaccinated was 8.8 per cent., and amongst the unvaccinated 46.3 per cent.; while, of persons over ten, 32.2 per cent. of the vaccinated were attacked, and 50 per cent. of the unvaccinated. The immunity acquired by vaccination in infancy becomes feeble as the child grows up, and the effect of this is shown by the shifting of the age incidence of smallpox from childhood to higher ages. If revaccination at the age of ten was universally practised, smallpox would be stamped out.

3. *The variolous test.* Before inoculation was made illegal, this test—that is, the inoculation of vaccinated persons with smallpox virus—was applied to very large numbers of persons, and very few reacted positively; whereas it was very rare for an unvaccinated person who had not suffered from smallpox to fail to react positively.

4. *The immunity of the well-associated staff of smallpox hospitals.* In these hospitals the staff, if protected by vaccination and revaccination, as they almost invariably are, mingle with the patients with impunity. But in the fever hospitals a certain proportion of them will always be found to catch scarlet fever, diphtheria, etc., as the case may be.

5. *Clinical effects.* The effect of vaccination in modifying an attack of smallpox has been mentioned in the section on that disease. Modification occurs much more frequently amongst the vaccinated than the unvaccinated, and presents certain differences in the two classes. It has also been proved that the more efficiently vaccination has been performed, as shown by the area of the formed scars, the more secure is the child from a severe attack of smallpox.

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SCARLET FEVER.

SYNONYMS.—Scarlatina.

INTRODUCTION.—We do not possess such cogent evidence of the antiquity of scarlet fever as of smallpox, measles, and diphtheria. There can, however, be hardly any doubt that the disease did exist many centuries ago, but that it was

confused with measles on the one hand, and diphtheria and various forms of septicæmia on the other. Ingrassias of Palermo described in 1593, under the names "*rosalia*" and "*rossaria*," an epidemic at Naples, which was distinguished by him from measles, and may well have been scarlet fever. In his account he makes use of the word "*scarlatina*," which had previously been employed of some disease by Lancisotti in 1525. After the time of Ingrassias there are accounts by several writers which strongly suggest the continued prevalence of the disease in Europe. Thus, Sennert of Wittenberg described it in 1619. He quotes Ingrassias, but prefers to connect the disease with measles. Sydenham, in the middle of the seventeenth century, was the first writer, so far as is known, to make use of the term "*scarlet fever*" (*scarlatina febris*); yet he writes of it as if it were nothing new, and Hirsch was of the opinion that probably Sydenham was not the first to use the term. Even then it was some time before the distinctness of the disease was admitted by the whole faculty. Morton, for instance, believed it to be a variety of measles. So late as 1859 diphtheria was not separated from scarlet fever in the Registrar-General's Reports.

Scarlet fever reached the American colonies probably at the beginning of the eighteenth century. A severe epidemic of an eruptive anginous fever, described by Douglass, prevailed in New England during the years 1735 and 1736.

Ætiology.—Dissemination.—There are certainly three ways by which the virus of scarlet fever may be disseminated: (1) By the direct transmission from the sick to the healthy, (2) by infected fomites, and (3) by infected milk.

1. The disease is directly disseminated in much the same way as is diphtheria, by such acts as kissing. But it is very probable that the virus is scattered for a short distance through the air by coughing and sneezing.

2. The tendency, nowadays, is to minimize the importance which was at one time attached to the agency of infected articles in the dissemination of infectious diseases, and to emphasize that of the living person who is either suffering or recovering from one of them. But in the case of scarlet fever there is a considerable body of evidence to show that the infection may be harboured in clothing and boots, and on eating and drinking utensils and other articles, in some instances for considerable periods of time.

3. Lastly, there can be no doubt that milk is occasionally the vehicle of dissemination, and in the case of a milk epidemic the question arises, Whence has the milk been infected? In some instances it has been shown that the source of infection has been human, and that some person engaged in the collection or distribution of the milk has been suffering or has recently recovered from scarlet fever. In others it is believed that the source has been bovine, and the contamination has been attributed to a disease of the cow (bovine scarlet fever). This belief dates from Power's investigations in 1886 of an epidemic in which the infected milk was derived from certain diseased cows at Hendon. The bovine disease was general, with local ulcerative manifestations in the udder. Lately Savage has suggested that the local ulceration of the udder is secondarily infected from a human source with the virus of scarlet fever, and therefore that the cow acts merely as a passive carrier of the virus, as is also believed to be the case in diphtheria.

Variations of Virulence.—One of the most remarkable features of scarlet fever during recent years is the reduction in the mortality, as calculated at so many deaths for so many persons living. Though subject to considerable variation,

the annual mortality per million living since registration of deaths was established in 1838 was, in England and Wales, only twice below 500 before 1883—namely, in 1860 and 1861. In 1863 it reached its highest point, 1,478. It was 1,418 in 1864, 1,446 in 1870, and 1,050 in 1874. Since that date, with the exception of occasional slight rises, it has been steadily falling. From 1875 to 1884 there was an uninterrupted decline from 755 to 218. The most notable fall was in 1884-85, from 402 to 233. Since 1884 the mortality has never been so high as 300. The lowest point reached was 80, in 1903.

The same diminution in mortality has taken place in Scotland and Ireland. The cause of it is probably complex; but an important factor appears to be a lowered virulence, as shown by a lessened fatality (case mortality). The prevalence does not appear, on the whole, to have declined to any considerable extent. Of recent years the fatality in the fever hospitals of this country has been 2 to 3 per cent.

Distribution.—Scarlet fever is a disease of temperate climates. It has seldom gained a hold upon hot countries. To India it has been introduced from time to time by persons coming from Europe, but has very seldom spread beyond the coast or among the natives.

Season.—In the United Kingdom it is more prevalent from the latter part of the summer to the beginning of the winter. The maximum number of cases occurs in October, the minimum in March and April.

Age.—Most of the patients are under ten years of age. When the cases are arranged in age periods of five years, it is found that the period 5 to 9 contains the greatest number. The disease very seldom attacks infants under one year. At all ages more males than females are attacked.

Sepsidæ Cases.—It frequently happens that isolated cases of scarlet fever occur, which cannot be connected either with previous cases or infected articles; in fact, cases have been recorded which suggest that the causal organism, whatever it may be, may remain about a person's fauces or nose for a considerable length of time before it is rekindled into activity. Thus, of two children in a certain house, one was seized with scarlet fever and was removed to an isolation hospital, and the usual disinfection was carried out. Five weeks later, while this child was still in hospital, the other child, a boy not quite two years of age, while playing with a tin whistle, ran against a wall of a room in such a way as to drive one end of the whistle against his palate so that a wound was inflicted. The child was at once taken to a hospital, which was some way off, and admitted. The wound was sewn up. On the eighth day after admission, no other case of scarlet fever having occurred in the ward, the child developed scarlet fever. The wound had not healed at the time. It is suggested that the infective agent was deposited on the fauces at the time the first child was attacked with scarlet fever, but that it remained inert until the inflammation set up by the wound inflicted five weeks later stirred it into activity. In other cases children of the well-to-do classes have been known to develop scarlet fever a few days after an operation on the fauces, such as the removal of enlarged tonsils, under circumstances which preclude all supposition that they received the infection at any time within a week before the operation.

Breath Cases.—There are reasons for believing that the virus of scarlet fever is highly tenacious of life. Not only have we the evidence of its remaining active for long periods on infected clothing, as has already been mentioned, but every now

and then we meet with cases in which, though the patient has apparently quite recovered from an attack of the disease, he is obviously infectious. In some cases it is not, perhaps, correct to say that the patient has quite recovered. He may have been left with some slight, seemingly trivial, inflammation, especially of the fauces or nasal passages. But in some there is no such sequel. The patients to whom I refer, whether they have completely recovered or not, remain infectious for weeks or months, as is shown by the fact that they infect susceptible persons who are brought into contact with them. These cases have been, and still are, the bugbear of fever hospital authorities. The secondary cases to which the apparently recovered infectious case, the "infecting case," gives rise are known colloquially as "return cases." There are usually infecting cases to the number of about 4 per cent. amongst the patients discharged from fever hospitals. But they are by no means confined to these institutions. A remarkable case, unconnected with a hospital, which came under the notice of the writer, will be found recorded in the Transactions of the Epidemiological Section of the Royal Society of Medicine (vol. ii., p. 76).

Carriers.—From what has just been stated, it appears that there are scarlet fever carriers, just as there are diphtheria and typhoid fever carriers, and the scarlet fever is more mischievous than the diphtheria carrier.

Incubation Period.—This may be as short as twelve hours. Usually it is two or three days. For the reasons given above, when the probability of the virus being latent on the fauces was being considered, it is difficult to fix the maximum incubation period.

SYMPTOMATOLOGY.—For the purpose of description cases of scarlet fever may be divided into three main classes—the simple or benign, the anginous, and the malignant. Recently it has become the fashion to apply other names to the two last of these, and to call them "septic" and "toxic" scarlet fever respectively. Personally I think the old names are the best, and I prefer to retain them here. They are purely clinical designations, and as such are extremely useful. They beg no pathological questions as do the words "septic" and "toxic." Moreover, the term "septic" has the disadvantage that it has been applied to scarlet fever in two different senses. Some writers have restricted the word to those cases of scarlet fever in which the throat symptoms dominate the clinical picture, the old "scarlatina anginosa"; others have used it of any case of scarlet fever in which there is a septic complication, such as purulent otitis or rhinitis, irrespective of the prominence of the faucial symptoms.

But while it is convenient to retain the three main classes, it must not be forgotten that there are cases intermediate between the several groups; also that an anginous case may present, in addition to its own, the symptoms which are distinctive of the malignant variety, and that a case, benign for the first two or three days, may gradually acquire anginous characteristics. It is rare, however, for a benign case to take on purely malignant features. Whatever form the disease ultimately assumes, the initial symptoms are fairly constant, although their severity varies considerably.

Onset.—Unlike that of diphtheria, the onset of an attack of scarlet fever is abrupt, often markedly so. The initial symptoms are vomiting, headache, and sore throat. The child looks dull, prefers to lie down, refuses food, but may be thirsty. The temperature will be found to be raised and the pulse-rate accelerated. The skin is hot and the cheeks are flushed. The rash comes out in twenty-four

to forty-eight hours, but may appear later, up to the fifth or sixth day. On the other hand, it may be amongst the earliest signs of the disease, and show itself simultaneously with the other symptoms. It will be advantageous to say a few words about each of these symptoms separately.

Vomiting.—In the average case of the present day the patient vomits but once or twice. Only in severe cases is vomiting repeated. There are, however, mild cases in which the vomiting is so frequent as to produce alarming collapse, from which the child quickly rallies as soon as the vomiting has ceased.

The headache, mostly frontal, is not usually severe. Sore throat is a most variable symptom, being in some cases pronounced, in others slight, or even wanting. It may be the first symptom to appear, or it may come after the headache and vomiting and pyrexia. Its severity depends upon the degree of the faecal inflammation.

The temperature rises quickly, but is seldom very high for the first few hours.

Pulse.—It is a statement common to many writers, that the pulse-rate in scarlet fever increased in frequency out of proportion to the temperature. But this is true only within limits. In any acute infectious disease occurring in a child the pulse-rate may be very frequent if the temperature is over 100° F.; but below that, and especially when the temperature is little above the normal, it is, so far as my experience goes, only in scarlet fever that the rate is higher than the temperature would lead the observer to expect.

In cases of any severity all the symptoms mentioned above are present; but in mild cases, and these form the majority at the present time, one or two may be absent, especially the vomiting and sore throat. Very exceptionally are there initial symptoms other than those mentioned. Occasionally there is slight shivering, but a marked rigor is most unusual. Initial convulsions are very rare. I have only once seen an initial rash, and that was urticarial.

Rash.—The rash of scarlet fever consists of two elements: a diffuse uniform erythema, upon which are set, close together, a number of small red points, which constitute the other element and give the whole rash its punctate appearance. In most cases the punctate character is evident from the beginning, but in not a few the rash is not punctate for the first day, even though it may be intense. The puncta are really minute papules, and in a few cases they are palpable, especially on the outer surfaces of the extremities. The fineness of the punctation depends upon the fineness of the skin. If this be smooth and soft, the puncta are small and cannot be felt; if it be coarse, they are larger and may be palpable. The rash disappears on pressure; but often minute petechiae are present, especially about the root of the neck and on the bends of the elbows. When the rash is very intense the skin may be swollen. Of the whole rash, the proportions which are borne by the uniform erythema and the puncta vary considerably; but if one of the elements is absent it is usually the uniform erythema. It is on the limbs, and especially the legs, that the rash may lack the punctate feature.

The distribution of the rash is as follows: Trunk and limbs, except the palms and soles; neck, to the lower border of the jaw, at the edge of which it merges with the flask on the lace. From the neck, posteriorly, it runs in a narrow band up in front of and behind the ear to the temple, but it is very seldom marked in these regions. It is extremely rare to see it on the face.

In some cases of scarlatina anginosa the rash assumes on the limbs, and especially about the extensor surfaces of the elbows and knees, the form of papules

and macules, somewhat like those of measles. The punctate erythema may also be present in these situations, but usually is not. This morbilliform erythema may in rare cases invade the trunk and face, but on the trunk the punctate erythema is always present with the macules and papules. The morbilliform may appear after the punctate erythema has vanished.

In most cases the rash comes out first about the root of the neck and on the upper part of the chest, but it quickly spreads to the rest of the trunk and limbs. It is not, however, at all uncommon for it to appear all at once and universally over the regions which are usually affected. The duration of the rash is very variable—from a few hours to a week or more. Usually it is about three days. The colour ranges from a faint pink to a vivid scarlet red, like that of a boiled lobster. The more vivid, the more lasting it is. Generally speaking the rash is most intense and enduring in the anginous form of the disease; but it is by no means uncommon to see quite a vivid rash in the mildest of cases.

Not always does the rash occupy the whole of the trunk and limbs. When limited, it is usually confined to the trunk, and even to a portion of it, especially the chest.

While it is in most cases uniformly distributed, its intensity may vary in different localities. But though it may thus have a patchy aspect, the patches of greater or less intensity merge gradually into one another, and never have an abrupt edge. In a few cases the patches are symmetrically disposed. Occasionally there is no rash, but of this something will be said later.

The cheeks may be deeply flushed. In many cases the skin round the nose and mouth is unusually pale. This pallor is not due to contrast with the redness of the cheeks, for it may be observed for some time after the flush has disappeared. It seems to be caused by a local contraction of the arterioles. Occasionally an area of skin to which constant pressure has been applied—as, for instance, by a garter—is left pale and unaffected when the rash comes out. Often, too, when one's finger-nail is briskly drawn across the erythematous skin, a white streak appears after a few seconds, and stays for a minute or two—a condition which is the opposite to that known as the *macule cerisea*. Sometimes in the centre of the white streak there is a longitudinal red one, or, again, the ordinary *macule cerisea* may be produced. Unfortunately, no one of these phenomena is of any use in diagnosis, for they may be set up on almost any erythema, and sometimes when there is no erythema, as in typhoid fever.

When the rash is intense, miliaria may appear in large numbers. The most common seat is the lower part of the abdomen and the upper part of the thighs, but other regions of the body may be affected.

A profuse rash will cause considerable itching. During the eruptive stage the skin is dry, but is hot to the touch in proportion to the height of the patient's temperature. In many cases, just before peeling begins, the skin becomes harsh and loses its natural soft, smooth texture.

Desquamation.—In most cases, at the end of a week or ten days, the superficial layers of the cuticle begin to peel off. This process usually starts about the root of the neck and upper part of the chest or on the pubic region. The peeling takes one of three forms, of which the most common is that known as "pinhole" or "naked." A small white speck or dot appears on the skin. This consists of the loosened horny layer which is on the summit of a papilla, and is in process of being shed in consequence of wrinkling due to changes in the deeper layers of

the skin. Very soon this little area of horny layer breaks off, leaving the portion from which it has separated as a free edge which has the appearance of a pinhole in the cuticle. As the horny layer continues to peel off, the edge of the pinhole breaks away, and so the hole or ring grows larger, finally merging with adjacent rings to form irregular-shaped figures, and the pinhole appearance is lost. This form of desquamation does not occur on the face.

Another common form of peeling is that in which the cuticle separates in dry, branny scales. This is met with especially in infants, and in any case where the rash has been slight.

In both forms little tags of skin at the roots of the nails are commonly to be seen.

In the third variety the cuticle comes off in a thick layer of extensive area. On the fingers it may separate in the shape, as it were, of the fingers of a glove. The skin is left red and somewhat tender. Once or twice I have seen the skin raw and bleeding, but such a condition is very rare in scarlet fever. This form of desquamation is met with only in severe cases with an intense rash.

As a rule the peeling is in proportion to the intensity of the rash and the pyrexia, but not constantly so; for cases without any rash may peel well, though they have usually been cases with considerable fever. On the other hand, occasionally, a case with a fairly well marked rash hardly peels at all. In mild cases with little or no rash there is frequently no peeling. Infants under one year seldom peel much, and often fail to do so entirely.

The process of desquamation occupies four to eight weeks, sometimes longer. The palms and soles are the last places to peel.

In severe cases there may be considerable loss of hair, but the hair grows again when the patient is restored to health. In such cases, also, an atrophic furrow may form across the nails. I have never known the nails to be shed as a result of peeling.

In most cases the tongue quickly becomes covered with a white fur, through which the swollen papillae protrude as red points. The fur, which is composed partly of epithelium, separates, in one case in patches, in another in longitudinal streaks, leaving the tongue raw. The appearance of the raw, red tongue, studded with the enlarged papillae, has been compared to that of a strawberry or a raspberry.

The bowels are usually confined. The urine is diminished in quantity and high-coloured in severe cases, and may also contain a trace of albumin. In mild cases it is often not altered from the normal. The blood shows the leucocytosis which is commonly met with in most of the acute febrile diseases.

FORMS OF SCARLET FEVER.—In an average case of *benign scarlet fever* the symptoms are all of moderate or slight intensity. The tonsils may be enlarged by inflammation; sometimes the mucous membrane of the whole of the fauces is moderately inflamed. A few patches of exudate may appear on the tonsils, or even on the adjacent part of the palate, and slight superficial ulceration may result. The temperature may quickly reach any height up to 103° F., but seldom remains high for any length of time, and slowly falls, during three to seven days, to normal. With the fall of temperature the faucial lesion clears up. In a few rare cases the temperature falls by crisis within thirty-six hours. The child may be restless and light-headed for a night or two.

In the very mildest cases the symptoms may be so trivial as almost to escape observation. In fact, in not a few at the present day, especially amongst the

poorer classes, they do escape, and it is only the occurrence of some complication that compels attention. It is chiefly in cases of this description that there is little or no rash. It should also be especially noted that in many of these cases, even when the rash is fairly pronounced and there is fever, the fauces are absolutely normal, and there is little, sometimes no, sore throat.

In *exanthema* significant the initial symptoms are as a rule more severe than in the mild form, but occasionally a case is met with which, beginning as a mild case, gradually becomes worse, so that at the end of a week all the symptoms of the anginous form are present. The temperature is high, and the patient is restless and delirious. The most characteristic feature is that to which this form owes its name—*exit, the angina*. The throat is extremely painful, so that the child refuses to swallow. The fauces rapidly become much inflamed and swollen, and covered with loose, stringy mucus in such quantity that it is difficult to obtain a view of the mucous membrane.

In the most severe cases the oedema is followed by extensive sloughing, so that one or both tonsils, the uvula, and portions of the palate and its pillars, may disappear. The writer has known nearly the whole of the soft palate with its pillars and the tonsils to slough away. Such cases are usually fatal at an early period, but if the sloughing has not been too deep or extensive the resulting ulceration slowly heals, and recovery may ultimately ensue. In other cases the oedema is not so extreme as to lead to extensive sloughing. Yellowish or greyish patches are to be seen on the tonsils, the edge of the pillars, the velum, and the uvula, and especially in the angle on each side of the base of the latter. These patches may consist of exudation only, but more often they are made up of exudate and slough, and when they separate an unhealthy-looking ulcerated surface remains. In a few cases an oval greyish patch, occasionally two patches, are to be seen on one or both of the anterior pillars opposite the tonsil. After a few days the patch, which is but the expression of a slough, comes away and leaves a perforation. My belief is that this local slough is due to the pressure of a tonsil enlarged by inflammation on an oedematous anterior pillar. If these perforations are large they become permanent; if small, they shrink so as to leave no trace.

Usually the ulceration is confined to the parts which have sloughed, but occasionally, instead of remaining localized and healing, it spreads over the rest of the palate to the pharynx and posterior nares, and even to the larynx, which is invaded in ten to fourteen days from the beginning of the attack. When the sloughing and ulceration of the fauces are deep, profuse and even fatal hæmorrhage may take place from a large (but not necessarily one of the largest) bloodvessel of the neck. There is nearly always a discharge from the nose. At first it is thin and watery, but it soon becomes purulent. The nostrils and upper lip become excoriated. The cervical glands are invariably enlarged and painful. They are moderately hard, and become matted together. Often they suppurate. In severe cases, not only are the glands inflamed, but also the surrounding cellular tissue and the overlying skin. There results a dusky red, brawny swelling, exquisitely painful, stretching from ear to ear beneath the chin and filling up the front of the neck. This is what is known as "bull-neck," and is similar to the condition termed "*angina leucon*." The skin and subcutaneous tissue slough, and an extensive ulcerated surface is exposed, in which the muscles, nerves, and bloodvessels can be seen as it were dissected out. But sometimes even these structures are involved in the gangrenous process, and serious hæmorrhage may take place.

The rash in cases of scarlatina anginosa is not necessarily vivid or enduring. If it has come out early, it is usually gone by the fifth or sixth day, but its appearance may be delayed till the fifth or sixth day. Besides the usual punctate erythema, there is not infrequently present a rash which consists of macules and papules, discrete or confluent. This rash commonly appears after the punctate erythema has faded, but may come out quite early and accompany the solitary scarlatiniform exanthema. This papular and macular erythema is in most cases confined to the extensor surfaces of the limbs, and especially to the joints, but the forehead and cheeks may be affected. Very seldom is the trunk involved. The rash may persist for several days. Occasionally, in addition to the papules and macules, patches of erythema marginatum may be seen on the extremities. These rashes are probably of septic origin. They occur in other septic diseases than scarlet fever. In scarlet fever the septic focus is the faucial ulceration.

In scarlatina anginosa the temperature remains elevated, more or less, till the faucial lesion begins to heal—that is, for three to six weeks. The temperature does not stay at the same level, but jumps irregularly up and down.

At the present day anginous scarlet fever furnishes most of the fatal cases. Death may take place at any time from the end of a week to the sixth or seventh week, and is due to septicæmia or some complication. In cases in which death comes late, emaciation, at times extreme, occurs. The pulse-rate usually remains frequent. Diarrhoea and vomiting may be present. The angles of the mouth are often ulcerated. If recovery ensue, lines radiating from the mouth may be left, and these, combined with the scars resulting from the faucial ulceration, and perhaps also the perforations mentioned above, may give rise to a suspicion of previous syphilis. In rare cases ulceration takes place round the anus. In this variety of scarlet fever complications are common.

In *malignant scarlet fever* the symptoms are severe from the onset. There are frequent vomiting, a much increased pulse-rate, considerable elevation of temperature, dyspnoea, prostration, and muscular tremor and subcutis. In young children the dyspnoea takes the form of hurried, in older children of deep, sighing respiration. Yet little can be found amiss with the lungs. The rash, which is seldom delayed beyond the second day, may or may not be profuse. After a day or two it is usually inclined to be purplish instead of brick-red in colour, and the ears, nose, lips, fingers, and toes, are cyanotic. At first the face presents a yellowish, waxy appearance, but later this is lost in the dusky hue induced by the failure of the heart. The faucial lesion seldom consists of more than a moderate general inflammation, sometimes with some swelling, altogether out of proportion to the severity of the febrile symptoms. At first the pulse is full and bounding, but it loses this character after a day or two, and becomes small, feeble, and racing. The tongue is dry and brown, and sordes collect about the gums and lips. For the first day or so the patient is delirious; then he becomes comatose, and passes his excreta involuntarily. Young and weakly children seldom live beyond three days; older and robust children may live for four or five. In children the event is invariably fatal. Fortunately, at the present time malignant scarlet fever is uncommon. Occasionally the symptoms of this form of the disease are met with in scarlatina anginosa.

One or two other varieties require mention. Now and then a case of scarlet fever, in which there is little faucial inflammation, instead of resolving at the end

if a week or ten days, will continue for three, four, or five weeks. During this period the temperature remains moderately elevated, and the chart resembles that of typhoid fever. The inflammation of the fauces clears up, and there is no complication to account for the continued pyrexia. One is inclined to suspect concurrent typhoid fever, but the writer has seen cases in which the blood-serum has failed to agglutinate both typhoid and paratyphoid bacilli, and in one case which was fatal there were no intestinal lesions. This variety is usually known as scarlet fever of the typhoid form.

Some writers have separated *scarlatina sine eruptione* as a distinct form, but as a matter of fact the rash may be wanting in every variety of the disease. Absence of rash is most common in the mildest form, but it must not be forgotten that it may occur in cases, even severe cases, of scarlatina anginosa, and also in malignant scarlet fever. In the last variety, however, absence of the eruption is most probably due to the death of the patient before the rash has had time to come out.

The so-called surgical scarlet fever is merely scarlet fever occurring in a patient who is the subject of a wound which has been inflicted either purposely by a surgeon or accidentally. The symptoms are just the same as in those cases in which there is no wound, and I have never seen a case which bore out the suggestion that the specific infection gained an entrance through the wound. Such a case, however, has been recorded by Claude Ker. Nor have I met with any evidence that convinced me that children who were the subjects of wounds were more prone to catch scarlet fever than those who were suffering from other illnesses, with the possible exception of burns and scalds. These cases form a somewhat high proportion of the cases of wounds which contract scarlet fever. Moreover, the rash of this disease nearly always comes out within three days of the infliction of the burn, so that there would appear to be some connection between scarlet fever and burns and scalds. It may, however, be only a question of the frequency of the incidence of these accidents and of this disease upon certain ages.

COMPLICATIONS.—Though the table of complications of scarlet fever published in the Medical Supplement to the Annual Reports of the Metropolitan Asylums Board contains some twenty-five items, yet only five of them can be said to be at all common. During the ten years 1900-1909, 153,607 cases of scarlet fever were treated in the hospitals of the Board. Of these, 29,349, or 19.1 per cent., developed otitis; 15,363, or 9.9 per cent., albuminuria not definitely due to nephritis; 12,449, or 8.1 per cent., adenitis, simple or suppurating; 7,509, or 4.9 per cent., nephritis; and 5,599, or 3.6 per cent., rheumatism. Secondary tonsillitis, ulcerative stomatitis, and a relapse, occurred in about 1 or 2 per cent. of the cases.

Otitis.—This complication is found most often in an attack of scarlet fever of the anginous form. Usually it arises in the first week or two, but may appear for the first time during convalescence. The middle ear is most frequently attacked. The first symptom is usually a clear, watery discharge, but this may be preceded by a day or two by earache and moderate pyrexia, or in an infant by crying and restlessness. At first the discharge is not profuse, but it increases in quantity in two or three days, and becomes mucopurulent. Under treatment it may clear up in a few days, but in not a few cases it proves very intractable, and yields only after careful and prolonged attention.

It is hardly necessary in this place to do more than mention the fact that otitis media, especially if protracted, may lead to mastoid abscess, necrosis of some portion of the temporal bone, destruction of the ossicles, facial paralysis, meningitis,

gita, cerebral abscess, sinus thrombosis, or pyæmia. As a rule these grave complications do not arise till several weeks or months have elapsed, but I have known a cerebral abscess to occur within six weeks of the beginning of otitis, and most extensive inflammation of the mastoid and petrous portions of the temporal bone, with paracyst infiltration, within ten days of the onset of a severe attack of scarlet fever. It is seldom that noticeable, permanent deafness results when the middle ear alone is affected. In the rare cases of total loss of hearing there have been reasons for supposing that the internal ear was also involved.

The albuminuria of the Asylums Board's statistics includes every case, from one in which the albuminuria occurs once only to those in which it lasts for several days. In none of them has there been any other sign of nephritis. I am of the opinion, however, that not a few cases which are returned as albuminuria are in reality cases of very slight nephritis. An albuminuria beginning towards the end of the third week, and running on for a week or ten days, should always be regarded with suspicion, for that is particularly the period during which the common form of post-scarlatinal nephritis occurs.

The albuminuria of the febrile stage is seldom of importance. At the same time it should be noted that early albuminuria may be one of the indications—perhaps the only one—of a latent nephritis, as will be shown presently.

Nephritis.—Though inflammation of the kidneys is intimately associated with scarlet fever, not only in the professional, but also in the lay mind, it will be seen from the figures quoted above that it does not frequently follow an attack of the fever. The figures, however, refer only to the last ten years, and during that period scarlet fever has been of exceptional mildness—at any rate in London. As with most of the complications, nephritis is associated more with severe than with slight attacks. Hence its comparative infrequency of late years. But it used to be more frequent, and it has occurred in as many as 17 per cent. of the cases during one year. But while nephritis more often follows severe than mild attacks of scarlet fever, it certainly does at times supervene upon attacks that are so slight as to be regarded as trivial. Not infrequently an attack of scarlet fever occurring in a child of the poorer classes has been brought to light by the onset of nephritis two or three weeks later. No age or sex is exempt from this complication; but it is somewhat more frequent in males and in children of five to ten years of age. It is prone to run in families, so that if several members of a family suffer from scarlet fever, and one undergoes an attack of nephritis, so also will the others.

There are more forms than one of nephritis associated with scarlet fever. A broad division can, in the first place, be drawn between the usual form which comes on two or three weeks after, and that which occurs during the attack of the fever. The latter variety is not common. It occurs in very severe cases of scarlatina anginosa in which there are considerable cervical cellulitis and adenitis. The only signs of the renal complication are, save in rare instances, albuminuria and a diminished excretion, though seldom marked suppression, of urine. There is no hæmatæria. As the child is very seriously ill from the affection of the fauces, the symptoms due to nephritis are masked or are attributed to other causes. These cases are usually fatal, and it is only by an autopsy that the nephritis is discovered. The kidneys are very large, engorged with blood, speckled with small hæmorrhages, especially in the cortex, and show diffuse parenchymatous and interstitial inflammation. Kidneys such as these may be found associated with

septicæmia due to various causes, and the nephritis now described is caused not so much by the scarlet fever as by the septicæmia which depends upon the focal infection.

But it is different with that form of nephritis which is post-scarlatinal. Though not so frequent as is usually supposed, yet it is frequent enough to be set down as the characteristic sequel of scarlet fever. And as it is different in its clinical manifestations, so it would appear to be different in its pathology from the early nephritis just mentioned. It must be attributed to the specific virus of scarlet fever, whatever that may be. Its clinical manifestations, however, are not uniform. Two main forms are met with; the one begins suddenly, the other insidiously. Each arises at about the same period—namely, towards the end of the third or during the fourth week, occasionally considerably earlier or later.

In the cases which begin suddenly the patient may have gone to bed overnight feeling well, and the urine last voided may have been quite normal. He wakes the next morning with a headache and feeling sick, and may actually vomit. He looks pale and has no appetite. The urine passed during the night or the first thing in the morning is found to contain a considerable quantity of blood, so as to have a red or brownish-red hue, and to deposit a chocolate coloured sediment, in which blood-cells and casts can be found on microscopical examination. Thus, within a few hours the patient may have passed from a state of comparative health to one of acute nephritis. I have, indeed, more than once known the onset of the renal trouble to be so abrupt that the very minute at which it occurred could be stated. A patient, whose urine last voided was quite clear, has been seized with a rigor while up and about, and the urine passed next after the attack of shivering has been full of blood. Besides the symptoms already mentioned, there is often a rise of temperature for a few hours.

In hospital practice, when a patient exhibits these symptoms, he is confined to bed and placed upon appropriate diet and treatment. In the vast majority of cases the subsequent course of the nephritis is as follows: The urine is at first somewhat diminished in quantity, and for a week or two the amount of blood and albumin remains stationary. Then gradually the blood diminishes, and finally disappears, and albumin alone can be detected. The quantity of urine increases, and may after three or four weeks be greater than normal, to become normal again later. The albumin meanwhile also lessens, and in its turn disappears, as do the casts. There is not infrequently, even in cases in which no other complication exists, irregular pyrexia, and the temperature chart may resemble that of pyæmia. But in not a few cases there is hardly any elevation of temperature from beginning to end. The patient quickly becomes anæmic, and remains so for some time after the hæmaturia has passed off. If hæmaturia persists he may be extremely anæmic. Dropsy is very rarely to be seen. Occasionally there is a little puffiness about the eyes or oedema of the lumbar region. Usually the bowels are constipated.

In that form of nephritis in which the onset is insidious, the earliest sign is a trace of albumin in the urine, detected at the usual period—the end of the third or beginning of the fourth week. The amount of albumin remains stationary for some days, or increases slightly day by day. Then blood appears, at first in small quantity, to increase later, till the appearance of the urine is exactly that of the form of nephritis, which begins suddenly. At this stage the patient may complain of headache, there may be irregular pyrexia, and, in short, that

condition is reached, after several days, which in the abrupt form is present in a few hours or a day or two. Cases with an insidious onset are less common than those with an acute onset.

Provided that there are no other complications, the duration of an attack of nephritis is about seven weeks. The insidious cases are usually the most protracted. On the other hand, a patient in whom the onset is extremely acute may be well, so far as the nephritis is concerned, in ten days.

It has been already mentioned that albuminuria setting in towards the end of the third week is always to be looked upon with suspicion. Occasionally cases occur in which there is constant though slight albuminuria, lasting for one to three weeks from the end of the third week, without blood, with little if any alteration in the quantity of the urine, with only a rare cast, and with no constitutional symptoms save slight lassitude and loss of appetite. Such cases are to be regarded as instances of nephritis, with a lesion of the kidneys which may be described as gross. Not only is the time of the occurrence of the albuminuria highly suggestive, but I have met with cases in which, while one or two members of a family have suffered from well-marked nephritis, another has shown albuminuria only.

So far I have been writing of nephritis as it is ordinarily met with. A few words must be said concerning certain symptoms which are not very common, though they are to be attributed to interference with the functions of the kidneys. Some patients become drowsy, and even comatose, a state which is usually associated with scantiness of urine, and is to be regarded with apprehension. In others convulsions occur. They begin with twitching of the facial muscles, and the movements may be limited to them. But not infrequently other muscles are successively involved till the patient is in a state of universal clonic spasm. He is then nearly always unconscious. The condition is a most alarming one to witness, but as a rule the patients recover. There is no sign by which it can be predicted that a given case of nephritis will be the subject of convulsions. They most often arise entirely without warning in mild cases in which the daily amount of urine is normal, or even above normal.

It has been said above that dropsy is very rarely seen in hospital cases. The same remark is true of cases treated at home. But when the attack of scarlet fever and the onset of nephritis have been overlooked, the most extensive anasarca may be seen. I have, however, once or twice witnessed severe dropsy in a patient who had been under close medical observation and care from the very beginning.

Occasionally symptoms suggestive of acute meningitis supervene during the course of nephritis. In an infant whose urine it is difficult to collect for examination, meningitis may be erroneously diagnosed, and nephritis overlooked, especially if otitis happens to be present.

About half the cases of nephritis are accompanied by another complication, such as otitis, adenitis, and secondary inflammation of the fauces. Not more than 8 per cent. of renal cases are fatal, and death is usually due to some intercurrent affection, as pneumonia. Very rarely does the disease become chronic; when it does, small, granular, red kidneys are the result.

Of adenitis little need be said. The glands involved are nearly always those of the neck. Usually the inflammation comes on during, or soon after, the acute stage of an attack of sanguineous scarlet fever. But the complication is not infrequently met with in mild cases and during convalescence.

The connection between scarlet fever and arthritis, the so-called scarlatinal rheumatism, has long been known. Now, there are certainly three conditions in which the joints may be involved in scarlet fever. The first, and by far the most common, is the ordinary rheumatism. The second is an acute, suppurative, arthritis which is part of a pyæmia. The third is also a suppurative inflammation, but there is no evidence of general pyæmia. Usually one, but occasionally two, of the large joints, are affected, and recovery ensues. In the first and second varieties there is multiple arthritis, and the pyæmic form is very fatal. All forms of arthritis are prone to occur more often in the anginous variety of scarlet fever than in any other.

Rheumatism usually sets in as the attack of scarlet fever is passing off—that is, at the end of the first or during the second week. It attacks most commonly children over eight years of age. Those who have suffered from rheumatism or rheuma at any time previously are liable to an attack of rheumatism when suffering from scarlet fever. The joints most often involved are those of the hands and wrists, but any other joint may be affected. Rarely is the swelling extensive. The pain and swelling shift from one joint to another. There is moderate pyrexia. An attack lasts from two or three days to a week. A slight relapse is met with now and again. It is seldom that any lesion of the heart is to be detected while the patient remains in hospital (six to eight weeks). But Peyster has advanced reasons for believing that the cardiac lesions may show themselves later, after the patient has left the fever hospital, and therefore that scarlatinal rheumatism is a cause of chronic heart disease in more cases than has usually been supposed.

Secondary rashes are not uncommon. They are most often met with in cases of scarlatina anginosa after the proper rash has disappeared, but occasionally the two are concomitant. The usual form has been described already, but sometimes the form assumed is that of an urticaria or a uniform punctate erythema. In a few instances these rashes are found after mild attacks of scarlet fever.

Children are prone to be affected during convalescence with eczema. The junction of the ear with the head is the most common site. Impetigo about the nose and ears is also not infrequent; and many patients suffer from troublesome eczema.

A slight degree of ulcerative stomatitis will be found in many cases of scarlatina anginosa. But occasionally during convalescence, from not only this but also the milder forms of the disease, the mouth becomes inflamed and ulcerated. If the affection of the mouth supervenes early upon a severe attack of scarlet fever, it may become serious. A number of small ulcers rapidly appear on the buccal mucous membrane and the tongue. By coalescence they become large and serious. The gums are swollen and ulcerated, and the lips, especially the lower, swollen and fissured. There is a profuse flow of saliva, and a highly foetid odour issues from the mouth. There are febrile symptoms, the child wastes, and is troubled with vomiting, and death from septicæmia may result. In rare cases necrosis supervenes on the stomatitis. Ulcerative stomatitis is a communicable disease.

Tonsillitis during convalescence is seldom more than slight, but (inasmuch as *Staphylococcus* occasionally attacks children who are recovering from scarlet fever, especially if the disease happens to be prevalent, it is advisable to examine bacterio-

logically every case of secondary tonsillitis, in order that the graver disease may be detected and treated early.

Another not uncommon secondary throat affection is *retropharyngeal abscess*.

Occasionally slight vaginitis with a whitish discharge occurs during convalescence. It yields readily to simple treatment, and is not due to the gonococcus.

Rhinitis must be specially mentioned: though hardly to be termed a complication, it is of importance because it is believed to be a means of the continuance of infection. While it most often follows severe, it is not infrequently found after mild attacks of scarlet fever. The discharge from the nose is watery and not excessive. It is often accompanied by the formation of small superficial ulcers on, or just within, the anterior nares. It is often resistant to treatment and prone to recur. Occasionally a discharge from the nose appears to be kept up by the presence of adenoids, which an attack of scarlet fever may bring into existence. The diphtheria bacillus is another cause of rhinorrhea.

Amongst complications which are more or less rare may be mentioned acute bronchitis, met with almost solely in children of one or two years of age; *conjunctivitis* and other ocular lesions, including *dacryocystitis*; acute pneumonia, *endo- and pericarditis* (apart from rheumatism), *chorea*, *peritonitis* (occurring during nephritis), and *jaundice*.

Relapses—that is to say, the recurrence of the symptoms of an attack of scarlet fever before the patient has recovered from the primary attack—occur in less than 1 per cent. of the cases. *Second attacks* are occasionally met with.

MORBED ANATOMY.—There is little to be seen in post-mortem examinations of cases of scarlet fever besides what has been observed or diagnosed during life. In cases fatal during the febrile stage the blood coagulates slowly and imperfectly. There is evidence of increased activity of the lymphoid tissue throughout the body, shown by enlargement of many of the lymphatic glands and protrudence of the Peyer's patches and solitary follicles in the intestines. The rash, unless it has been petechial, disappears at death. In malignant cases, beyond engorgement with blood and moderate enlargement of the tonsils, the fauces may not present any deviation from their normal aspect. Microscopical examination of the skin reveals marked dilatation of the blood and lymphatic vessels of the upper part of the corium, especially in the papillae. The bloodvessels are full of leucocytes, which can also be seen without and around the vessels and in the spaces between the cells of the epidermis. There is slight proliferation of the cells of the rete Malpighii and swelling of those above them. Similar changes are to be seen in the mucous membranes of the tongue, mouth, palate, and pharynx.

The kidneys during the eruptive stage are usually normal to the naked eye, though the microscope may show cloudy swelling of the epithelium and slight inflammatory changes. The changes found in the acute nephritis of this stage have been mentioned previously. If death occurs early during an attack of the ordinary post-scarlatinal nephritis, the kidneys are usually found to be enlarged, and to have a coarse, mottled appearance on section. There is cellular infiltration, especially of the cortical portion of the organ. The glomeruli are not necessarily affected, and glomerulitis is not a characteristic of scarlatinal nephritis. The inflammatory lesions may be unequally spread through the kidney.

The heart shows interstitial myocarditis with fatty degeneration of the muscle fibres. The liver cells may also be in a state of fatty degeneration.

PATHOLOGY.—In spite of the large amount of investigation which has been undertaken with the object of finding out the cause, bacterial or protozoic, of scarlet fever, it cannot be stated that a satisfactory result has been attained. *Streptococci* can usually be obtained from the fauces and from suppurative complications, and in many cases from the blood. One of the most frequent of these cocci is the *Streptococcus conglomeratus* of Kurth, the *Streptococcus scarlatinae* of Klein and Mervyn Gordon. This organism derives its name from the fact that when grown in broth it forms conglomerate masses at the bottom of the vessel. But the general opinion is that this organism is not the real cause of the disease. Scarlet fever does not readily, if at all, attack animals. Grünbaum in 1904, and Landsteiner, Levaditi, and Prosek in 1911, seem to have succeeded in a few instances in infecting chimpanzees by swabbing the animals' fauces with exudation obtained from the fauces of, or by subcutaneously injecting defibrinated blood from, cases of scarlet fever. Monkeys of a lower order than the chimpanzee did not appear to be capable of infection even by these methods. Sticker in 1893 gave scarlet fever to himself and a few healthy children by inoculation with blood and oral and faucal mucus from cases of scarlet fever. All these observations, however, throw little light on the cause of the disease. On the other hand, Gabritschewsky claims to have succeeded in preparing a vaccine against scarlet fever by using cultures of a streptococcus obtained from a human being ill of that disease. In some instances vaccination with his virus produced the symptoms of a slight attack of scarlet fever. But further evidence is required before a just judgment can be pronounced on Gabritschewsky's work. Pursuing investigation in another direction, Mallory a few years ago described certain bodies which, he stated, are to be found in the skin in cases of scarlet fever, and suggested that they are protozoa and the long-sought-for cause of the disease. These bodies are of two kinds—the granular and the radiate. In size they vary from 2μ to 7μ , but are occasionally larger. The granular bodies somewhat resemble amoebae in appearance, and contain one or more small vacuoles. The radiate bodies consist of a central spherical body, surrounded, as seen in optical section, by ten to eighteen narrow segments. Occasionally the segments are seen free from the central body and one another. These bodies are found in the lymph vessels of the corium just below the epidermis, between the epithelial cells of the epidermis, and in vacuoles in the cells. Deval observed the bodies in the fluid of vesicles artificially produced on the skin of five cases of scarlet fever.

From all that has just been stated, it is evident that we are far from a knowledge of the true cause of scarlet fever. And certain experiments on complement deviation carried out by Karl and Jessie Koessler seem to indicate that the serum of scarlet fever patients contains specific antibodies for an unknown virus, which would appear to be present especially in the lymph glands of the neck.

DIAGNOSIS.—The diagnosis of scarlet fever may be conveniently considered under three heads: (1) Difficulties met with on account of the sore throat; (2) difficulties on account of the rash; and (3) difficulties connected with designation.

1. *Sore Throat.*—The patient may be seen before the rash has come out, or may be a case of scarlet fever without a rash, and then the differentiation from diphtheria, tonsillitis, and other forms of faucal inflammation is often far from easy. The appearance of the rash is seldom delayed beyond the second day. The early symptoms which are suggestive of scarlet fever are—Sore throat, vomiting, headache, stream-oral pallor, enlargement of the filiform papillae of the tongue, and, if

the temperature is not very high, an increase in the frequency of the pulse-rate out of proportion to the temperature. But not even the combination of all these circumstances is conclusive, unless there is also a definite history of exposure to infection.

For the points which distinguish severe cases of scarlet fever without a rash from diphtheria and from septic inflammation of the fauces, an affection which closely resembles the anginous form of scarlet fever, the reader is referred to the section on Diphtheria. But he should be warned never to omit to examine a case of sore throat for a rash. I have repeatedly known cases of scarlet fever diagnosed as diphtheria simply because a rash has not been looked for.

2. *The Rash*.—Cases in which a punctate erythema is the only evidence of scarlet fever are extremely rare. Such an erythema, present with no other sign, may be caused by an excess of hard, yellow soap and by certain drugs, especially belladonna and quinine, less often by the salicylic compounds, opium and morphia, chloral hydrate, chloralamide, and iodide of potassium. Very occasionally there is pyrexia with the erythema, due to the causes just enumerated; but not infrequently the punctate erythema is accompanied by some other form of erythema, such as papules and macules, and the face may be affected, a fact which negatives scarlet fever. An attack of measles and of chicken-pox may be heralded by a punctate rash, for an account of which the sections on those diseases must be consulted.

Septic rashes are seldom scarlatiniform; the most common variety is a macular rash most profuse on the limbs, especially the extensor surfaces.

A transient erythema, often met with in infants, is a frequent source of error. This rash is not only fleeting, but is patchy and very seldom punctate. The child may have vomited and be feverish, but there is no faucal lesion.

Recurrent scarlatiniform erythema is difficult to distinguish from scarlet fever, especially at the first attack. There may be vomiting, sore throat, and pyrexia. But the rash, though not infrequently punctate, is prone to be patchy and to be confined to the trunk. It usually remains out longer than the rash of scarlet fever. Peeling takes place early, in the form of thin flakes; but in some cases the skin presents a curious network of fine cracks. Sometimes after desquamation patches of red skin, having a greasy, glistening appearance, remain. Cases of this disease are very likely to be set down as second, third, and even fourth attacks of scarlet fever. It is not an infectious disease. The infectious disease most often mistaken for scarlet fever is rubella. Fortunately it is not always prevalent. The diagnosis is discussed in the section dealing with it. In the year 1902 rubella was epidemic in London during the spring and early summer, and the disease headed the list of cases sent to the Asylums Board's Hospitals in mistake for scarlet fever.

3. *Desquamation*.—Errors under this head are due to two misbeliefs; the one is that every case of scarlet fever peels, and the other is that every child found to be peeling must necessarily have recently undergone an attack of scarlet fever. But every case of scarlet fever does not peel, and in the paragraph on this subject it is pointed out under what circumstances peeling is likely to be absent. On the other hand, any erythema or dermatitis may be followed by desquamation more or less pronounced. I have seen cases of measles, rubella, serum rash, and septic rash followed by profuse desquamation, which in some of the cases has been of the "pinhole" variety already described. Hence, not even "pinhole" desquamation is diagnostic. But this form of peeling is much more common after scarlet fever than after any other erythema, and desquamation following a

week or two after an attack of vomiting and sore throat can hardly be due to any other disease than scarlet fever. The occurrence of rheumatism, otitis media, chlostris, or late nephritis, would clinch the diagnosis in a doubtful case.

PROGNOSIS.—In benign cases this is almost invariably favourable. In anginous cases it depends upon the age of the patient and the severity of the faucial lesion. The younger the child the more guarded should be the prognosis. Complications are to be expected most in the class of anginous cases. Malignant scarlet fever is always fatal in children. A continued high temperature (over 103° F.), repeated vomiting, a pulse-rate of over 150 a minute for more than two days, rapid wasting, are all bad signs. Extensive cervical cellulitis and the onset of laryngeal symptoms must always be regarded with anxiety. The majority of cases complicated in these ways are fatal. Cases of scarlatina anginosa, in which, though the temperature has fallen to and remained at normal, yet the pulse-rate is persistently over 120, the faucial lesion is slow to heal, and the patient wastes, are very liable after all to end fatally, sometimes in consequence of the supervention of some complication.

Post-scarlatinal nephritis is seldom fatal. Rarely even does it become chronic. Drowsiness and coma are the most unfavourable symptoms.

Rheumatism is seldom severe in children, and is apt to be overlooked; hence cardiac implication, especially endocardial, may escape observation. It is highly probable that permanent impairment of the heart is more frequent after scarlatinal rheumatism than is generally supposed.

TREATMENT.—The patient should be confined to bed, and placed upon a milk and farinaceous diet, which should be continued till three or four days after the temperature has settled to normal. Then the diet should be changed and gradually increased, till, by the end of three weeks, the patient is taking his usual food. He may be allowed to sit up in blankets or in a dressing-gown when his diet is first changed. At the end of a fortnight or three weeks, if the weather be favourable, he may be permitted to go out of doors.

Pyrexia is best treated by sponging with water at 85° to 90° F. every three or four hours. If this measure has no effect, resort should be had to the wet pack, for which the sheets should be wrung out of water at 60° to 70° F. The packs should be applied for fifteen to thirty minutes every four hours. Warm baths are also useful, especially for older children. Antipyretic drugs, at one time much in vogue, may be tried if these methods fail; of these acetanilide (antifebrin), in 2 to 5 grains doses, is the best. Any one of the measures just mentioned will be found beneficial for delirium, which is seldom met with apart from pyrexia. Chloral hydrate and potassium bromide are also useful for this condition. For frequent vomiting peptonized milk, albumin-water, or rectal feeding, are beneficial.

The treatment of the faucial lesion depends upon its extent. If it be limited, the most efficacious is swabbing every two to four hours with solution of perchloride of mercury 1 in 1,000 to 2,000, or with medicinal iodine, pure or diluted, or with carbolic acid, 1 in 10 to 20. But if the lesion is very extensive or severe, with much secretion, careful flushing of the fauces and nasal passages with warm water, or a saturated solution of boracic acid, or the alkaline solution of which the formula is given in the chapter on diphtheria, is the best treatment. Flushing or irrigation is most safely and easily performed with a douche can (placed 1½ to 2 feet above the child's head), tube and nozzle. The latter should be short, so that its insertion

between the teeth shall not do any damage to the palate. A full syringe may also be used for this purpose. When the inflammation has subsided, one of the local applications mentioned above should be used for the ulcerated surface.

The treatment of the complications of scarlet fever calls for no special notice. For rheumatism, confinement to bed, a milk diet, and salicylate of sodium or aspirin are recommended. Earache, which seldom persists after otitis has commenced, will be relieved by puncture of the membrana tympani if it be bulging, or by the instillation of two or three drops of warm lanolin into the ear. In respect of nephritis the essential point is to detect and treat it early. Hence the urine should be examined for albumin at least every other day, and at the first appearance of albumin or blood the patient should be kept in bed and his diet limited to milk. A saline purge should also be administered. The treatment is the same as is employed in acute nephritis generally. In cases in which albuminuria persists, the patient should not be confined to bed nor restricted to a milk diet for too long a period. Such cases will recover more speedily if allowed up and permitted to have a more generous diet.

Cases with severe facial inflammation and with various inflammatory, and especially suppurative, complications have been treated with anti-streptococcic serum or with strepto- or staphylococcic vaccines, *smack* or autografts. Though I have had under my observation a number of cases treated by these methods, in my experience their action is uncertain.

An essential factor in the successful treatment of a severe case of scarlet fever is the careful devotion of the nurse. Her object should be to prevent the accumulation of the discharges, to keep the cavities of the mouth and nose clean, and to see that the patient takes ample nourishment and obtains sufficient sleep.

In the malignant form an effort should be made to save the patient by the free use of stimulants, especially strychnine by hypodermic injection; but the outlook in these cases is always gloomy.

It is impossible to be quite certain how long any individual case of scarlet fever will remain infectious. But, generally, it may be stated that most are free from infection at the end of six weeks. Many mild and uncomplicated cases are free much earlier. For some years past I have been discharging from hospital nearly all such cases at the end of four weeks, while they were still desquamating. The evidence goes to show that by this time the desquamating cuticle has ceased to be infectious, if it ever has been so. Most authorities are of the opinion that in those patients who become carriers the infection persists in the fauces and nasal passages, and the recurrence of inflammation of these regions, however slight, is prone to revive the infectivity of the patient. Hence cases of persistent rhinorrhoea and relapsing tonsillitis and facial catarrh should always be looked upon with great suspicion from this point of view. Such cases should not be regarded as free from infection till they are well, or till the end of twelve weeks if treatment has failed to cure them. No case of any sort should be discharged straight from the scarlet fever wards, but should be placed for a day or two, before it leaves the hospital in a ward which has not been occupied by infectious patients. Precautions should be taken to prevent the patient from being placed in circumstances which are likely to favour the recurrence of catarrh of the throat and nose. For instance, it is most undesirable to give him a warm bath in cold weather immediately before he is to leave the hospital. It is also

inadvisable to allow a child who has just recovered from an attack of scarlet fever to sleep with children who have not had the disease, for at least a fortnight after being released from isolation.

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CHICKENPOX.

SYNONYMS.—Varicella; Glass pox.

INTRODUCTION.—So closely in some instances does chickenpox resemble smallpox that it is not surprising that the two diseases were not clearly distinguished from one another till a comparatively recent date. Hints of the existence of chickenpox nearly a thousand years ago are to be found in Rhazes. Towards the end of the sixteenth century it seems to have been described by Vidius under the name "crystallæ" or "varicella crystallina." The designation "chickenpox" was first employed by Morton in 1694, and "varicella" by Vogel in 1764. But none of these writers drew any distinction between chickenpox and smallpox. While Pott, in 1780, appears to have been aware of their essential difference, to Heberden must be given the credit of having, in 1767, insisted upon their separation. In spite of his teaching, however, the claims of chickenpox to separate recognition were contested with some force by more than one well-known writer for upwards of another century.

ETIOLOGY. Chickenpox owes any importance it possesses to the readiness with which it is confused with smallpox. It is an extremely mild, even a trivial affection; and fortunately so, for it is very prevalent all over the globe, and highly infectious. Of no disease, except smallpox, is the infection more easily carried by third persons or infected articles. The writer has frequently met with instances of these modes of dissemination. The infection is chiefly spread, however, by children who are actually suffering from the disease. As it is not fatal, there is little statistical evidence of the extent of its prevalence. The most complete records with which I am acquainted are those published by the London County Council in the Annual Report of the Medical Officer for 1902. In consequence of the prevalence of smallpox during that year, chickenpox was made a notifiable disease in London from February 7, 1902, to January 6, 1903. During that period of eleven months 25,009 cases were notified, which gave an attack-rate of 5.8 per 1,000 living. Probably there were between 27,000 and 28,000 cases during the whole year. The disease was especially prevalent from the end of May to the middle of July, and during November and December. The month of least prevalence was September. Of these 25,009 cases 24,132 were under fifteen, and of these 22,748 were under ten. No fewer than 2,451 were under one, a high incidence compared with what obtains in scarlet fever and diphtheria. The figures show that males are rather more liable to attack than females.

Incubation Period.—I have never known this to be shorter than twelve or longer than twenty-two days.

SYMPTOMATOLOGY.—In the vast majority of cases the eruption is the first sign. But occasionally, even in children, for a day or two before this there may be slight pyrexia, and the child seems out of sorts. It is by no means uncommon to find an erythema heralding the proper eruption by a few hours. Usually this erythema is bright red, as often punctate as not, uniformly distributed over, and almost limited to, the trunk, but occasionally overflowing on to the adjacent parts of the limbs. Very rarely does the erythema take the form of urticaria, or a patchy or macular rash. I have never known the initial symptoms in children to be at all comparable to those of smallpox. Once out of about 1,000 cases I have seen an attack begin with a rigor, and two or three times with vomiting. Occasionally the outcrop of the characteristic eruption is preceded by itching.

The Break.—The eruption most often comes out first somewhere on the trunk. In the course of a day or two the face and scalp are affected, and then the limbs. But the eruption may appear first on the face. It has not the orderly invasion of that of smallpox. If the eruption is at all copious, it comes out in successive crops during several days. Very rarely it makes its first appearance on the palate or pharynx. The number of poeks is very variable, from three or four up to hundreds. An average case will have twenty or thirty. Occasionally the eruption invades the mucous membranes, especially of the fauces, rarely the mouth, tongue, epiglottis, larynx, conjunctiva, and prepuce.

The distribution of the eruption is important, because of the aid it affords in the diagnosis from smallpox. The trunk is most affected, especially the back, then the face and scalp, and lastly the extremities, and of these the proximal rather than the distal portions. Though in some cases the eruption is scattered fairly evenly over the limbs, yet even then the distal parts are not more affected than the proximal.

The poeks appear first as small pink spots, two or three lines in diameter, and

Acute on pressure. They quickly become papular, and then vesicular. In some cases the vesicular stage is reached so quickly that no macular or papular stage is seen. The fully developed vesicles are mostly hemispherical and superficial. There is no inflamed base, though not infrequently a slight erythematous areola surrounds many of them. On the thighs, buttocks, and groin, less often on other regions, a few oval vesicles may be seen. The vesicles present a pearly appearance, are not depressed in the center, and collapse entirely on being pricked in one place. The contents consist of slightly opalescent fluid. In the early stage they may look just like drops of water on the skin. After a day or two the vesicles begin to dry up. The process of desiccation starts in the centre, and a yellowish-brown scab forms, which soon falls off, leaving either no trace or a slight pink stain, which disappears after a short time. Usually the whole process from the appearance of the macule to the shedding of the scab occupies ten or twelve days, but it may take as long as three or four weeks.

Not infrequently, before desiccation occurs, some of the vesicles flatten out and increase in diameter till they attain nearly twice their original size. Their edges are then irregular and crusted, and the contents semipureulent. Inflammation may take place beneath them, and the resulting scabs contain the remains of the epithelium involved, and a slightly depressed round scar is left. Interspersed amongst the fully developed vesicles are to be seen a good many abortive ones, small red spots and papules, some of which are surmounted by an imperfect vesicle. Inasmuch as the eruption comes out in crops, pocks in every stage of development may be found in equal numbers over quite a limited area of skin, papules, vesicles, pustules, scabs, and abortive lesions. On a mucous membrane a perfect vesicle is rarely met with, because the upper layers of epithelium, which are being raised by the exudation of serum to form the wall of the vesicle, quickly rupture, and a small whitish or grey spot, surrounded by a red areola, remains.

The eruption is accompanied by itching, which in a few instances is almost intolerable, especially during the scabbing stage; and the pocks will be scratched even to the extent of causing a little bleeding.

The temperature in varicella is very variable. Usually it rises a few degrees while the eruption is coming out, and may reach a height of 100° F. It may rise and fall with every fresh crop of spots, or if the eruption is profuse and comes out rapidly, it may be continuously raised for several days. Occasionally, even when there are many pocks, there is hardly any pyrexia. It is unusual for the temperature to rise above a degree or so when the eruption is very scanty. The pulse follows the temperature. In two or three cases in which the eruption has been very profuse and has come out rapidly, the writer has known the respiration rate to be considerably increased, and has observed signs of slight bronchial catarrh. One child under his care, in whom the eruption was exceptionally profuse, but was otherwise normal, was seriously ill for three or four days. But in most cases the constitutional symptoms are trivial.

Formæ.—Chickenpox seldom departs from the form described above. Still, in two instances the vesicles become confluent, especially on the trunk and limbs. Still more rarely the confluent vesicles pustulate, and there is septic absorption from the pustules with secondary fever, as in smallpox. Another rare form is *varicella bullæ*. In this large blebs form rapidly round, possibly from the vesicles. They may attain a size of several inches in diameter, and then

have an irregular roundish shape, are flat, and contain a slightly turbid fluid. Their rupture reveals a painful excoriated surface. In infants the constitutional symptoms are severe, and the case may terminate fatally. *Hæmorrhagic varicella* is very rare. In it hæmorrhages into the skin and subcutaneous tissues and from various mucous surfaces supervene during the course of an ordinary attack, and death ensues from exhaustion. Sometimes an attack of chickenpox is modified in that while there are a considerable number of papules, no perfect vesicles are formed.

COMPLICATIONS.—These are uncommon. I have three times known symptoms of laryngeal obstruction to arise during the eruptive stage. Two of the patients recovered, one after intubation. The third, an infant of ten months, died from broncho-pneumonia after tracheotomy. At the autopsy no trace of any vesicle could be seen in the larynx. But in another case I saw a vesicle on the epiglottis during life.

In infants difficulty in micturition may be caused by oedema due to pocks upon the prepuce or the inner surfaces of the labæ majora. If pocks occur in the eyelid, especially the conjunctival surface, inflammation of the lid and conjunctiva may supervene, and in a few instances gangrene of the lid has followed.

Secondary infection of the vesicles may lead to very troublesome impetigo, especially on the scalp.

Occasionally the skin round and beneath a pock becomes acutely inflamed; the vesicle enlarges and pustulates, and hæmorrhage may take place into it. After a few days a thick dark brown or black crust, of the size of a sixpenny to a half-crown piece, forms. When the crust separates, it is found that sloughing has taken place to a variable depth beneath it. The skin may not be involved throughout its entire thickness. On the other hand, the destructive process may have gone deeply into the underlying tissues, on the scalp even to the bone. Should several adjacent pocks have undergone these changes, the sloughs become confluent, and a large ulcer results. When discrete, the ulcers usually present a very sharp, well-defined edge. The glands which drain the affected pocks are inflamed; there is considerable fever, and the child is seriously ill, and may succumb, usually to some pulmonary complication, in most instances tuberculous. Pocks on the buttocks, thighs, groins, back, and scalp, are the most frequent seat of this gangrenous inflammation; but the writer has seen it on the palate diagnosed as diphtheria. The term *varicella gangrenosa* has been applied to this complication; for such it is, since the same condition may be found complicating other vesicular eruptions—e.g., herpes and vaccinia, and may occur even without any previous vesicles (*dermatitis gangrenosa infantum*).

Nephritis and arthritis are very rare complications. Erythematous rashes, similar to those which precede the eruption of pocks, may follow it. As with the initial rashes, the most frequent is a scarlatiniform erythema. Relapses are almost unknown, as also are second attacks.

MOOR'S ANATOMY.—As varicella is rarely fatal except from some complication, there is little to be added to what has been described as visible during life. It is stated that in those cases in which extensive gangrene has arisen, tuberculous disease of the lungs is usually present.

The microscope has revealed that the earliest stage in the formation of the vesicle is hyperæmia of the papillary layer of the corium, followed by vacuolar

degeneration of many of the cells of the upper and middle layers of the rete Malpighii, and infiltration of the intercellular spaces with serum. Rupture of the cells leads to the fusion of the vacuoles in them with the distended intercellular spaces to form the vesicle. As this process is less widely spread amongst the upper than amongst the lower layers of cells, the vesicle is fan-shaped, with the handle upwards, and not with the handle downwards as in the smallpox vesicle. Towards the apex of the vesicle can be seen the remains of the ruptured cells, and also of cells which have not undergone degeneration, but have been compressed by the distended cells around them. If suppuration takes place, the lowest layers of the rete Malpighii and even the corium may be involved. Unna states that the nuclei of the cells which degenerate are changed into giant cells. The wall of the vesicle is formed of the keratin layer of the epithelium.

Pathology.—Nothing is known of the essential cause of chickenpox.

Diagnosis.—From *Scarlet Fever*.—The initial punctate erythema often leads to a diagnosis of scarlet fever. But, save in the rare cases in which the eruption begins on the fauces, there is no sore throat, the pulse-rate is not increased out of proportion to the temperature, there is no enlargement of the papillae of the tongue, and no circum-oral pallor. Vomiting is very seldom present. The eruption of pox is seldom delayed more than twenty-four hours.

From Smallpox.—The disease most often confused with chickenpox is smallpox. For the points of distinction between the two, see p. 102. Confluent and haemorrhagic cases of chickenpox are very apt to raise a suspicion of smallpox. Fortunately they are rare. But a close attention to the distribution of the pox will suffice to establish the nature of the case.

From Pemphigus.—Bullous varicella may be mistaken for pemphigus; but here the presence, or absence, as the case may be, of normal vesicles and scabs should be sufficient for diagnosis. Multiple infection of the skin with pus-forming micro-organisms, especially staphylococci, gives rise at times to pustules which are somewhat like those of chickenpox. But these septic pustules do not pass through a vesicular stage. In them the papule is followed at once by the pustule.

Prognosis.—This is almost invariably good. Except in the haemorrhagic form the disease is never fatal unless it attacks a child already much debilitated by some previous illness. Very rarely does one of the complications prove fatal.

Treatment.—Most cases require no treatment, except confinement to bed while the eruption is coming out. All should be isolated from the first sign of illness, usually the rash, till every scab has separated. Should the pox become inflamed, they should be treated with boracic fomentations, followed by some stimulating antiseptic ointment to the ulcers left by the separation of the crusts.

If the eruption is profuse on the scalp, the hair should be cut short. The child should be prevented from scratching the pox by confining its hands in cardboard splints. Itching will be relieved by warm baths or by sponging with tepid water. Starch and zinc powder should be dusted over the pox.

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DIPHTHERIA.

SYNONYM OF THE LARYNGEAL FORM.—Membranous croup.

INTRODUCTION.—There can hardly be any doubt that the disease we now know as diphtheria was amongst those of which Aretæus the Cappadocian wrote in the chapter "On Ulcers about the Tonsils" in his work on the causes and symptoms of acute diseases. Aretæus lived in the second century A.D. Two centuries later Ælius of Amida seems to have been acquainted with the disease; then for twelve centuries history knew it not. But at last, in 1517, Sebastian Franck gave an account of an epidemic throat affection prevalent in Holland and the Rhine Provinces which may well have been diphtheria. During the sixteenth century the disease seems to have been epidemic in Spain and Italy, and is referred to under various names by a succession of medical writers. From Southern Europe it made its way northward during the seventeenth century, and the eighteenth witnessed its transmission to North America. Samuel Bard, of New York, in 1771 singled it out from the group of maladies of the throat with which he was familiar; but his ideas failed to gain general publicity, and it was not till fifty years later that, by the insistence of Bretonneau and his pupil Trousseau, and by the former's invention of a special name for it, the disease was marked for all time. The name coined by Bretonneau was *diphtheritis*, but he subsequently modified it to *diphtheria*.

ÆTIOLOGY.—The infection of diphtheria may be spread either directly from one person to another, as in the act of kissing, or indirectly, as by the common use of eating and drinking utensils, handkerchiefs, and the like. Probably it is very seldom conveyed through even a short space of air. As will be seen later, the organism which is the essential cause of the disease is well known. It would, therefore, at first sight seem to be an easy task to stamp the disease out; but there are certain facts which render that process very difficult. In the first place, the organism gives rise in some persons to morbid states so insignificant or so different from the ordinary form of the disease that their true nature goes unrecognized till after considerable mischief in the way of dissemination has been done. Secondly, the causal organism possesses considerable tenacity of life, and even when a person who has been attacked by the disease has completely recovered he may harbour the organism in his secretions for a long period without any harm to himself, but with disastrous results to others; indeed, in instances which, though they are relatively few, are absolutely considerable in numbers, persons who have been in contact with patients may acquire the organism and harbour it on a healthy mucous surface. These persons—that is, both recovered patients and contacts—are termed "carriers," because they carry the infective agent about them without showing any sign of so doing.

MILK.—Another means of dissemination is by an infected supply of milk. In some instances the infection is derived directly from a person who is the subject of the disease or is a carrier. In others there are reasons for believing that the milk is infected indirectly through the cow. An ulcerated udder may act as a temporary carrier of the causal organism, derived from a human being. In a few instances cats and dogs, and even horses, have been known to suffer from the disease.

This brief statement of the various conditions under which the infection may

and be transmitted from one person to another renders it easily comprehensible how difficult it is to free any given population from the infectious virus.

Distribution.—Diphtheria, though of wide distribution through the world, is chiefly a disease of temperate and cold climates. The epidemic prevalence, as estimated by the recorded deaths, has been shown to vary in most countries inversely as the rainfall, so that when the rainfall is above the average the disease is less prevalent than when it is below it. In this country diphtheria was unusually prevalent during the years 1860 to 1864, and again during 1882 to 1902; but recently the number of deaths has fallen, and the death-rate per million living for 1908 was 116, which is the lowest recorded since 1883, when it was 121. Its seasonal prevalence is during the months September to December. At one time the disease was rife more amongst the rural than the urban population; but since 1870 the incidence has steadily become more urban. There are strong reasons for thinking that a potent factor in this change is the aggregation of children in schools which was begun by the Education Act of that year, and has been augmented by several Acts passed since.

Age.—Diphtheria attacks principally children under ten, and the attack-rate for those under five does not differ very widely from that for those between five and ten. Females suffer in larger numbers than males. In localities where diphtheria is prevalent synchronously with other acute infectious diseases of childhood, it is prone to pick out a victim from amongst the subjects of one or other of them, more especially scarlet fever and measles.

The *Incubation Period* varies from twenty-four hours to four days, and occasionally is longer. But it is a question whether in many cases it is correct to speak of an incubation period at all, because the person affected may have received the causal organism on to one of his mucous membranes a long period before it assumes activity and begins to do him harm. A person, that is to say, may be a carrier for some time before he becomes a patient.

Pathology.—Diphtheria is a disease caused by the action of a certain organism—the diphtheria bacillus. The bacillus gains a lodgment on a mucous membrane, or less commonly on the skin, both of which have possibly been already rendered more or less vulnerable by some previous lesion. In consequence of the activity of the bacillus, an exudation is poured forth which often speedily becomes of a distinctly membranous character. In this exudate the bacillus forms a toxin, which may be absorbed by the lymphatics and give rise to morbid changes in various tissues and organs. In a case of diphtheria, therefore, there may be two sets of symptoms: the local symptoms caused by the presence of the membrane, and the symptoms due to the toxæmia.

The mucous membrane most often attacked is that of the fauces, and next is frequently those of the nasal passages and the larynx and trachea. Other mucous surfaces and the skin are seldom the seat of the disease. The disease may be confined to a special or limited region, but often two or more are simultaneously involved, especially those of the fauces, nose, and larynx.

Faucial diphtheria begins most insidiously. The writer has on more than one occasion accidentally discovered a patch of membrane on the tonsil of a child in whom no other sign of illness was present. So long as the patch of exudation is small, and so long as there is no absorption of toxin, there is hardly any sign of illness sufficiently obvious to attract a parent's attention. But when symptoms

are present they are pyrexia, slight sore throat, and slight enlargement of the cervical glands. Occasionally there is vomiting, but so marked a sign as a rigor is exceptional. On inspection of the fauces, a thin bluish or yellowish-white film is seen, limited usually to a portion of one tonsil. In the course of twelve to twenty-four hours the film will grow in area and thickness to form a definite membrane, which covers the whole tonsil, and encroaches upon the adjoining pillars of the fauces and soft palate. In another day or two the whole arch of the fauces may be enveloped in a thick yellowish membrane with a well-defined edge. In spite of the extent of the exudation, there is on the whole very little pain or swelling of the mucous membrane, but swallowing and speech are somewhat impeded by the mere presence of the thick layer of exudation. As the local disease has been progressing, the temperature may have risen to any point up to 104° F., and the pulse-rate is increased in proportion. But the temperature does not remain elevated for more than a few hours at a time. Sometimes it will fall never to rise again; at others it rises and falls once or twice before the disease begins to abate. The local lesion will usually have attained its greatest extent by the fourth or fifth day, and by that time toxic symptoms will have become apparent. The blood-pressure falls, the pulse is easily compressible, the patient is markedly pale, the glands in the neck are moderately enlarged, and there may be soft, dusky puffiness of the neck without pain or redness. Albumin appears in the urine, which is a little diminished in quantity. The tongue is furred and the bowels confined. The knee-jerks are usually abolished. About the fifth or sixth day the membrane begins to decompose. It becomes of a grey or black hue, sloughs up, and separates. In some cases it comes away en masse, and the mucous membrane, denuded of its epithelium, presents a raw appearance. In others, in which it has been more intimately adherent to the mucous membrane, it disintegrates slowly, and does not completely disappear for several days. Just before, and while it is breaking up, the exudate gives off a characteristic and very offensive odour. With its disappearance it by no means follows that the general condition of the patient improves. The course now taken by the illness depends largely upon the degree of toxæmia. Supposing the case to end fatally, the following is the chain of symptoms: At first the patient vomits occasionally; then the vomiting becomes frequent, so that not only can he keep nothing down, but he vomits and retches quite apart from anything he may take by the mouth. The temperature and pulse-rate fall till the former is two or three degrees below normal, and the latter is usually normal, but in a few instances subnormal. The writer has known the pulse-rate, estimated by auscultation of the heart, to fall below thirty a minute. The heart's action becomes feeble and irregular, the surface of the body is cold, and the face, ears, and finger-tips, dusky. The urine, nearly always albuminous in these cases, gradually lessens in quantity, so that a few ounces only are voided during the twenty-four hours. For a day or two before the fatal termination there may be total suppression. Occasionally the liver is enlarged. The patient does not lose unconsciousness. In some instances he is apathetic and quiet, in others he is restless, in a few he will take an interest in his surroundings, and will play with toys and picture-books, so that to the unpractised eye he appears to be getting better. In some cases death steals upon him so quietly that it is difficult to say exactly when life ceases; at others it seizes him suddenly though painlessly, it may be after a convulsive attack.

I have given a description of an uncomplicated fatal case because in it are

included descriptions of all uncomplicated but not fatal cases. These present all grades of severity, as estimated by the extent of the exudation and the symptoms of toxæmia, from the mildest case, in which there is a small patch of membrane limited to a portion of one tonsil, and disappearing after two or three days without a sign of toxæmia, up to those which are fatal in ten to fourteen days, such as that described above.

While exudation may be seen on the whole or part of a tonsil, or on both tonsils, or on the uvula or palate, or covering all these structures as far forwards as the hard palate, it rarely occupies the tongue, the inside of the cheeks, the hard palate, gums, lips, or floor of the mouth—that is, it very rarely appears on any part that can be reached by the tip of the tongue. The constant movement of the tongue against the structures just mentioned seems to hinder the formation of false membrane upon them and the tongue.

The exudation is not always definitely membranous; it may be pulsatious or cheesy. Again, though it usually begins to form at one spot, and that mostly on a tonsil, its place of origin may be anywhere on the fauces, and occasionally there is more than one focus. It is especially liable to begin in a tonsillar cyst.

In about 5 per cent. of all cases of diphtheria, and especially in faucial diphtheria, hæmorrhages take place into the skin, subcutaneous and other tissues. Small superficial purpuric spots and deeper bruise-like extravasations are seen in the skin, especially of the limbs. Their number is not as a rule large, but I have more than once seen a patient covered with them from head to foot. Bleeding may also occur from one or more mucous surfaces. Blood is constantly oozing from the pharynx and gums and trickling from the nose; the patient vomits and even coughs up blood, and there is melæna. Hæmaturia, however, is very rare. The hæmorrhagic condition is an expression of profound toxæmia, of which such symptoms as albuminuria, scantiness of urine, and cardiac failure, are likewise present. Recoveries from this form of the disease, which is known as hæmorrhagic diphtheria, are few and far between.

Laryngeal diphtheria is usually secondary to, and occurs within a week or ten days of, faucial implication; but it must not be forgotten that diphtheria may begin in the larynx, the *croup d'emblée* of French writers. And if, as is often the case, the fauces are but slightly affected, the first sign of diphtheria may be one of those due to the invasion of the larynx. Of this there are four signs: Inspiratory stridor, apnoea, a frequent harsh cough, and recession of the chest walls. These are signs of obstruction of the larynx, and are not peculiar to the obstruction caused by diphtherial inflammation. To connect these four signs it is very convenient to make use of the old North Country word "croup," which employed in this way possesses neither an ætiological nor a pathological, but only a clinical, significance. Each of the four signs may vary in degree in the same case, and one or another may even be wanting; indeed, it is so very uncommon even to find, at an autopsy in a case of toxic diphtheria, that the larynx is lined with false membrane, even though during life there has been little or no evidence of laryngeal invasion (*croup fruste*, concealed croup; it should be remembered that the word "croup" is almost invariably used by the French to signify laryngeal diphtheria). Usually the first sign that the disease has reached the larynx is the harsh, frequent metallic cough; the last is recession of the thoracic walls, especially at the epigastrium and along the lower ribs. When these symptoms have gone on for some time, the

respiratory embarrassment becomes obvious, and the patient is restless and cyanosed. If he is left undisturbed, his unavailing attempts to obtain air are piteous in the extreme, and he dies of slow suffocation. In most cases the patient undergoes at intervals of varying length attacks of dyspnoea, doubtless due to spasm of the laryngeal muscles. Between the attacks the signs of laryngeal implication remain, but are not distressing. In other cases the signs of laryngeal obstruction slowly become more marked, and there are no spasmodic attacks. Lastly, in a large proportion of the cases the laryngeal symptoms are at no time severe enough to warrant surgical interference, and the patient recovers without it.

Unfortunately, in not a few cases the membranous inflammation spreads down to the trachea, bronchi, and even to the bronchioles; but in the smallest tubes the exudation is less definitely membranous. Hollow white membranous casts of the trachea and bronchi may be coughed up, even in patients upon whom neither incubation nor tracheotomy has been performed. Unless there is accompanying facial exudation of considerable extent, it is seldom that toxic symptoms appear in laryngeal diphtheria. The symptoms of laryngeal, tracheal, and pulmonary diphtheria are almost entirely those due to the blocking of the respiratory passages by the false membrane. About 18 per cent. of cases admitted to hospital are laryngeal.

Nasal diphtheria occurs with or without, and may precede or follow, faucial diphtheria. The symptoms are a discharge from one or both nostrils—at first clear, later mucopurulent, occasionally blood-stained. One or both of the nasal passages may be blocked, and membranous casts of the nasal fossae may be expelled by syringing. Sometimes there is a little bleeding, but serious epistaxis is uncommon. The nostrils are frequently excoriated, and, if the child is not restrained from picking them, ulceration may ensue. When diphtheria is limited to the nasal passages, toxæmia very seldom results. The patient presents only the slightest constitutional symptoms, and often none at all. Hence this form of the disease is often overlooked or not recognized.

The three forms of diphtheria just described are those most frequently met with. Those now to be described are comparatively rare. The most common are the first two—*valvular and ocular diphtheria*.

Valvular, like laryngeal, is more often than not consecutive to faucial diphtheria. But whereas in laryngeal diphtheria the inflammation spreads directly from the fauces to the larynx, the *valva* is inoculated by the child's fingers, which have been infected by contact with the nose or mouth. This form may occur alone, but whether the genital ever precedes the faucial implication is doubtful. As in faucial diphtheria, the onset is insidious, and it is the signs of toxæmia that lead to the discovery of the local lesion. But occasionally pain or discomfort in micturition are fairly early signs. In every case which the writer has seen, the diphtherial exudation was already extensive when it came under his observation. A grey membrane, looking like a slough, lines the inner surface of one or both labia majora, and may spread to the lower lip also. In children it does not go beyond these structures. The affected labium majus is considerably swollen, and is red and moderately firm to the touch. A mucopurulent discharge is usually present. The nearest lymphatic glands are enlarged and slightly painful. The appearance of the part suggests at first sight erysipelas. The temperature is raised, and the signs of toxæmia—namely, albuminuria, vomiting, and cardiac failure—are present. The membrane disappears in much the same way as in faucial diphtheria, and

edema is anything but very superficial ulceration left. Death may occur within a few days from toxemia, or paralysis may supervene.

Oral diphtheria may be primary or secondary. One or both eyes may be attacked, but usually one only. A thin white membrane, which can be readily peeled off, lines the palpebral conjunctiva of one or both lids, and there is a little swelling of the lids and injection of the ocular conjunctiva. But membrane may form on the eye, and beneath it corneal ulceration take place, and even perforation and panophthalmitis. In a few cases there is much more inflammatory reaction than in the cases just mentioned. The eyelids are not only extremely swollen, but inflamed, and the conjunctivitis is intense. The exudation, though membranous, cannot be stripped off. There are a profuse purulent discharge and considerable pain, and the child is feverish. In this form of the disease the danger to the eye is very great. In a case in which there was loss of both eyes, the patient was suffering from scarlet fever as well as diphtheria, so that the ocular lesion was probably of mixed bacterial origin.

Diphtheria of the prepuce the writer has met with only two or three times. In one instance it occurred shortly after circumcision, and was, strictly speaking, wound diphtheria.

Cutaneous diphtheria occurs in several forms. In one a wound or raw surface is the seat of the false membrane. The best example the writer has had under his own observation occurred in a child suffering from facial diphtheria, who was at the same time the subject of extensive weeping eczema of one side of the face and neck. Thin but distinct patches of false membrane formed upon the eczematous skin. In another variety diphtheria of the skin may occur as the result of inoculation. This form especially affects the fingers at the roots of the nails. Obstinate whitlows, which discharge thin pus, arise. The disease is secondary to faucial or nasal diphtheria. Usually the whitlow beads without any damage to the finger, but one or two cases have been recorded in which gangrene, leading to loss of the terminal phalanx, has occurred. I have met with two cases in which there was extensive cutaneous gangrene, due to the local action of the diphtheria bacillus. In one the history given was that the lesion started as a small bleb. In this case repeated vomiting and prolonged albuminuria were present, and the patient died of secondary paralysis. Lastly, of recent years it has been shown that the diphtheria bacillus may be the cause of multiple cutaneous lesions, which are usually vesicles or pustules. If untreated with antitoxin, the eruption may run a very chronic course. Associated with these lesions may be conjunctivitis, rhinitis, or otitis. In one reported case slight diphtheria of the vulva was at one time present.

Other varieties of diphtheria are extremely rare. Cases have been published in which the oesophagus, stomach, external auditory canal, and bladder (in a case of extraversion), have been involved.

In all forms of diphtheria in which definite membrane is present, treatment by antitoxin leads to the separation or resolution of the membrane within a few days. But in former days it was so unusual an event for membrane to re-form time after time, so that the illness went on for several weeks till death or recovery brought it to an end. Such cases were termed *prolonged diphtheria*. They are very rarely met with at the present day. But possibly the diphtheria bacillus may keep up a chronic lesion which is not obviously diphtherial, as in the case of the skin eruptions already mentioned. And, in fact, a case has been reported of ulceration

round the inner canthus of one eye, with chronic inflammation of the pharynx, nose, and vulva, presumably due to the specific organism.

Complications.—There are not many of these which are at all common. The most frequent is paralysis, which is, however, a sequel rather than a complication. The symptoms usually show themselves first during the third or fourth week from the onset of the attack of diphtheria; but they may occur as early as the first week, while the exudation is still present on the fauces, and as late as the seventh. I have, indeed, known them to arise as late as the twelfth. In the interval between the attack of diphtheria and the onset of paralysis the patient may seem to be well. Paralysis is seen more often in children than in adults; in fact, the younger the patient the more likely is he to develop paralysis. About 15 per cent. of all cases of diphtheria admitted into the Eastern Hospital become affected with this complication. If those cases are excluded which are fatal before there has been time for paralysis to supervene, the incidence among the survivors is about 20 per cent. The more toxic the case, the more is paralysis to be expected, so that in most cases of palsy there has been considerable faucial exudation, and albuminuria more or less persistent. Faucial more than any variety of diphtheria is prone to be succeeded by paralysis, because in it there is a greater facility for absorption of the toxin which causes the nerve lesions. Pure laryngeal and tracheal diphtheria are seldom thus complicated, for in them the false membrane is very loosely attached, and there is therefore less opportunity for toxic absorption. Occasionally, however, paralysis, even serious, follows an attack of diphtheria which has been so slight as to be overlooked or neglected. Paralysis may also be a sequel of vulval and cutaneous diphtheria, especially of the gangrenous variety. Since the introduction of the serum treatment the incidence of paralysis has increased, in all probability because under it a larger number of patients survive the acute stage of the disease. For a further account of this complication the reader is referred to Chapter XIV., p. 826.

Cardiac complications are, unfortunately, fairly frequent. They occur not only during the acute stage, but also during convalescence. Dilatation with irregularity of action is the most common. Usually the dilatation takes place slowly, but in a few cases its onset is very sudden, and may then be attended with very severe, even agonising, pain in the cardiac region, and be followed by collapse. The pulse-rate is increased in frequency; indeed, an increase in the pulse-rate may be the only sign of cardiac trouble. It sometimes persists for several weeks after an attack of diphtheria. Syncopal seizures are to be feared for some hour or five weeks after a severe attack. They may prove fatal, and are most likely to occur in patients with cardiac symptoms and in cases of paralysis; but they are by no means unknown in uncomplicated cases which appear to be progressing favourably. Cardiac thrombosis is very uncommon.

The next most frequent complication is *otitis media*, which is met with in about 5 per cent. of all cases. It is seldom serious, and clears up quickly under appropriate treatment. *Cerebral edema*, supervening after the faucial exudation has disappeared, is occasionally seen, as also is *isauritis*. *Lobar pneumonia* may occur in laryngeal cases, especially those which have been submitted to tracheotomy. *Allergic edema*, which is found in about a quarter of the cases, is to be regarded rather as a symptom than a complication. *Nephritis* is rare; usually it arises early. Rare, too, is *peritonitis* arising after the disappearance of the membrane. It is seldom fatal.

In the account of faucial diphtheria given above, it is stated that the formation of the local exudate is unattended by ulceration. But now and then a case is met with in which there is a good deal of faucial edema followed by considerable ulceration. Such cases are accompanied by febrile symptoms, and in them *exanthema erythema*, usually some variety of *erythema multiforme*, may come out on the skin. The rash is prone to be limited to, or be most intense upon, the extremities, especially about the joints. It may be very much like one of the rashes produced by serum. The cases under consideration are, in the opinion of most pathologists, due to the influence of bacteria other than the diphtheria bacillus, especially streptococci, acting in conjunction with the diphtheria bacilli and they are therefore termed "associated" or "mixed" cases of diphtheria.

Eclipses are to be looked for in about 1 per cent. of all cases, and second attacks are by no means uncommon.

MORBID ANATOMY.—The most obvious and sometimes the only gross morbid condition noted after death is the false membrane, concerning the aspect of which there is nothing to add to the account given of the symptoms of the disease. The false membrane is much more firmly adherent to the mucous membrane of the fauces than it is elsewhere; in fact, in the nasal passages, larynx, trachea, and bronchi, it is very loosely attached.

If an autopsy be made in a case which has proved fatal after the exudation has cleared off, superficial necrosis of the mucous membrane may be observed, and there may be slight hemorrhage on or beneath the surface. But as often as not nothing abnormal is to be seen, and it is not possible by mere inspection to postulate the cause of death. The skin and internal organs are often extremely pale. In a few cases it is possible to see with the naked eye that the heart and liver have undergone fatty change.

In hemorrhagic diphtheria, besides the hemorrhages which have been seen during life, it will usually be found that bleeding more or less extensive has taken place in one or more of the following tissues and structures: retropharyngeal; retro-peritoneal, especially around the kidneys; pleura; lungs; stomach; muscles, especially the thoracic and diaphragm; peri- and endocardium, and muscle of heart.

Should any complications have been present, the corresponding post-mortem changes will be found. Even when there has been suppression of urine, the kidneys rarely always present a healthy appearance.

Microscopically the false membrane is found to have a homogeneous granular or fibrillated appearance; but in membrane from the larynx and trachea only the latter condition is present. Scattered through the membrane are to be seen a few leucocytes, and also such bacteria as streptococci. But the diphtheria bacilli are confined to the surface, and are found neither in the deeper parts of the exudate nor in the mucous membrane. When the exudate is closely attached to the mucous membrane, it may be difficult to make out where one begins and the other ends, in consequence of the superficial necrosis of the latter, in which also signs of slight inflammation can be observed.

The heart muscle will almost invariably be found in a state of fatty degeneration in all cases which have been fatal during the acute stage or immediately after it. Occasionally the liver and kidneys are in the same condition. Inflammatory changes in any of these three organs are uncommon. For the changes

which are to be seen in the nerves in paralysis, the section on Diphtheritic Paralysis must be consulted (see p. 826).

PATHOLOGY.—In a previous paragraph it has been pointed out that an attack of diphtheria is favoured by several conditions, such as the age of the patient, the season of the year, and the climate. But even if all these conditions are fulfilled, no person can possibly exhibit the group of symptoms which are the expression of the disease we call "diphtheria," unless a certain micro-organism is present to avail itself of the favourable conditions. On the contrary, one or more of the conditions referred to may be wanting, and yet the presence of the special organism may make itself felt. The indispensable factor in the production of an attack of diphtheria is this organism, which is a bacillus known as the *Bacillus diphtherie* of Klob and Loeffler. It is a non-motile bacillus from 1.5 to 5 μ in length, which grows readily at the temperature of the body in broth and on blood-serum, agar, and gelatine. On serum it forms, after twelve to eighteen hours' incubation at 37° C., small, opaque, white colonies. Its microscopic appearance varies with the medium and age of the culture. The bacilli from a twenty-four hours' growth on blood-serum are a little thicker at one end than the other, which is finely pointed, and some of them are slightly curved. They are arranged in masses in which they are interlaced with one another to form figures like Chinese letters. They can be stained with the aniline dyes, and are not decolorized by Gram's method. Bacilli from young cultures are uniformly stained, but those from old ones take the dye irregularly, so that they present a barred or beaded appearance. In old cultures the bacilli are often markedly club-shaped. They multiply by transverse fission, and not by spore formation.

The bacilli are found on the surface of the exudation—that is to say, they are, to speak strictly, outside the body. Exceptionally they may be found in the glands nearest to the local exudation, or in the lungs and other organs. But it is a very rare occurrence for them to enter the circulation or become generalized throughout the body, and probably such a general invasion takes place only within the last few hours of a fatal case. Usually the bacilli are confined to the local exudation, and in this they produce a toxin the absorption of which gives rise to some of the symptoms and most of the morbid changes which have already been described. The proofs of this view of the pathology of the disease are as follows:

1. If an abrasion be produced on certain mucous membranes of guinea-pigs and rabbits, and the bacilli be rubbed into the abraded surface, a membranous exudate is formed, and the animal may die of cardiac failure or of paralysis.

2. If diphtheria bacilli be injected subcutaneously into guinea-pigs and other animals, local oedema and necrosis are produced, followed, if the animal does not die within a few days, by wasting and paralysis.

3. If the bacillus be grown in broth with a free supply of air, it forms a layer on the surface of the broth. If the broth is then filtered, the filtrate, when injected into most animals, will give rise to local oedema and necrosis, followed by wasting and paralysis and fatty changes in certain of the organs and tissues. This filtrate—i.e., the broth containing the toxic bodies produced by the bacilli—is known as "diphtheria toxin."

4. From the blood and tissues, and especially the spleen, of persons who have died of diphtheria, a complex substance can be extracted, which when injected into animals produces the same results as those mentioned in paragraph 3.

5. A complex poison, similar to that mentioned in the last paragraph, can be extracted from the false membrane of cases of diphtheria.

6. If the toxin produced by the growth of the bacilli in broth be subcutaneously injected in sufficiently large amount into an animal the animal will speedily die. In the injection of some smaller quantity will not cause death, though it may give rise to local and constitutional symptoms. Small quantities may produce no symptoms at all. If toxin be injected every few days, at first in small and afterwards in gradually increasing quantities, it will after a time be found possible to inject such a large amount as would, if injected into a similar control animal for the first time, quickly lead to a fatal result. The animal on whom the series of injections has been practised has become accustomed or immunized to the poison. It will also be found to have become immunized against the bacillus.

7. The blood-serum of an animal thus immunized will, when injected into an animal in which diphtheria has been set up in the manner stated in paragraph 1, stop the spread of the local disease and protect the animal from toxæmia.

8. The serum of an immunized animal will, if mixed in sufficient quantity with diphtheria toxin, mitigate or absolutely prevent, according to the quantity injected, the effects of the toxin. This serum acts as an antidote to the toxin, and is known as "antitoxin." The serum of horses treated in the manner described in paragraph 6, or by some modification of it, is the serum used for therapeutic purposes.

Some authorities held that the influence of the streptococci, which are so often found associated with the diphtheria bacilli, enhances the propensity for doing mischief which the latter organisms possess. With this view the writer cannot agree. In many of the worst cases in his experience pure cultures of the bacillus are obtained; and on the other hand, while cases in which both diphtheria bacilli and streptococci are present may be severe, they are not at all uncommonly quite benign. The suppurative complications, which are not frequent in diphtheria, are usually due to streptococci.

In laryngeal, tracheal and pulmonary diphtheria the cause of death may be exclusively pure and simple. In other forms of the disease it is due to heart failure consequent upon fatty degeneration of the cardiac muscle.

Rhinos.—In any case in which a definite false membrane is present, no matter where the seat of the lesion be, diphtheria should be diagnosed. It is quite true that there are membranous inflammations which are not due to the diphtheria bacillus, but they are extremely rare, and are to be diagnosed only by careful and repeated bacteriological investigation. The observer should, however, make himself quite sure that what he sees is false membrane, and not the surface of a slough. Not infrequently cases of faucial ulceration are sent to hospital as cases of diphtheria because the surface of the ulcer is mistaken for false membrane; and this is a most common error that the opposite one of mistaking membrane for slough, as was done by many writers before the days of Bacteriology. It was lately beautifully emphasized that in pure diphtheria sloughing and ulceration are rare. It may be added that, of non-membranous cases sent to hospital as faucial diphtheria, the vast majority fail to yield the diphtheria bacillus on bacteriological examination, or to exhibit any of the constitutional symptoms of the specific disease.

Faucial Diphtheria.—The following are the infantile diseases in which the faucal lesion may resemble that of diphtheria closely enough to lead to error in

diagnosis! Scarlet fever, ulcerative stomatitis, other less defined forms of ulceration of the fauces, septic inflammation of the fauces, tuberculous ulceration.

Scarlet Fever.—In every case of sore throat inspection of the skin should be made for a rash. But in some cases of scarlet fever, even severe, the rash may be wanting, or so faint and transient as to escape detection. In those instances of the disease, however, which are likely to be mistaken for diphtheria, the inflammation involves the whole extent of the fauces, and is not limited to the immediate neighbourhood of the exudation. Usually much glairy mucus or mucopurulent is poured out, and this at first obscures the exact nature of the lesion. Pain is a prominent symptom. The glands are enlarged, tender, and frequently matted together. The patient is restless, and may be delirious, especially at night. The temperature remains raised for several successive days. The onset is sudden, with vomiting, headache, and sore throat. All these symptoms constitute a group which, though it may be met with in certain forms of septic inflammation of the fauces, is never seen in diphtheria.

Septic Ulceration of the Faoes.—This affection bears a closer resemblance to scarlet fever of the anginous form than to diphtheria. But the onset is not so sudden and pronounced as in scarlet fever. Yellowish exudation, simulating diphtheritic membrane, is present on one or both tonsils, and sometimes on the uvula and soft palate; and when it separates, ulceration more or less deep is observed. The larynx may be involved in the ulcerative process, and the glands and cellular tissue of the neck inflamed. The constitutional symptoms are like those of scarlet fever, but if there is a rash it is blotchy and best marked on the extremities.

In connection with this form of faucial affection must be considered that which is known as "Vincent's angina" (vide also Chapter VI., p. 262). This is relatively not a common disease. Usually a membranous patch forms on one of the tonsils. The patch is really a slough, the separation of which exposes a deep ulcer. A characteristic foetid odour, different from that of diphtheria, is said to be present. The cervical glands are moderately enlarged. In most cases one tonsil only is affected, the constitutional symptoms are slight, and the ulcer heals quickly under local treatment. But soon and then the ulceration will spread to the other tonsil, the adjacent part of the palate, the pharynx, and finally even to the larynx. The lesion is supposed to be caused by the symbiosis of two organisms—a fusiform bacillus and a spirillum. But in my opinion it has by no means been proved that this is really the case, and Vincent himself has stated that the clinical signs of his angina are not sufficiently characteristic to be diagnosed without the aid of bacteriology. Moreover, sometimes neither of the two organisms is present in cases of faucial ulceration which present the lesion described as being more or less distinctive of the disease. "Vincent's angina," therefore, is neither clinically nor bacteriologically a clearly defined disease. Ulceration of the fauces, just like that described under the name of "Vincent's angina," may be produced by different organisms. Clinically all these cases of "septic ulceration of the fauces" constitute a group in which there is considerable diversity in the extent and severity of local lesion, and in which may be found all the intermediate conditions from the almost trivial to the most severe; but any one of these diverse lesions may be caused by any one of several organisms, and it is not at present possible to pick out any special lesion as being caused by any special organism.

From the close clinical resemblance these affections have to diphtheria, they have been described by some authorities under the term "pseudo-diphtheria."

Stomatitis Stomatitis.—In severe cases of this disease whitish patches are seen upon the buccal mucous membrane and tongue. They are raised above the surrounding surface. When the patches are removed, ulcers are revealed beneath them. The gums are often involved, and occasionally also the tonsils and palate, and it is on this account that the diagnosis of diphtheria is made. A quantity of thick, tenacious mucus is secreted, and a very offensive smell is given off. The fact that the patches occur on the buccal mucous membrane, gums, and tongue, is sufficient to negative diphtheria. But it must be remembered that stomatitis of this form occasionally complicates any of the acute infectious diseases, including diphtheria. Usually, however, it sets in late.

In tuberculous ulceration of the fauces the ulcerated surface appears to be covered with coarse, fatty granulations. The exudation is neither profuse nor tenacious. The symptoms of tubercle of other organs are usually present.

Phagedena of the fauces is, fortunately, a rare disease. A small ulcer is formed which has a very red edge. It spreads rapidly by a necrotic process which involves all the tissues as it advances. An extremely offensive and penetrating odour arises from the ulcer, which is singularly free from pain. There is fever, and the patient wastes rapidly. Death results from exhaustion.

Various non-membranous and non-ulcerative form of faucial inflammation are frequently mistaken for diphtheria. Undoubtedly the diphtheria bacillus is sometimes responsible for such diseases. But apart from the results of a bacteriological examination, there is no justification for the diagnosis of diphtheria in the absence of definite membrane, unless some other corroborative evidence is available, such as the presence of an unequivocal case of the disease in another member of the family, or unless the larynx is also involved. A tonsillitis which is succeeded in a few days by laryngitis should always be regarded with extreme suspicion. Tonsillitis of the follicular variety is very rarely, and quincy is never, diphtherial. Erysipelas or cellulitis of the fauces is accompanied by pain, considerable oedema, and severe febrile symptoms, all of which are absent in diphtheria.

In doubtful cases the best course to follow is to make a bacteriological examination, and base the final diagnosis upon its result.

Laryngeal Diphtheria.—If, in a child who is suffering from acute laryngeal obstruction, exudation, even though limited and not definitely membranous, is found upon the fauces, then the diagnosis of diphtheria will in the vast majority of cases be the correct one. Difficulty of diagnosis arises when there is nothing amiss to be seen with the fauces. Examination should in the first place be made for Koplik's spots, because sometimes an attack of measles begins with laryngitis. If the spots are absent the diagnosis of measles is negatived. The next step is to make a digital examination of the cricoid of the larynx and the structures immediately surrounding it, because the obstruction may be caused by a foreign body (though this is rarely mistaken for diphtheria), or by some swelling outside, but pressing upon the larynx. Of these, the one most often confused with diphtheria is a retro-pharyngeal abscess, which presents as a doughy swelling. Should no tumour be present, the laryngeal symptoms are most likely due to the action of the diphtheria bacillus, or one of the less malignant organisms, such as streptococci, the *Moraxella catarrhalis*, the influenza bacillus, or the micro-organisms of whooping-cough, a disease which, like measles, sometimes begins with catarrh of the larynx. As has already been pointed out, diphtheria may commence in the larynx, so that in the cases now under discussion it cannot be excluded unless the bacteriological

examinations are negative. Occasionally the child will cough up a fragment of membrane, and the diagnosis is established at once. Otherwise the diagnosis depends upon the bacteriological findings.

The only other disease of childhood which is mistaken for laryngeal diphtheria is laryngismus stridulus. In this the attacks are paroxysmal, and occur mostly at night and very suddenly. During the intervals the laryngeal symptoms are in abeyance. The victims of this affection are usually tickety children under two years of age.

Fibrous rhinitis can be distinguished from nasal diphtheria only by bacteriological examination (see Chapter VI, p. 263).

Diphtheria of the ear may be mistaken for cellulitis, erysipelas, and otitis media. But in these diseases there is no definite membrane on the inner aspect of the labia majora, as in diphtheria; and in diphtheria there is no ulceration.

The various forms of cutaneous diphtheria mentioned in the account of the clinical symptoms can only be diagnosed by bacteriological examination, except those cases in which membrane forms upon a wound.

PROGNOSIS.—This varies with the age of the patient, the seat of the disease, and the length of time which has elapsed between the onset of the illness and the administration of antitoxin. The younger the patient, the more unfavourable the prognosis. Of the various forms of the disease, the laryngeal is fraught with the most danger. And it has been abundantly proved that, though delay in applying any form of treatment is detrimental to the patient's chance of recovery, yet it is much more so with the antitoxin treatment than with any other. This is because the object of the antitoxin treatment is to neutralize whatever toxin may have been absorbed from the local exudation before it can have produced degenerative changes in the cardiac muscle and other tissues. Once these changes have been set up, no amount of antitoxin can undo the mischief. Toxic cases which go untreated with antitoxin for five days or longer often fail to receive benefit from the treatment, because in them the toxin has had time to do its deadly work. On the other hand, in cases treated on the first day the toxin, even if produced and absorbed so early, is neutralized and prevented from doing harm.

Of individual symptoms the most grave are—Large extent of membrane; intimate adherence of membrane to underlying mucous membrane; persistence of membrane; persistence of albuminuria, especially if in large amount; scantiness of urine; frequent vomiting; infrequent and irregular pulses; enlargement of the liver; a blotchy rash. In cases in which the albuminuria is persistent the danger is not always immediate, but the medical attendant should be on the alert for paralysis and late cardiac troubles. Hemorrhages into and beneath the skin, even though sparse, are of very bad omen. Purely laryngeal and tracheal cases are seldom followed by paralysis. In them the danger lies in the obstruction of the air-passages.

TREATMENT.—There are three cardinal principles which govern the treatment of diphtheria: the administration of the specific antitoxin treatment as soon as possible, the removal of the false membrane, and the assurance of rest.

The nature of antitoxin and the need for its early use have already been explained, but it is necessary to make a few remarks on the dosage and the method of administration. The dose is reckoned in units. A unit is the amount of immune serum which, when mixed with 100 minimal lethal doses

of toxin and injected into a guinea-pig of 250 grammes weight, prevents the animal from dying within four days. Freshly-made antitoxic serum is standardized by comparing it with a standard antitoxin prepared at the Institute for Experimental Therapy at Frankfurt-on-Main. The comparison is carried out by means of a freshly-prepared toxin against which the standard and the new, unstandardized antitoxin are tested.

The dosage of antitoxin is not proportional to the age of the patient, but to the severity of the attack (as estimated by extent of membrane and signs of toxæmia), which is to a large extent dependent upon the length of the illness. When the case is seen early, and there are no signs of toxæmia, 2,000 to 4,000 units should be given. But if, as occasionally happens, there are severe symptoms present on the first or second day, a larger amount, up to 10,000, should be injected. In cases which are very seriously ill when brought under treatment, 18,000 should be given at once. In every case, if there is no improvement next day half the original amount should be given. In any case it is safer to administer too much than too little.

The serum is most conveniently given by subcutaneous injection. The syringe employed should be one which can be easily cleaned and sterilized, and should have a capacity of about 20 c.c. The site most suitable for injection is to one side of the abdomen, a little below the level of the umbilicus. It is true that experiments on animals have proved that the most efficacious method of administration is by intravenous injection; but there are practical objections in its way in respect of children in whom the blood-pressure is low, as is the case in diphtheria. It is extremely difficult, without dissection, to pick out even a large vein.

The injection of serum will in about one-third of the cases give rise to an erythematous rash seven to fourteen days later. The rash is usually either urticaria or a variety of erythema multiforme. There may be moderate febrile symptoms, and in a few cases, 5 per cent., signs of slight arthritis or peri-arthritis of one or more of the larger joints. Exceptionally, more severe reactions are met with, especially in those who have been treated with serum three weeks or longer previously, or who are the subjects of asthma or some kindred disease. These severe reactions either are exaggerations of those commonly seen, or are shown by a rigor with a high temperature, and perhaps collapse, or take the form of an asthma-like attack. A few cases have ended fatally. Hence I do not advocate the use of antitoxin in doubtful cases, except when it is a question of laryngeal diphtheria, which is so prone to be fatal, nor as a prophylactic save under very special circumstances, such as the failure to eradicate diphtheria from a children's institution by other measures.

According to the observations of Anderson, serum kept at ordinary room temperature loses 20 per cent. of its potency every year. If kept at a temperature of 5° C., it loses only 8 per cent. annually. The dried residue of serum, if kept in the dark at 5° C., retains its power unimpaired for at least five years and a half.

If the false membrane is loosely attached, it should be removed with forceps; but if it is adherent to the mucous membrane, and is at the same time extensive, its removal is best accompanied by flushing the affected part every two to four hours with saturated boric solution or the following alkaline lotion: Bicarbonate of sodium, 1 drachm; bicarbonate of sodium, 1 drachm; chlorate of potassium, $\frac{1}{2}$ drachm; chloride of sodium, $\frac{1}{2}$ drachm; compound tincture of lavender, 1 drachm; water, to a pint. When localized to the tonsils, it may be removed by swabbing with a

1 in 2,000 perchloride of mercury solution, or with medicinal iodo diluted with equal parts of water. Irritating applications should be avoided. Far from hindering the extension of the exudation, they may favour it by setting up inflammation of the healthy and unaffected mucous membrane.

In children under three, or in any child who violently resists local treatment, it is advisable to trust to the action of antitoxin alone rather than to employ flushing or swabbing of the fauces, because the struggle between the child and the nurse, repeated every time an attempt is made to carry out the procedure, leads only to exhaustion, which is most detrimental to the child's progress.

Diphtheria of the vulva, and of the eye, should be treated locally by frequent irrigation with boracic solution. A caution must be given against applying antitoxic serum to the eye. I have seen the most disastrous results follow such application. The diphtheria bacillus grows readily in the serum, and the exudation rapidly increases.

When the larynx is involved, the child should be placed in a tent into which steam escapes from a bronchitis kettle. This measure will not infrequently avert operative interference. The need for the latter is shown by restlessness, want of sleep, or marked recession of the walls of the thorax. In hospital practice intubation, in private practice tracheotomy, should be resorted to. Intubation is not advised in private practice, because a medical man must be within a few minutes' reach of the patient. Intubation is also unmitable in cases in which the patient is in the last stage of asphyxia. In a few cases intubation increases the dyspnoea, most probably by the blocking of the lower end of the tube by membrane, and tracheotomy has to be performed. In the cases of extreme urgency above mentioned, it is dangerous to risk such increase of dyspnoea, and tracheotomy should be done at once.

Rest is most essential in diphtheria. When the exudation is extensive, and when there is any sign of toxæmia, the patient should be kept strictly recumbent, not only while the exudation is present, but for several days after its disappearance. Then he may be allowed to sit up in bed, and the effect of this slight exertion on the circulation observed. At the end of another week or fortnight, according to the severity of the attack, he may be allowed to be out of bed and to sit in a chair, and again the effect on the heart should be noted. At the end of another week or more, should no ill effects have arisen, he may be permitted to walk about a little. Any cardiac irregularity or weakness which reveals itself during these periods is an indication for insisting that the patient shall return to bed. The necessity for prolonged rest after an attack of diphtheria of even moderate severity cannot be too strongly urged. The onset of paralysis, too, should always be regarded as an indication for confinement to bed.

All that has been said above as regards bodily rest applies with almost equal force in respect of mental rest. Every form of mental excitement is to be avoided, such as repeated and unexpected visits from friends, and long or animated conversations. As a matter of fact, most children who are suffering from severe attacks seem to prefer to be left alone, and the best course is not to disturb their fortunate apathy, except for absolutely necessary attention.

If there is frequent vomiting, the child should be fed by means of nutrient enemata. The administration of 20 to 30 minims of tincture of belladonna every four hours, and of 20 grains of potassium bromide every twelve hours, by rectum, is worthy of a trial.

In my experience, stimulants are of no avail for the cardiac failure of the toxic stage, nor have I seen any beneficial results from adrenalin, the use of which is recommended by more than one authority. But in the syncopic attacks which may supervene later, stimulants are often extremely valuable in tiding the patient over a few critical moments.

There is nothing special to be said about the diet. So long as the exudate is present or there are febrile or toxic symptoms, the patient should be kept on liquid nourishment. But when the membrane has disappeared, and convalescence is beginning, he may be allowed solid food. The management of the period of convalescence also calls for no special remarks; but attention is again drawn to the importance of avoiding any exercise which throws an undue strain on the heart.

The child who is recovering from diphtheria should be kept isolated for at least three weeks from the disappearance of the exudate, or until two consecutive bacteriological examinations of the region which has been affected have yielded results which are negative so far as the specific organism is concerned.

Unfortunately, in some instances it may be several weeks, or even months, before two successive examinations prove to be negative; the patient, in fact, becomes a chronic carrier. The treatment of such cases has, so far, been very unsatisfactory. The local application of antiseptics is useless, and so is the injection of the specific antitoxin serum. In a few cases the vaccine treatment introduced by Tanner Hewlett appears to have been successful. This vaccine is the endotoxin of the diphtheria bacillus. The initial dose is 2 milligrammes given subcutaneously, followed by doses of 5 milligrammes at intervals of a week to ten days. Recently, spraying the fauces with a twenty-four-hour-old broth culture of *Staphylococcus pyogenes aureus*, with the idea of crowding out the diphtheria bacilli, has been advocated by Skiles and others; but the results of this method are dubious. Should an abnormal, and especially an inflammatory, condition of the fauces or nasal passages be present, every effort should be made to cure it. Thus, hypertrophied tonsils and adenoids should be removed.

In dealing with outbreaks of diphtheria in schools and institutions, the writer is of the opinion that wholesale swabbing of the inmates, with isolation of any carrier that may be detected, whether he has previously suffered from diphtheria or not, is quite unnecessary. The medical officer's attention should be concentrated in ascertaining, first, by inspection, whether any of the inmates are the subjects of slight nasal or faucal inflammation, and, secondly, by bacteriological examination, whether the diphtheria bacillus is present in these cases. If it be, they should be isolated and treated. He is further of opinion that it is quite unnecessary to isolate chronic carriers if they are healthy. Prolonged isolation of a chronic carrier can be justified only when there is chronic inflammation of the fauces or nose, presumably due to the diphtheria bacillus.

(EDITORIAL NOTE.—The Editors feel obliged to point out that the opinion expressed in the foregoing paragraph is opposed to that held by the majority of bacteriologists, school medical officers, and medical officers of health. They consider that the usual view, to which they adhere, may be stated as follows: From time to time in schools and institutions there occur protected epidemics in which fresh outbreaks take place every few weeks or months. These can often be traced to the presence among the inmates of a "carrier" who himself remains perfectly healthy. The most valuable means for the detection of such "carriers" is a systematic bacteriological examination of swabs

from the throats of the inmates without exception. The consequent discovery of the "carrier" who has been the source of infection has often resulted in the complete cessation of the outbreaks. In the treatment of such "carriers" the removal of enlarged tonsils and adenoids is essential, and may be said to be invariably successful in eliminating the organism. Microscopical examination of the tonsils removed from these "carriers" demonstrates the presence of the Klebs-Loeffler bacillus in the deeper tissues.)

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MEASLES.

SYNONYMS.—Morbilli; Rubella.

INTRODUCTION.—Though Rhazes in the tenth century seems to have known that measles was a different disease from smallpox, yet for some hundreds of years after his time it continued to be confounded, not only with smallpox, but with scarlet fever and other eruptive infectious diseases. By the close of the sixteenth century, however, it had been distinguished, in this country at any rate, from smallpox; and in the London bills of mortality, which were begun in 1629, the deaths from the two diseases were recorded separately. Finally, Sydenham, about 1674, established the disease in its proper position.

ETIOLOGY.—Measles is highly infectious from the beginning of the illness to the disappearance of the rash. In the vast majority of cases the infection is spread directly from one child to another; seldom is it conveyed by a third person, and rarely is it harboured in clothing. The virus is certainly contained in the secretion from the mucous membrane of the nose, and perhaps also from the rest of the respiratory tract, and is disseminated by the acts of sneezing and coughing which are so frequent in the earlier stages.

Measles is met with all over the globe. In the United Kingdom it is constantly present in the large towns, and its prevalence assumes epidemic form about every two years. Epidemics vary much in severity. For the first ten years of the present century measles in England and Wales, as judged by the annual death-

rates per million living, was milder than during any decade since the establishment of the registration of deaths, and the rate for 1908, 228, was the lowest on record. The highest was 602 in 1887. During the first four months of 1911 London was swept by an outbreak which, arising in November, 1910, reached its climax in the middle of the following March. During the week ending March 18, 197 deaths were registered in London, a number 134 above the average for that week for the previous five years, and the highest recorded since the introduction of registration.

Seasonal Variation.—Measles is prevalent mostly in the winter and spring, and especially in November and December, and March and April. But occasionally the usual prevalence is postponed to the summer.

Age.—Children form the bulk of the patients who suffer from measles. Wilson, from an analysis of the notifications for the twenty years ending 1902 for the city of Aberdeen, the longest series of notifications, so far as I know, in this country, has shown that it attacks children especially from the second to the seventh year. Inasmuch as measles is very infectious, and one attack affords an almost complete protection against another, most persons suffer from it in childhood, and therefore comparatively few cases are met with in adults. But the experiences derived from epidemics in localities from which the disease has been absent for some generations, as on more than one occasion in the Faroe Islands, demonstrates that, when a whole community is unprotected, persons of all ages are attacked. In Aberdeen it was found that more females than males suffered from the disease.

The common domestic animals seem to be insusceptible to measles, but there are isolated instances of attacks in captive monkeys.

The **Incubation Period** is very variable. Commonly it is from ten to fourteen days, but I have had more than one case in which there were reasons for supposing it to be as short as six, or, on the other hand, as long as twenty-one days. In one case it was apparently twenty-five days.

SYMPTOMATOLOGY.—In a very few cases the patient exhibits slight febrile symptoms immediately on becoming infected, or from time to time during what in an ordinary case is the incubation period. But in the vast majority of instances he remains quite well during this interval, at the end of which appear the signs of attack of the upper respiratory tract, coughing, sneezing, suffusion of the eyes, and a watery running from the nose. Even thus early the conjunctivae may be injected. The child loses his appetite, has a slight headache, and his face is puffy and wears a sleepy look. The temperature usually rises a degree or two, or even more, and the pulse and respiration rates are accelerated. There are, in short, all the symptoms of what is known as a "cold in the head." But there is one sign ("Koplik's spots"), almost invariably present at this stage, which permits of an accurate diagnosis. If the buccal mucous membrane be carefully examined, in a good light, minute whitish specks will be seen on each side in the middle of the cheek. Each speck is the centre of a small round, red area. Occasionally, indeed, especially if the case is seen very early, the small red areas are free from the central speck. The specks are quite superficial, but can only with difficulty be removed by scraping. Their number varies considerably. At first they are scanty, two or three on each side; but they increase in multitude daily till, just before the appearance of the rash, they are present in considerable numbers. Occasionally they crowd the mucous membrane of the mouth, and are seen also on that of the lower lip as it is reflected from the gum. Not infrequently they run into one another to form

larger, though still small, irregular-shaped spots, resembling aphthous ulcers. Though in most cases they appear very early in the disease, occasionally they do not come out till towards the close of the initial stage. With the subsidence of the rash they begin to fade, and in two or three days not one is to be seen. When present in moderate numbers, the spots are often arranged in a horizontal line along the cheek opposite to the junction of the upper and lower molars when the mouth is closed. The whole of the buccal mucous membrane is redder than normal, presents a blotchy appearance, and is dry and shiny. Rarely does it look healthy, though I have seen a few cases in which the specks described above were visible on an otherwise normal mucous membrane. These specks are now known as "Koplik's spots." They are pathognomonic of measles, and their recognition will save the observer from committing errors in diagnosis, which not infrequently arise from the occurrence of two other common symptoms. The first of these is an erythematous initial or prodromal rash, which comes out on the first or second day, and is not the rash proper to the disease. This erythema takes one of three forms: (1) A diffuse redness all over the trunk, but sometimes overflowing on to the limbs. Not uncommonly it is punctate just like the rash of scarlet fever. This is by far the most frequently met with variety of initial erythema. (2) A blotchy rash something like the rash of measles itself, but not so profuse or so conspicuous, though it is more or less universally distributed. (3) Urticaria, also widely spread over the skin. Whether or not these rashes disappear before the proper rash comes out depends upon the length of the prodromal period. They are met with in about 40 per cent. of the cases.

The second of the two symptoms is laryngitis, which is frequent. In what proportion of cases it occurs is doubtful, but cases of early nocturnal laryngitis are often sent to the hospital as diphtheria because of urgent laryngeal obstruction. Usually there are concomitant symptoms which suggest measles to the experienced observer; but all doubt will be removed by inspection of the mouth for Koplik's spots. The laryngeal symptoms nearly always pass off on the appearance of the rash.

One other feature of the prodromal stage of measles remains to be mentioned, which also used to be a trap for the unwary in the days before Koplik pointed out the significance of the spots that go by his name. It has been stated above that an attack of measles commonly begins with symptoms resembling those of an ordinary "cold in the head." Now, in not a few cases, after a day or two, even when there has been a prodromal rash, all these symptoms disappear, the temperature falls to normal, and the patient is seemingly well again. The medical attendant may be thrown off his guard, even if he has suspected measles, and may in consequence be led to relax any precautions he has enjoined with the object of checking the spread of infection. However, after an interval of one or one and a half days, which may extend to two or three, all the symptoms recur and the rash comes out. This remission is quite common in the early stage of measles, though it is more striking in some cases than others. But during the remission the Koplik's spots, which in nearly every case will have been visible before the remission, continue to increase in numbers, and are a certain guide to the nature of the disease. In few cases is there no remission of any description, but, on the other hand, there may be more than one. In the cases in which the remission is wasting the temperature is variable, as are the other symptoms. Seldom is the former continuously raised to the same level.

Towards the end of the initial stage, which varies in length from one to six, or even eight, days, but which is commonly three or four, the catarrhal and febrile symptoms become more pronounced. Often the breathing is hurried and the patient is a little cyanosed, so that his aspect suggests that he is the subject of acute pulmonary inflammation. Yet in most cases auscultation fails to reveal abnormal physical signs of any importance. These come later. One is led to believe, therefore, that at this period the mucous membrane of the bronchioles is in a state of engorgement without secretion, a condition which can be observed in the nose, mouth, and pharynx. The mucous membranes are then dry, and it is not till three or four days later that there is any secretion from them. At this stage there is evidence of intestinal catarrh in the frequent passage of light and loose stools. Certain deviations from the usual course of the pro-eruptive symptoms will be pointed out later.

The Rash.—The characteristic rash shows itself first behind the ears and round the mouth. But in some cases it comes out at once more or less universally, though at the beginning it is sparse. In a few instances the buttocks and upper parts of the thighs are chosen for its first appearance. It consists of small, round, pink macules, visible on pressure. For two or three days the macules increase in number and size, and, becoming confluent, form patches of irregular shape and grouping. Most of the macules can be felt, even when the skin upon which they are situated is stretched; but they are not so palpable as the papules of smallpox. At its height the distribution of the rash is universal, so that the child is smothered from head to foot with it. The confluence may finally be such that large tracts of skin are involved in a uniform erythema, which betrays its origin from discrete lesions by leaving here and there islets of unaffected skin. The colour varies from a pale pink to a crimson red, but a blue tinge may be imparted to it by respiratory or cardiac embarrassment. The rash fades quickly, once it has reached its height, and, when it has been intense, leaves behind a faint brown staining which persists for several days. It is not unusual for the rash to become hemorrhagic. The hemorrhages are sometimes petechial; at others they occupy the whole of many of the macules, so that in confluent cases extensive tracts of skin are involved in this way. Especially does this occur on the face and ears. In a few instances the spots of which the rash is composed are so small that even at an early stage of its course the general aspect closely resembles that of the rash of scarlet fever. But situation of this efflorescence is much more common just at the time the rash is beginning to fade, especially if it has been very confluent but not of a deep colour.

Branny desquamation commonly follows the fading of the rash. Occasionally the flakes of cuticle are of considerable size, and I have several times seen well-marked pinhole desquamation such as is so often present in scarlet fever.

During the evolution of the rash the temperature rises, and reaches its acme, which may be any degree up to 105° F., during the greatest eruptive intensity; but as soon as the rash begins to disappear the temperature falls somewhat abruptly, and regains the normal in two or three days. The catarrhal symptoms also subside with the rash and temperature, and in uncomplicated cases it is at this period that the patient feels most ill. The inflammation of the eyes, pharynx, and nasal passages, leads to photophobia, a frequent, short, suffocative cough, soreness of the throat, and itching of the nose. The mouth and throat are dry, and the child is very thirsty. The irritation of skin and mucous membranes, together with the pyrexia, produce restlessness, loss of sleep, and delirium. As the rash loses

its intensity the mucous membranes regain their moisture, and an excess of secretion from them annoys the patient. The eyelids are glued together by the inspissation of the mucopurulent secretion, and the nostrils and upper lip are excoriated by an acid coryza. The tongue is covered with white fur before the rash appears; the papillæ are enlarged, and may protrude as red points through the fur. Later the fur, which consists chiefly of epithelial debris and dried mucus, separates, and leaves the papillæ showing prominently on the raw red tongue. This is, in fact, the so-called "strawberry tongue," which is as common in measles as in scarlet fever. The urine is diminished and high-coloured, but not often albuminous, and the bowels continue to be relaxed. But with the subsidence of the temperature the various symptoms rapidly abate, and convalescence is quickly established. In a few cases the lymphatic glands are moderately enlarged, especially those of the neck. All may be involved, as in rubella, but not with such frequency as in that disease.

The blood in measles presents certain differences from its condition in most acute infectious diseases. It has been found that during the incubation period there is an increase in the number of leucocytes, which is followed by a decrease during the initial and eruptive stages. The eosinophil cells are either diminished or are normal in number.

Varieties of Measles.—1. The initial period may be very short or absent; that is to say, the rash may come out on the first or second day. On the other hand, the eruption may be deferred till the twelfth day. When the rash is delayed, the symptoms of the initial stage are those usually met with, but they are seldom severe, and vary much in intensity from day to day. The temperature oscillates, and is rarely much elevated for more than a few hours at a time. The face may present a puffy and somewhat blotchy appearance, as if the rash was struggling to come out. In some of these cases Koplik's spots do not appear for several days.

2. The rash may be entirely absent (measilli sine morbillis), or be so scanty and transient as almost to escape observation. When it should be coming out the temperature rises, only to fall quickly to the normal. There can be no doubt that cases in which the rash is absent do occur. They have been known to give rise to others of the ordinary type, and Koplik's spots have been found in them. I have observed one or two cases in which there were reasons for thinking that a light malarial rash present on the first day or two, and followed by no other rash, was really an initial erythema, and that the rash proper never came out—that is to say, the attack aborted.

3. Catarrhal symptoms may be absent (measilli sine catarrho). In past times, doubtless, many cases thus diagnosed were cases of rubella. But there can be no question that in an attack of measles the only symptoms may be slight pyrexia and the rash. These cases are almost invariably benign.

4. I have seen, though only in one or two instances, an attack of measles without any rise of temperature, when the latter has been taken every four hours. Catarrhal symptoms have also been wanting, and the rash has not been intense.

5. Malignant measles, in which the disease kills in two or three days by its intensity alone, is not common; but I am acquainted with two varieties of it. In the one the temperature is high from the beginning, there may be an initial rash, but the proper rash appears on the second or third day. All the other symptoms are exalted, and dyspnoea is an early and prominent feature. The child dies

incoercible and dyspnoic, and perhaps after convulsions, on the third or fourth day. No gross changes are to be found in the lungs after death. In the other form the attack begins much in the ordinary way, but on the second or third day collapse sets in, the temperature falls and is subnormal and the patient succumbs on the third or fourth day. Diarrhoea may be present, but is not severe enough to account for the collapse. A third variety, the hæmorrhagic form of measles (black measles), the writer has never met with. In it bleeding takes place into the skin and subcutaneous tissues and from various mucous membranes, and a fatal termination is the rule. This form should not be confused with those cases of measles mentioned above, in which the rash is hæmorrhagic; these are by no means commonly severe, and are seldom fatal.

Complications.—The most frequent, and, because so often fatal, the most important, complication is lobular pneumonia. It has been mentioned that in most severe cases of measles the respirations are hurried just before and during the eruptive stage, and that often no abnormal physical signs can be detected in the lungs. In many instances, however, at the time of, or even before, the rash, crisp rales are heard all over the lungs, and very quickly signs of lobular consolidation supervene. In such cases the fading of the rash is not accompanied by a fall of temperature or any improvement in the patient's condition. So long as the pneumonia remains, which may be for three or four weeks, the patient continues to be ill. It is unnecessary in this place to enter into details of the symptoms of the lobular pneumonia of measles, since there is nothing peculiar to them. But in infants of the poorer classes the condition invades the lungs with remarkable celerity, and rapidly brings about a fatal issue. Lobular pneumonia is uncommon, as also are pleurisy and empyema.

It has been already stated that the symptoms of laryngitis may be amongst the early symptoms of measles. Occasionally laryngeal symptoms arise during the eruptive stage and later. Their occurrence at these periods should raise a suspicion of diphtheria, which may complicate measles. Early laryngeal symptoms are rarely due to diphtheria.

Inflammation of the larynx is fairly common. The clinical course of the cases in which it is found suggests that it is secondary to acute laryngitis. Tracheitis and bronchitis are also frequent. The capillary variety of the latter is met with in infants, and is very fatal.

The complications which stand next in importance are those of the eye. Slight conjunctivitis may be looked upon as part of an ordinary attack; but occasionally the inflammation is severe, and then the cornea may be invaded and ulceration set up. In the gravest form, not only is the conjunctiva most intensely inflamed and swollen, but the eyelids become infiltrated throughout their whole thickness, and are swollen and brawny, so that they cannot be separated. Under these circumstances the sight may be permanently impaired to a most serious extent, in consequence of secondary corneal opacities, or may be lost as a result of some inflammation of the whole eyeball. In some of these cases the course of the disease is fulminating. This form of conjunctivitis is mistaken for ocular diphtheria, because a greyish exudate, having the appearance of a false membrane, forms on the inner surface of the eyelid. Phlyctenular conjunctivitis may also occur. Chronic ophthalmia is not an infrequent sequel of measles, especially if the case has been neglected.

Somnolence is common. It is seldom that there is not some slight inflammation;

of the mouth, but during the eruptive stage ulceration may take place. Usually a number of small ulcers appear on the buccal mucous membrane, which quickly heal under treatment. But sometimes the ulceration is serpiginous, and involves not only the cheeks, but also the tongue, palate, pharynx, gums, and lips. An excess of thick,ropy mucus is poured out, and a highly offensive odour is given off. There are febrile symptoms, which are prone to be severe, and death may terminate the case after ten days' or a fortnight's illness, during which the patient wastes rapidly. In a few cases cancrum oris supervenes. I have never seen this complication arise in measles except upon a previous ulcerative stomatitis.

Otitis media frequently occurs, and more serious ear complications may follow—namely, mastoid abscess, necrosis of bone, followed by meningitis or pyæmia.

Now and then the cervical lymphatic glands are enlarged by inflammation, but they rarely suppurate. Very occasionally there is cellulitis of the neck.

An attack of measles is not infrequently followed by cutaneous eruptions. The most common consists of a succession of small pustules, which are slow to heal. A troublesome eczema is another sequel. In rare cases vesicles and bullæ, resembling pemphigus, break out on different parts of the skin.

A more formidable sequel is some form of tuberculosis, most commonly of the lungs. This may be either an acute, caseating, lobular pneumonia or a chronic tuberculosis, generalized or confined to the lungs. In one group of cases, what appears to be in the first instance a simple lobular pneumonia does not clear up, and the child dies after a few weeks' illness. An autopsy then reveals the tuberculous nature of the lesion. In another group there is an interval between the subsidence of the attack of measles and the onset of the tuberculous symptoms. During this interval the patient appears to be in fair health, though very seldom is he in good health. The immediate cause of death may be tuberculous meningitis.

Troublesome diarrhoea is not uncommon during the eruptive stage, and it may continue for some time after. The patient wastes and recovery is tedious. Tuberculous ulceration of the bowel may be the cause. Wasting after an attack of measles should always raise a suspicion of tuberculosis. The nervous system is very rarely affected in measles.

Relapses and second attacks are, in my experience, very exceptional. I have met with only one such case. In it the patient underwent two severe attacks, the second of which was three months after the first.

MORDED ANATOMY.—UNNA states that the first stage in the development of the rash is hyperæmia of the cutaneous capillaries. This is followed by spastic contraction of the vessels, œdema, and escape of the colouring matter of the blood. In the deeper parts of the skin the lymph spaces are much distended with serum, but there is very little exudation of leucocytes. The epithelial cells undergo colloid changes. Koplik's spots are due to localized areas of degeneration of the epithelium. In one case under my observation, which was fatal while the spots were still visible, it was impossible to pick them out in the microscopic sections, and the whole mucous membrane was in a state of inflammation. In this case Koplik's spots could be seen after death in the mouth, and spots exactly like them were visible also on the mucous membrane of the pharynx, larynx, and trachea, which were all acutely inflamed.

For the rest, there are no marked changes in any of the organs that can be said to be special to measles, unless it be interstitial emphysema, which is commonly

found in cases where there is lobular pneumonia. Microscopically the pneumonia does not present any distinguishing features.

The exact relationship between an attack of measles and the tuberculosis which occasionally follows has not been determined. Whether the tuberculous focus is present in the bronchial or other glands previously, and is stirred into activity by the measles, or whether the lesion, more especially of the lungs, caused by the measles, damages the tissues to such a degree as to afford an easy and unopposed entrance to the tuberculous virus during or after the attack, is not known.

Another interesting question is that which is concerned with the cause of the ordinary lobular pneumonia which is so frequent. By some it is believed to be a secondary infection by strepto- and other cocci. But I am inclined to the opinion (that an inflammation is in the first instance set up by the true cause of measles, at present unknown, and that only in some cases is this pneumonia aggravated by secondary invasion of the organisms mentioned).

The real cause of measles is unknown. By analogy we suppose that it is a living micro-organism. That it is contained in the nasal secretions and the blood we know from certain experiments that have been made on human beings and monkeys. Bacteria subcutaneously injected blood taken from a case of measles, mixed with peptone broth and incubated for a few hours, into two men who had never had measles, with the result that they both developed the disease. Lately Goldberger and Anderson have produced measles in monkeys by injecting into their veins not only a mixture of the buccal, pharyngeal, and nasal secretion, but also blood from cases of human measles. The blood of an animal which developed measles as the result of one of these injections was injected into two other monkeys, and one of them developed measles six days later. The virus of measles appears to be a 'filter-passer.'

DIAGNOSIS.—The presence of Koplik's spots from the very beginning of the disease up to the height of the rash will in most instances enable a positive diagnosis to be made with ease. If, in a case possessing a scarlatiniform rash or symptoms of laryngitis, these spots are to be seen, the patient is suffering from measles and not from scarlet fever or diphtheria. It is true that in a few exceptional cases measles and scarlet fever, or measles and diphtheria, may be present at the same time in the same patient. In such cases measles is diagnosed by the Koplik's spots, scarlet fever by the condition of the fauces, and diphtheria by the presence of false membrane and the diphtheria bacillus. It would be impossible to diagnose scarlet fever coexisting with measles (each in the eruptive stage) in a case in which the scarlet fever was of a very benign character with no marked facial lesion.

If the observer is acquainted with Koplik's spots, he can hardly make a mistake over them; but small air bubbles, minute particles of food and of inspissated mucus, and little ulcers, may be mistaken for the spots. All of these, however, except the ulcers, can easily be removed, whereas Koplik's spots cannot; and the smallest ulcer is bigger than the largest spot. Even if many of the spots have become confluent, there will always be some which have remained discrete and of the usual size. Lastly, certain yellow spots, which are occasionally to be found in persons of all ages who are apparently healthy, may be mistaken for Koplik's spots. They are of a yellow colour, and are to be seen low down on the buccal mucous membrane opposite the posterior molar teeth. Some ten or a dozen are usually present, and the posterior ones are larger than Koplik's spots, though the anterior may be of the same size as the latter. Their colour differs

from that of the spots. They appear to be enlarged mucous glands. In the case of a young woman who was under my observation they appeared quite suddenly.

In the eruptive stage measles may be mistaken for rubella, smallpox, typhus, and septic and drug rashes. For the distinction between it and the two last of these diseases the sections which deal with them should be consulted. There is some resemblance between the rashes of typhus and measles; but in few cases of typhus does the rash invade the face, which is never free in measles. The rash of typhus is not palpable, and there are neither Koplik's spots nor early catarrh of the respiratory tract in typhus. The subcuticular mottling and the almost invariable occurrence of minute hemorrhages in the macules of which the bulk of the rash is composed, further distinguish typhus.

Septic rashes are often morbilliform; but their distribution over the skin differs from that of the rash of measles in that they are usually confined to the extremities, and especially to their extensor aspects. The other symptoms of measles are wanting, as is also the case with the drug rashes, including those due to various serums. A rash produced by a soap-and-water eczema is liable to be mistaken for measles. But eczema rashes are rarely accompanied by other symptoms (*see* p. 1153).

PROGNOSIS.—Generally speaking, this depends on the character of the epidemic and the social condition and age of the patient. Epidemics vary very widely in virulence. Thus, in Aberdeen, during the ten years 1883 to 1902, the case-mortality varied from 0 to 25 per cent. The disease is much more fatal amongst the poor than the well-to-do. The case-mortality is highest in children under three, and is especially high in those under one. In individual cases lobular pneumonia and laryngitis are very unfavorable complications, and the persistence of lung signs after the rash has faded is of grave significance. Convulsions are always to be regarded with apprehension; less grave, but still serious, is continued diarrhoea. Occasionally measles is complicated by the presence of some other infectious disease. The most fatal combination is that of measles and diphtheria, because the latter disease is so prone to invade the already inflamed respiratory tract.

TREATMENT.—In hospital practice the large majority of the cases which are complicated with lobular pneumonia are already suffering from that affection when they are admitted to hospital. This fact, combined with the knowledge that they are seldom sent to hospital before the rash comes out, suggests that the pulmonary complications are very largely due to insufficient attention to the patient during the initial stage. There are two essential points to be insisted upon from the very beginning of the illness. They are that the patient shall be supplied with plenty of fresh air, and at the same time kept sufficiently warm. In a well-arranged hospital there should be no difficulty in securing these requirements, nor should there be in a decent house which is not overcrowded. It is a good plan to allot two rooms to the patient—one for day and the other for night use—so that air can be thoroughly aired while the other is occupied. In other respects the treatment should be that which is usual for most acute febrile conditions. The diet should be restricted to milk, jellies, custard, and the like; and pyrexia should be treated by sponging with tepid water. It rarely requires more energetic treatment before the eruptive stage. If laryngitis is present, relief will in most instances be afforded by keeping the air round the child moist with steam from a bronchitis kettle. In a large ward it will usually be necessary to place the patient in a tent in order to secure the requisite moisture. Operative interference should be post-

pend as long as possible, because, with the advent of the rash, there is every prospect of the subsidence of the laryngeal symptoms. But if the laryngeal obstruction is such as to require surgical relief, intubation should be performed in hospital, and tracheotomy in private practice.

Should bronchitis or lobular pneumonia set in, the child should be wrapped in a cotton-wool jacket, and, if the cough is harsh and dry, placed in a steam tent, as has been recommended for laryngitis. Nourishment should be given frequently in small quantities, so that the stomach is not overloaded, to the embarrassment of respiration. If there is the least appearance of cyanosis, oxygen should be administered frequently. Braady, 20 to 30 minims every two to four hours, is certainly beneficial in pulmonary cases. When the cough becomes looser and bronchial secretion is re-established, the use of steam should be discontinued, for it is then very likely to increase the secretion to an undesirable degree.

Occasionally a persistently high temperature, due in a few cases to the measles itself, in most to pulmonary complications, will require treatment. Sponging with tepid water should be tried in the first place; if that fails, cold water should be used. In several cases the application of ice-poultices to the chest has been very beneficial. Cases treated in this way require careful watching for collapse supervene.

Clasie Ker, of the City Hospital, Edinburgh, speaks highly of the out-of-door treatment during the daytime, in nearly all weathers, of measles complicated with pericnemitis. I have had no opportunity of trying it, as the situation and structural arrangement of the Eastern Hospital are not suited to the purpose. It is believed by some authorities that lobular pneumonia will spread from one measles patient to another in a ward, and they therefore recommend the removal of cases affected with that complication to special rooms or wards. But though particular observations on this point were made in two large wards of twenty-one beds each at the Eastern Hospital during the early spring of 1911, the evidence that the dissemination suggested took place was by no means patent. Of 229 consecutive cases, 43, or 19.5 per cent., were the subjects of lung complications on admission. Of the 177 which were uncomplicated in this way on admission, 13, or 6.3 per cent., developed pneumonia afterwards. As some of these would have developed pneumonia under any circumstances, it is clear that very few, if any, took it from those cases which were already suffering from it on admission. Careful attention to the eyes will usually avert serious complications. At the least appearance of conjunctivitis the eyes should be shielded from the light by a screen around the bed or a blind lowered over the window. Frequent irrigation with boracic lotion should be employed, and, should the inflammation show signs of spreading to deeper structures, the pupil should be dilated at once with atropine, and the case treated on ordinary principles.

Slight diarrhoea during the eruptive stage requires no special treatment. Should it persist after the rash has vanished, intestinal astringents, with small doses of opium, should be given. If there is reason to suppose from the symptoms that the diarrhoea is due to inflammation of the large intestine, the bowel should be washed out two or three times a day with warm water. If the diarrhoea produces collapse, hypodermic subcutaneous injection with warm saline solution is worthy of a trial.

An attack of convulsions should be treated by a hot bath, followed by the administration of oxygen. This complication occurs mostly in pulmonary cases.

Of the treatment of the remaining complications of measles, there is nothing special to be said.

An uncomplicated case of measles is certainly free from infection in three weeks from the appearance of the rash. In such cases the patients may be allowed out of bed a week or so after the temperature has become normal, and out of doors, in suitable weather, a few days later. Probably most complicated cases are free from infection in much the same time as the simple cases; but those complicated with pneumonia are rarely fit to be about before the end of five or six weeks, and it is safest not to regard them as free from infection till the expiration of that period.

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RUBELLA.

SYNONYMS.—German measles; Rosteln; Roseola; Rubenla; Epidemic rose-rash.

INTRODUCTION.—This disease owes one of its names to the fact that it was first differentiated from scarlet fever and measles by the German writers De Bergen in 1752 and Orlov in 1758. Observed but misunderstood by Wilan in 1808, it was recognized in London by Maiten in 1815; but the general acceptance of its existence was retarded, partly by Hildebrand's absurd supposition in 1828, that it was a hybrid between scarlet fever and measles, partly by a want of clearness as to its clinical aspects on the part of some of its delineators, partly by its varied nomenclature, and, in no small degree, by its own duplicity. At the present time there is hardly a writer with special experience who denies its existence, and its least misapplied name, "rubella," proposed in 1866 by Yeale, is the one most in favour.

ÆTIOLOGY.—Rubella is an epidemic disease. It seems to occur in limited rather than in widespread outbreaks, and is to be met with all over Europe, in America, Egypt, India, Australia, and New Zealand. In the United Kingdom it is more prevalent in the spring and early summer than at any other season, but varies much in its prevalence from year to year. In the absence of large numbers of recorded cases it is impossible to give the age incidence with accuracy, but such figures as are available go to show that it attacks proportionately fewer children under five and more persons over five than does measles, and males and females in about equal numbers.

INFECTIVITY.—The disease is feebly infectious. I have on several occasions treated two or three cases in a ward with patients who were suffering from scarlet fever or diphtheria, and none of the latter have contracted rubella. I have no certain evidence of the spread of the disease by any other means than directly by the patient himself, either by contact or for a few feet through the air.

The shortest incubation period I have met with has been ten days; the longest, seventeen. Usually it is longer than that of measles.

SYMPTOMATOLOGY.—Children seldom show symptoms before the rash; but in a few cases I have observed some enlargement of the glands, especially of the neck, a day or two—in one case five days—before, with or without pyrexia. Occasionally there is vomiting, or sore throat, or coryza; but in the large majority of the cases the rash is the first noticeable sign of the disease.

The Rash.—In the most common form of rubella it begins on the face, as small, discrete, pale pink spots, definite on pressure, and hardly palpable. The skin of the scalp, forehead, and face is involved, right up to the lips. By the next day the rash will have disappeared from the face, leaving at the most a blush, and will have come out on the trunk and upper extremities. Towards the end of the second or on the third day the lower limbs will be invaded. On the trunk and extremities the rash alters its appearance within twenty-four hours, to assume the form of a diffuse erythema, which may be beset with puncta and even with petechiae, as in scarlet fever. Consequently, as by this time the face is quite free, the resemblance to mild scarlet fever is very close. Occasionally the rash comes out simultaneously all over the face and trunk, and it may be also the limbs. Its duration is from twenty-four to seventy-two hours.

In rare instances the rash is, or seems to be, a diffuse scarlatiniform erythema from the very commencement, and the face may then appear to be hardly at all involved; but it is quite possible that in these cases there has been a spotty phase, overlooked because fleeting. This is the true scarlatiniform variety of the disease; but not a few writers have included under this term not only this, but also the ordinary form, in which the rash is spotty at first. In the morbilliform variety the small spots enlarge and coalesce to form macules like those of ordinary measles; but even then, on the second or third day, there is often an attempt at the uniform punctate erythema of the scarlatiniform variety.

Usually there is no peeling. When it does occur, it is fine and branny. Both profuse and "pinhole" desquamation are rare in children. Peeling seldom persists for more than a few days.

Is rather more than three-fourths of the cases the superficial lymphatic glands are moderately enlarged. The mastoid and posterior cervical are the most constantly affected, but all may be. Occasionally the mastoid and cervical glands escape, while others are involved. Sometimes the enlargement is very noticeable. The glands are tender but firm to the touch. They are never matted together. I have never known them to suppurate. The enlargement subsides within three weeks.

The conjunctivæ are injected in about two-thirds of the cases, and there are itching and smarting of the eyes. There may be itching of the nasal passages, with sneezing and a watery discharge. In some cases there is catarrh of the pharynx, and occasionally tonsillitis. The buccal mucous membrane is quite normal. I have not seen the spotty appearance mentioned by Ker. The tongue is slightly furred, and constipation is the rule.

With the appearance of the rash the temperature may rise; "may," because, in about half the cases, there is no pyrexia. It seldom rises above 102° to 103° F., and quickly falls. The pulse-rate is a little accelerated if the temperature goes up, but not otherwise, and the respiration-rate is normal. The constitutional symptoms are very slight; in many cases the patient does not seem to be in the

smallest degree out of sorts except for the rash. In a few instances the temperature may rise to 105° or 106° F., but the patient seems to be little the worse for the high temperature.

According to Dukos, injection of the conjunctivæ ("pink eyes"), or enlargement of the lymphatic glands, with slight feverishness, may be the only sign of the disease.

Complications are rare. Slight and transient albuminuria and otorrhœa have been observed. I once saw a child with subacute arthritis and cardiac dilatation following rubella. He died rather suddenly, and his was the only fatal case I have seen. In four of my patients a distinct relapse occurred within a few days of the first attack. Second attacks seem to be almost unknown.

PATHOLOGY.—We are in complete ignorance of the essential cause of this disease.

DIAGNOSIS.—The diseases most frequently confused with rubella are measles, scarlet fever, drug rashes, and the accidental or secondary rashes met with under various conditions, especially in the acute infectious diseases. It is only very mild cases of measles or scarlet fever that are likely to be mistaken for rubella. The presence of Koplik's spots are pathognomonic of measles, so that search should at once be made for them. Should they be absent—and they quickly fade directly the rash comes out—it is very seldom in measles that the buccal mucous membrane looks normal, as it does in rubella; it is dry, blotchy, and glazed. Rubella can be excluded if the respirations are hurried, or if there is evidence of catarrh of the larynx or lungs. In the vast majority of cases of measles there is an initial illness of two or three days' duration before the rash comes out. From scarlet fever the diagnosis is easy if the case is seen early enough, while the rash is still spotty, and especially if it is visible on the face; but if the case is seen for the first time on the second or third day, it may be impossible to make the diagnosis on the spot. In fact, not a few cases of rubella in a late stage are sent into the scarlet fever wards as cases of scarlet fever, even by those who have had considerable experience of both diseases. When the disease is epidemic, much help may be derived from careful attention to incubation periods in groups of cases, and a close clinical study of three or four cases will often, by combining scattered features, reveal a picture of the disease which is not visible in the cases considered singly.

In such drug and accidental rashes as are likely to be mistaken for rubella, there are larger papules and macules than are usually met with in the infectious disease.

PROGNOSIS.—This is almost invariably good; I have only once seen a fatal case. Cochrane states that there was not a single death in 1,523 consecutive cases at the London Fever Hospital.

TREATMENT.—The patient should be isolated and confined to bed while the rash is out and while he feels at all ill. It is rarely necessary to take further measures. I never detain a patient in isolation for longer than a week.

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THE "FOURTH DISEASE."

In 1880 Filatow, of Moscow, put forward the view that the scarlatiniform variety of rubella was a distinct disease. Dukes independently arrived at the same conclusion in 1900, and tentatively called the affection the "fourth disease," scarlet fever, measles, and rubella being the other three. There has been much discussion on the subject since Dukes's paper was published, and now the opinion commonly held is that the case for a fourth disease, as described by Dukes, has not been made out. Dukes's own account of the disease, and especially of the rash, is far from being clear, and some of the writers who have supported him have confused the ordinary form of rubella with the scarlatiniform variety, strictly so called, if that disease.

But there does appear to be another infectious disease which is not scarlet fever, nor measles, nor rubella. In 1886 Tschanner, of Gratz, described as a local variety of rubella what Escherich, in 1897, showed to be a different malady. It is known as *Erythema infectiosum* or *Megalerythema epidemicum*. So far it has been met with in epidemic form only on the Continent and in the United States. Its seasonal prevalence is the spring and summer, and children from four to twelve are most subject to attack. In most instances the only symptom of the disease is the rash, which comes out after an incubation period of six to fourteen days. It starts on the cheeks, which become swollen and red. The skin round the mouth and nose is not involved, and is separated from the affected area by a sharp line along the naso-labial fold, posteriorly the erythematous skin has an irregular edge. After a day or two the limbs are involved, but upon them the rash assumes at first the form of largish, round, erythematous patches, which, as they spread to the periphery, fade in the centre. The coalescence of the advancing edges of the patches gives rise to irregularly disposed, festooned figures. In short, the rash consists of that variety of erythema multiforme which is known as "erythema marginatum." The rash begins at the proximal and gradually creeps over the distal portions of the limbs, affecting especially the extensor surfaces. It remains so for a week or ten days. The trunk is seldom involved. Usually there are no other symptoms of illness, but occasionally at the beginning of the attack the patient feels a little out of sorts and complains of a slight sore throat.

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GLANDULAR FEVER.

INTRODUCTION.—This disease was first described by E. Pfeiffer, of Wiesbaden, in 1889. Since then outbreaks have been recorded in Germany, France, Ireland, and the United States. Sporadic cases are occasionally met with, but in this country it is a rare disease.

Ætiology.—Very little is known of the disease from this point of view. Eighty per cent. of the recorded cases have been in children. It does not appear that the infection is very virulent, and the writer has heard of no instance in which it was disseminated, otherwise than directly by the patient.

The incubation period is believed to vary from five to ten days, being commonly about seven; but the circumstances of a case recorded by Burns go to show that it may be as short as twenty-four hours.

SYMPTOMATOLOGY.—The onset is usually sudden, and the first symptoms are headache, abdominal pain, and vomiting. The temperature rises to 102° F. or higher. Next the neck becomes stiff and painful, and is tender when touched. Enlargement of the lymphatic glands begins on the second to fifth day, but occasionally it is one of the earliest signs. The glands first to be affected are those under, and in front of, one of the sterno-mastoid muscles, usually the left. They are seldom swollen to a size larger than that of a pigeon's egg. They are freely movable and very tender. The skin over them is not inflamed. After a day or two the glands on the right side become affected, and then also the superficial glands in other regions (axillæ, elbows, groins). It is almost certain that in some cases the deep glands are also involved, especially the mesenteric. At any rate, symptoms occur which strongly suggest their implication. In a few cases the spleen and liver are enlarged. The invasion of the various glands may be spread over a week or ten days, and it is two or three weeks before they regain their normal size, which they do without suppuration. While the glandular enlargement is taking place, there is irregular pyrexia. The tongue is furred and the pulse frequent. The bowels are usually confined, but in severe cases there is some looseness, with passage of green mucus. The patient becomes anæmic, and convalescence is tedious. Complications are very rare; in a few instances there has been acute nephritis.

Of the ætioid nœstomy and pathology nothing certain is known.

DIAGNOSIS.—From acute leucæmia glandular fever is distinguished by the fact that in the former disease leucocytosis is marked and persistent, and the anemia is more pronounced. Hodgkin's disease is chronic, and in it the glandular enlargement remains unilateral for a longer period than the entire duration of a case of glandular fever. The note of glandular fever is the successive enlargement of groups of glands within a comparatively short space of time, followed by recovery, also within a relatively short period.

Judging from analogy with the other infectious diseases, one would expect to find that in very mild cases only one or two glands were affected; but even then the transient character of the symptoms would distinguish glandular fever from the graver disorders mentioned above, and from adenitis due to tubercle or syphilis. Glen Jones is of the opinion that glandular fever is more common than is generally supposed, and that it is not infrequently misdiagnosed as an aberrant form of typhoid fever or influenza.

The prognosis is almost invariably favourable. Fatal cases are extremely rare.

TREATMENT.—There is nothing special to be said under this head. The treatment that is applicable to any mild febrile disease should be adopted.

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MUMPS

SYNOPSIS.—Epidemic parotitis.

HYSTORY.—That mumps existed in ancient times is proved by the description of it which is given in the first book of his "Epidemics" by Hippocrates, about 2200 years ago. He mentioned that it was prevalent in Thasos, an island in the *Agæan*, and drew attention to the facts that the parotitis never resulted in suppuration, and that it was occasionally followed by orchitis. Hirsch states that not only Hippocrates, but other medical writers—Greek, Roman, and mediæval—were acquainted with the disease, and distinguished it from other forms of parotitis. But not till the beginning of the eighteenth century was particular attention paid to it from the epidemiological point of view.

ÆTIOLOGY.—Mumps is very widely distributed over the globe, and apparently no climate is inimical to it. While it is to a slight degree endemic in most large cities, it usually occurs in the form of small outbreaks in schools and similar institutions for the young. It is most commonly prevalent in wet, cold weather—*that is*, in this country in the winter and spring. As the disease is not notifiable and seldom fatal, the statistical evidence concerning it is very scanty. The largest annual number of deaths recorded during recent years in England and Wales was 118, in 1908. The age distribution of the deaths recorded annually suggests, however, that other forms of parotitis are included amongst them, for a large proportion are of persons over twenty-five years of age. The experience of most medical practitioners is that mumps occurs most frequently amongst children under fifteen. It may, however, occur at any age, and it is possible that it is most fatal amongst those past middle life. There is no evidence that it affects one sex more than the other. Rikráh quotes White and Coxley as recording instances of infection of the infant while still in the uterus of an infected mother.

The disease is disseminated almost entirely by patients in a direct manner. It is not highly infectious, and it is a question whether the virus is ever effectually conveyed by means of fomites.

The incubation period varies from fourteen to twenty-five days, but is commonly fifteen to twenty-one days.

SYMPTOMATOLOGY.—In most cases the first sign of illness is pain in the region of one parotid, most often the left. But occasionally for a day or two before this sets in the child is fretful, and complains of headache and sore throat, and there may be fever, slight shivering, drowsiness, vomiting, and diarrhoea. The parotid gland begins to swell very soon after pain is noticed, and in the course of two or three days presents a marked prominence in front of and below the ear. The swelling is doughy and has an ill-defined edge, and is more or less painful. Usually the skin is not red, nor does it pit. The submaxillary and sublingual glands on the same side may become affected. The parotid remains swollen for a few days, and then slowly resumes its normal size. In a mild case the period during which the gland remains swollen is only four or five days; in severe cases it may be nearly five weeks. Four or five days after one gland has become enlarged the other is usually involved, and the inflammation runs the same course as the first. In some cases both glands are simultaneously affected at the beginning of the attack. Besides the salivary glands, the cervical lymphatic glands may be enlarged.

In very mild cases the swelling of the parotids is hardly visible, but in those which are severe it may be so extreme that, especially if both glands are involved at the same time, the features of the patient are almost unrecognizable. In such cases the skin over and beyond the limits of the salivary glands is shiny, red, and oedematous, and the pain caused by the slightest movement of the lower jaw and by the act of swallowing is exquisite. Eulsiß states that the pain which is constantly present is aggravated by taking acids, such as lemon-juice, and that "even the sight of them may cause pain in neurotic subjects." In moderate cases the pain, though constant, may be very slight, and in very mild cases there may be neither pain nor tenderness.

The constitutional symptoms usually vary with the degree of swelling of the glands. When this is trivial there may be no other symptom, or at most slight pyrexia. But in severe cases the temperature may for several days remain at a considerable elevation—102° to 104° F., occasionally reaching 105° F. The tongue is furred and dry, the pulse full and frequent, and at times the patient may be delirious. The amount of saliva varies; it may be much diminished, and in consequence there is distressing dryness of the mouth. Occasionally it appears to be, and perhaps really is, increased; the increase may be apparent because pain keeps the patient from swallowing what is secreted, and so it collects in and runs from the mouth.

Parotitis.—Occasionally an attack of mumps shows itself by inflammation of the submaxillary and sublingual glands, and the parotids escape. Goodhart states that cervical adenitis may be the sole local sign. Orchitis or pancreatitis may also be the only local lesions. J. K. Sen records the case of a Hindoo boy, aged nine years, in whom the submaxillary and sublingual glands became affected after the parotitis had subsided. At the same time a high temperature, rigors, muscular twitching, and delirium, were present. These symptoms disappeared after a few hours, but were followed by agonizing pain in the head.

Complications.—These seem to vary in different outbreaks. One of the most recognized, orchitis, is met with not infrequently in boys approaching the age of puberty. According to Catlin, it occurs in about 16 per cent. of males of all ages. Usually it comes on about the seventh or eighth day of the illness, but it may occur later, and Gordon Sharp has recorded the case of a boy of twelve in whom it arose six weeks after the parotitis. Generally the body of the testis becomes swollen and extremely painful and tender; not often is the epididymis involved. The orchitis lasts for three to five days, and then subsides. Unfortunately, it is not infrequently followed by atrophy of the organ. Usually but one testis is affected. The constitutional symptoms may be severe; a temperature of 103° or 104° F.; delirium, sometimes maniacal; prostration; and a slow pulse-rate. The patient's condition may for a day or two seem to be serious, but recovery is the rule. In rare cases the orchitis precedes the parotitis. In females also, even in girls, the genital organs may be affected, especially the vulva and the ovaries. Gordon Sharp states that the sister, aged ten, of the boy mentioned above suffered from vulvitis eight weeks after the attack of mumps.

In some epidemics pancreatitis has been fairly frequent. Sharp saw several cases in an epidemic at Leeds during 1907 and 1908; Wilfred Edgecombe, 5 out of 33 cases of mumps in a boys' school; and Simerin, 10 out of 632. The symptoms are—acute pain, tenderness on pressure, and rigidity in the left hypochondrium; vomiting (in a few cases of bile and blood), which may be repeated; blood

and drops of fat in the stools. Occasionally the pancreas can be felt to be enlarged. The temperature is raised, and in some cases there is collapse. Usually the bowels are constipated. In a few cases there is jaundice. According to Sharp, pancreatitis, the orchitis, may precede the parotitis, or be the only sign of an attack of mumps (see Chapter V., p. 232).

Affections of the organs or sense of hearing are not infrequent. Thus, there may be otitis media, deafness (which may be permanent, and is then probably due to inflammation of the internal ear or to some central lesion), or tinnitus aurium. Inflammation of different parts of the eyes and neighbouring structures, such as the lacrimal gland, occasionally follows mumps. Another less common sequel is epiphora. Rare complications are arthritis, bronchitis, oedema of the larynx, skin eruptions, and symptoms pointing to meningitis or some other cerebral lesion. A relapse is occasionally met with, but second attacks are rare.

MOBIL ANATOMY.—Information on this point is scanty. It has been stated that the connective tissue rather than the parenchyma of the parotid is affected. Suppuration never occurs. There may be cellulitis around the parotid, and the neighbouring lymphatic glands may be enlarged. In a case fatal at the end of seven days, in which there was jaundice, Lemoine found the pancreas much enlarged, and more than twice its normal weight. Microscopically, there was hypertrophy of the glandular acini, with commencing degeneration of the cells.

PATHOLOGY.—Laveran and Catrin obtained a diplococcus in pure culture from the parotid, testis, and blood. It grew on ordinary media, and was decolorized by Gram's method. But it has not been proved that this organism is the cause of the disease.

DIAGNOSIS.—Occasionally acute parotitis occurs as a complication of other infectious diseases. The question may then arise whether the child is also the subject of mumps or not. In the absence of any evidence of exposure to the infection of mumps, or of any of the more pronounced symptoms of that disease, the diagnosis may for a few days be in suspense. In parotitis as a complication the enlargement of the gland never attains the magnitude seen in severe cases of mumps, and the constitutional symptoms are slight.

It is now well recognized that parotitis is not infrequent as a complication of certain chronic disorders (e.g., syphilis), and of more than one disease of the abdominal cavity and its contents, and that it is prone to follow certain operations, especially those upon abdominal organs. But neither the diseases nor the operations referred to are common in children, in whom acute parotitis is nearly always a sign of mumps, unless it appears in the course of another of the acute infectious diseases.

Diphtheria has not seldom been mistaken for mumps, because of the enlargement of the cervical glands. But the parotid is not enlarged in diphtheria, and the fauces are unaffected in mumps, so that a careful examination of the swelling and of the fauces will at once decide the nature of the disease. In glandular fever the parotids are not involved. An attack of acute orchitis or pancreatitis in a child should always rouse a suspicion of mumps.

PROGNOSIS.—This is almost always favourable. Even cases which present severe cerebral symptoms, especially the testicular cases, or symptoms of collapse (the pancreatic cases), nearly always end favourably. But occasionally orchitis ends in atrophy of the testis, and chronic enlargement results from the acute parotitis. Permanent deafness may be caused by an attack of mumps.

TREATMENT.—Confinement to bed should be enforced till a few days after the parotitis has subsided. During the parotid inflammation the patient may not be able to take nourishment in any but a liquid or semiliquid form. If he refuses food because of the pain caused by the act of swallowing, it will be necessary to give it by means of a tube passed through the nose.

The pain due to the inflammation of the parotid, testis, or pancreas will be relieved by the frequent application of hot fomentations, upon which a few drops of tincture of opium have been sprinkled, or by smearing the skin over the affected organ with glycerine and belladonna, and covering it with hot cotton-wool. For the pains in the neck and the constitutional symptoms, Sharp recommends 5-grain doses of salicylate of sodium and bicarbonate of sodium, with aerated soda-water. Delirium and pyrexia should be treated with sponging or wet packs. In pancreatitis, besides the local applications, 3-minim doses of laudanum in $\frac{1}{2}$ drachm of glycerine and water should be given every hour till the pain is relieved (Sharp). Until the pain has passed off, no solid food, and only a very limited amount of liquid, should be allowed. Care should be taken that the mouth is kept clean and moist (never allowed to become dry); it should be frequently irrigated with a mouth-wash. The patient should be considered to be infectious for three weeks from the onset of the parotitis.

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CHAPTER XX

INFECTIOUS DISEASES—Continued

THEODORE THOMPSON

PERTUSSIS.
TYPHOID FEVER.
PARATYPHOID FEVER.
DYSSENTERY.
SEPTICEMIA AND PYEMIA.
ERYSIPELAS.

OTHER MEDIA AND BRISLES.
ACUTE EPIDIDYMITIS.
GONORRHOEA.
ACTINOMYCOSIS.
MALARIA.

PERTUSSIS.

PERTUSSIS is a specific contagious disease characterized by catarrhal congestion of the upper respiratory passages and a paroxysmal cough ending in a long-drawn inspiration or "hoop." In infancy pertussis is one of the most fatal of diseases, but in older children it is a comparatively mild affection. It was first described in Paris by De Baillon (1578) under the name of "tussis quintana," or five-hourly cough.

Ætiology.—It occurs in epidemic form, and is directly contagious from person to person. The greatest number of cases occur in March and April, when the weather is usually cold and wet. It is essentially a disease of infants and young children, the majority of cases occurring between birth and seven years, while after ten years of age there is a marked falling off in the number of individuals affected. The disease is usually introduced into the family by the child attending a kindergarten or infants' school, and thus a house epidemic is started and all the younger children are affected. Even the youngest babies may be attacked, and congenital cases in babies born of mothers suffering from whooping-cough have been described. It should be remembered that elderly people are liable to the complaint, and usually suffer severely, so that care should be taken to avoid contact with infected children. Pertussis appears to be most contagious during the early period of the disease, or catarrhal stage, but there seems no doubt that cases remain infective all through the disease. Susceptibility to whooping-cough is very marked, and during epidemics a very large proportion of children are attacked. One attack protects from another. Second attacks are excessively rare. Return of the cough and hoop are due to other causes which will be mentioned later. The period of incubation is difficult to determine owing to the very gradual onset of the disease. It is probably about fourteen days. The relation of whooping-cough to measles is an important one. In many children the one disease closely follows the other. In both diseases there is a catarrh of the upper respiratory tract, and this may render

the patient susceptible to the second affection. As regards the specific germ of whooping-cough, it is most probably caused by a small Gram-negative bacillus (Bordet and Gengou), which is found in the sputum during the early days of the attack. It has been claimed that the serum of children recovering from pertussis has a marked agglutinating action on this bacillus, while the serum of healthy children has no such action.

Symptomatology.—Catarrhal and spasmodic stages are recognized. In the catarrhal stage the child commences with the symptoms of an ordinary cold. Sneezing, running from the eyes, slight cough and fever, are the early symptoms. In four or five days the cough, instead of abating, gradually increases in severity, becomes more paroxysmal in character, and tends to be worse at night. In an infant the face and neck become very congested during the coughing attack. The paroxysms increase in number and severity until the typical hoop is heard, which marks the commencement of the spasmodic stage.

The Spasmodic Stage.—The child knows that an attack is impending, and runs to its mother or nurse, or holds a chair for support. Older children complain of choking sensations or tickling in the throat before an attack. During an attack or fit the chest is first filled with air, and then a series of some ten to twenty forcible coughs of increasing intensity occur, during which time no air enters the chest. The child's face becomes blue, and in infants almost black; the veins on the face and scalp stand out prominently, the eyeballs protrude, water comes from the eyes, and the blue and swollen tongue is protruded from the mouth. There then follows the long-drawn inspiration which produces the characteristic hoop, the rima glottidis being narrowed and the hyoid bone drawn up. Immediately another series of short coughs follow and another hoop. During the entire paroxysm or fit the child may hoop five or six times; with the final effort a mass of tenacious mucus is brought up, usually of small amount. Vomiting of the stomach contents almost always follows an attack, and may occur so frequently that the child does not get enough food, and wastes rapidly. The number of attacks in the day varies very much. In mild cases there may be no more than four or five fits in the day, but in bad cases they may occur every half-hour during the day and night. The attacks may occur spontaneously or may appear to be induced by emotion (such as anger or laughing), swallowing, especially of cold liquids, by a cold draught of air, or by seeing or hearing a whooping-cough paroxysm in another child. During the night the paroxysms may occur during sleep and without waking the child. The paroxysm, which usually lasts two to five minutes in all, produces very varying effects upon the patient. In infants a very marked degree of asphyxia may be produced, and it may be necessary to perform artificial respiration. Moreover, infants with pertussis often do not hoop at all. During severe paroxysms expulsion of urine and feces may occur, and blood may be forced from the nose. After a severe attack the child may lie down and exhibit much lassitude, with increased breathing and sweating skin. In older and stronger children, however, the very slight effect of the coughing paroxysm is remarkable. They will often resume their interrupted meal or play quite quietly and without any distress after an apparently severe paroxysm. The paroxysms decrease when inflammatory complications arise, and in broncho-pneumonia they may disappear altogether during the height of the attack, and return when the pneumonia has cleared up.

The duration of the spasmodic stage varies very much, but lasts from about three weeks in mild cases to three or four months, if the disease occurs in the winter.

It may apparently subside, when a fresh attack of cold may result in the reappearance of the hooping attack, which may persist for weeks. The habit of paroxysmal cough, once established, is very liable to recur, and in every slight cold for many months after the original disease has passed away the child may have a paroxysm.

The paroxysmal stage gradually declines; first the severity of the individual paroxysms becomes less, and then their number diminishes, and less and less effort is required for expulsion of the sputum. During this stage, which lasts two or three weeks, the child generally feels quite well.

PHYSICAL SIGNS.—During the paroxysmal stage, between the attacks, the child presents a characteristic facies, which become bloated and swollen underneath the eyelids. Hemorrhages into the conjunctiva are not uncommon, but hemorrhages under the skin of the face are rare, and only occur in cachectic children.

Examination of the chest during the catarrhal stage is usually negative. During the paroxysmal stage a certain amount of pulmonary emphysema develops, and the strain on the right side of the heart may be evident in slight dilatation and increase of the second pulmonary sound. In the stage of decline coarse bronchial rales may be heard.

A sublingual ulcer occurs on the frænum in about one half of all cases. It is due to mechanical injury of the frænum by rubbing against the lower incisor teeth. It is single and of variable size, and at times bleeds freely. Infants with no teeth do not show this ulcer. The frænal ulcer is not typical of hooping-cough, but may occur in other varieties of spasmodic cough in children.

COMPLICATIONS AND SEQUELÆ.—*Hæmorrhages* are mechanical, and are due to the marked congestion during the coughing attacks. Epistaxis is the most common. Subconjunctival hæmorrhages and hæmorrhages into the eyelids occur. The mucus expectorated is commonly tinged with blood, but profuse hæmoptysis is uncommon. Cerebral hæmorrhages may be severe enough to produce death, and are usually unilateral. Hemiplegias, monoplegias, and aphasia, have been described. Paraplegia and acute polyneuritis have followed the disease.

Respiratory complications are most serious in pertussis. A secondary bronchopneumonia of septic type is liable to occur at the end of the paroxysmal stage. Owing to the exhaustion of the child, the prognosis is bad. Emphysema of the lungs is almost constantly present, due to the forcible coughing. Occasionally interstitial and subcutaneous emphysema is found, the air being forced into the connective tissues of the lung and mediastinum. Chronic asthma is an occasional sequel of hooping-cough.

Albuminuria from circulatory disturbances in the kidney is usually present, but nephritis is rare. The urine frequently contains crystals of uric acid. *Glycosuria* is occasionally present during and after hooping-cough, and may persist for some time. It may be due to intracranial hæmorrhages. The prognosis is good in such cases, and the sugar ultimately disappears.

In the early stages the blood shows a marked increase in white cells, both polymorphonuclear leucocytes and lymphocytes being increased.

Of the nervous complications, convulsions are the most frequent, and often cause death in young infants. They are probably due to oedema of the brain from venous stasis. The other nervous complications are due to hæmorrhages, and have been already referred to. Sudden blindness may be the result of detachment of the

retina or of a central lesion. In either case vision may be restored. Deafness may be the result of acute otitis media, or, more rarely, of a hæmorrhage into the nervous apparatus of hearing.

Digestive System.—The persistent vomiting may cause grave anæmia and wasting, and the feeding of such children becomes a matter of the greatest anxiety. *Ileo-colitis* is especially common in infants with whooping-cough, and often proves fatal.

Probably the most important sequel of pertussis is tuberculosis of the bronchial glands. This may not progress, but only too frequently tuberculous broncho-pneumonia and general tuberculosis result from a spread of the bacilli into surrounding structures.

Pathology.—The only constant lesions found in whooping-cough are catarrhal conditions of the larynx, trachea, and bronchi. The peribronchial lymph glands are very much swollen and engorged, and are commonly found to be secondarily infected by tuberculosis.

The exact causation of the hoop is somewhat doubtful. There is no doubt that in whooping-cough there is a very much increased excitability of the whole nervous system, and it has been suggested that the cough is a reflex one caused by irritation of the superior laryngeal nerve. The reflex effect must be taken to include the vomiting, which is certainly in some cases not mechanical, and may occur without the cough. The actual exciting cause is probably the small pellet of mucus in the trachea, since the paroxysm always continues until this has been expectorated. Another view is that the reflex vagal irritation is caused by the enlargement of the peribronchial and tracheal lymph glands, and attention is drawn to the fact that a very similar cough occurs when caseous enlargement of these glands takes place. In all cases it is probably a reflex spreading spasm, the result of stimulation of the afferent vagal fibres.

Diagnosis.—In typical cases the diagnosis is easy, the characteristic course of the disease and the whooping paroxysm being distinctive. In the catarrhal stage, the history of exposure to infection and the failure of belladonna or morphine to give relief are suggestive. The leucocytosis may prove of aid in distinguishing the complaint from the early stages of measles, while the latter disease will usually be differentiated by the presence of Koplik's spots on the buccal mucous membrane some days before the rash occurs. Even in the catarrhal stage of pertussis the cough tends to be paroxysmal in character and to be worse at night. It should be remembered that pertussis can occur without any hoop at all, especially in infants. Paroxysmal cough may also be produced by influenza, enlarged tonsils and adenoids, and by caseous enlargement of the bronchial glands. An hysterical cough occurring in paroxysms is not uncommon in neurotic children. It ceases during sleep, and does not affect the general health, while its hollow barking character is usually distinctive.

Very few conditions can simulate the "hoop." It is occasionally heard in cases of tuberculous bronchial glands, and the long-drawn inspiration of laryngismus stridulus may be mistaken for the hoop. In this affection there is no preceding spasmodic cough, a sudden arrest of breathing being followed by a crowing inspiration.

Prognosis.—The most important factor is the age of the patient. In infants it is a most fatal complaint, death resulting from convulsions, broncho-pneumonia,

or diphtheria. Two-thirds of the fatal cases occur during the first year of life. The prognosis is better in the summer than in the winter, because pulmonary complications are less liable to occur. In children over four years old the disease is not a serious one, and complications are rare. The severity of the disease is often measured by the number of paroxysms per diem, but the violence of the attacks should be taken into account as well. Hooping-cough is a very frequent precursor of tuberculosis, the catarrhal condition of the upper air-passages favouring the development of the tubercle bacillus, which usually settles in the glands of the neck and those about the trachea and bronchi.

TREATMENT.—Children with hooping-cough should always be isolated for at least six weeks, or in protracted cases until the expectoration has ceased. After exposure to infection, children should be placed in quarantine for twenty-one days. The sputum should be disinfected. In the case of infants and delicate children, they may be conveniently isolated in two rooms, but in the case of older children with slight forms of the disease it is difficult to isolate them, especially in towns. These children are usually much better when in the open air, and the best solution, where feasible, is to send them away to the country, where they can be more easily kept away from other children.

The most important therapeutic measures are to maintain the general health and frequently ventilate the rooms in which the children are kept. Close, stuffy rooms undoubtedly increase the number of the attacks. Young infants and delicate children should not be allowed out in cold or inclement weather. If the attack is protracted, a change to the seaside or country will usually bring the attack to an end.

The feeding of the patients is most important. In infants the food and bowels should be most carefully attended to, and any symptoms of indigestion should be at once met with the appropriate remedies, and not allowed to pass into an *ileocolitis*. In cases where the vomiting is very frequent, milk must be given in small quantities at frequent intervals, and in severe cases a little brandy may have to be given to maintain the child's strength.

There is no specific for medicinal treatment of hooping-cough. Local treatment of the throat by spraying with a 2 per cent. solution of resorcin has been recommended in the catarrhal stage, with a view to averting the disease; but while the method is harmless, it is rarely effectual. Inhalations are decidedly of value in allaying the catarrh. Eucalypti may be vaporized in the room over a lamp. During the catarrhal stage a mixture of ipecacuanha and ammonia is usually best; but when the cough becomes more paroxysmal sedatives must be used to limit the severity and frequency of the attacks. Of these, paregoric, antipyrin, and the bromides, are of the most value. A useful formula is—

Antipyrin	gr. ii.
Potassi bromid.	gr. v.
Tinct. camphoræ co.	ssvi.
Glycerin.	℥v.
Aquam. roseæ	ad ℥.

This may be given to a child two years old every four hours when the paroxysms are severe. Bromoform, 3 or 4 drops of which may be given on sugar, or 5 minims in capsules to older children, has no advantage over the bromides. Belladonna is recommended by some; it may be given in the catarrhal stage with the ammonia mixture, in the following formula:

Ammon. carb.	gr. i.
Vin. ipecac.	℥ss.
Tinct. belladon.	℥v.
Glycerin	℥v.
Aquas	ad ℥i.

Every four hours.

As the spasmodic stage approaches, the dose of tincture of belladonna should be gradually increased until physiological effects are produced. Belladonna certainly diminishes the severity of the paroxysms, but has no effect on the length of the disease, and its exhibition must be closely watched. Quinine, though warmly advised by some writers, has little effect. It should not be given to infants, because it tends to upset digestion; for older children it must be given in tasteless form, and full doses must be used.

A host of preparations have been praised for whooping-cough, but no drug has yet been found to cut short the disease. Management of the child's nutrition, as much fresh air as possible, and a wise use of sedatives to control the severity of the paroxysm, are better than indiscriminate drugging. During convalescence cod-liver-oil, aux varius, and iron, and if possible a change of air, are very useful in preventing the onset of tubercular infection.

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TYPHOID FEVER.

INTRODUCTION.—Typhoid fever is a general infection due to a specific micro-organism—the *Bacillus typhosus*. Classically the disease is distinguished by its gradual onset, a period of continuous fever, intestinal disturbance, an enlarged spleen, and a rose-coloured eruption. In children as a rule the disease is a comparatively mild one, and it is probable that in infants many cases are overlooked, a diagnosis of enterocolitis being made. The anatomical changes consist in hyperplasia and ulceration of the lymphoid follicles of the intestines, swelling of the mesenteric glands and spleen, and evidence of toxic changes in the parenchyma of other organs.

ÆTIOLOGY.—The *B. typhosus*, which has been shown to be the cause of typhoid fever, was first described by Eberth, in 1880. It is a short, thick, motile bacillus with rounded ends; it measures 2 to 4 μ in length and 0.5 μ in thickness, and has numerous flagella, which may be readily stained by special methods. It does not stain by Gram's method, nor is it acid-fast. In the tissues and in recent cultures it stains uniformly by the ordinary aniline dyes. The bacillus grows readily on the various nutritive media. Shortly stated, the important characteristics are that on agar the growth has a delicate transparent appearance, it is a facultative anaerobe, and in stab cultures growth takes place all along the track of the needle; it does not liquefy gelatine, and does not form gas; it does not coagulate milk nor form indol in peptone broth.

It has recently been shown that the *B. typhosus* is only one of a closely related group of bacilli, the other members of which are the *B. paratyphosus* A, *B. paraty-*

phosa B. B. enteritidis of Gärtner, the paratyphoid bacillus, and the constant inhabitant of the intestines, the *B. coli*. These bacilli differ slightly from one another, but the characters of the *B. typhosus* above enumerated will usually enable it to be distinguished from the other members of the group. It should be noted that both the paratyphoid bacilli may produce symptoms very similar to those of typhoid fever, while the paracolon and colon bacilli do not do so. The question of paratyphoid fever will be referred to later.

METHOD OF CONVEYANCE.—Typhoid bacilli can live in sterile water for weeks, but it is probable that under ordinary conditions of water-supply they do not long survive. They resist drying and freezing, but are killed in ten minutes by a temperature of 60° C., and by antiseptics such as carbolic acid (1:200) and corrosive sublimate (1:2:500). Sunlight rapidly destroys them.

Direct contagion is a not uncommon method of transmission. It is impossible for those in charge of a case of typhoid fever to avoid contamination of the fingers, and without scrupulous care in disinfection the germs may in this way become widely distributed. The same care should be exercised in the treatment of post-typhoidal abscesses as in typhoid fever itself. An abscess of the ribs occurring twenty years after an attack of typhoid fever was opened in a surgical ward, and gave a pure culture of typhoid bacilli. The Sister of the ward who dressed the case contracted typhoid fever about a fortnight afterwards.

Infection of drinking-water is the most common cause of epidemics, but articles of food are infrequently may carry infection. Both milk and beer may become contaminated through cleaning vessels with infected water. Ice and salads may sometimes convey the germs. The importance of oysters as a source of infection has excited much attention, but it is probable that very few cases, relatively, result from this method of infection. Recently, fried fish, so much consumed by the lower classes, has been blamed for an outbreak of typhoid fever. Typhoid bacilli have been found in the bodies of flies, and it is probable that infection of food may take place by their agency.

Contamination of the soil in the neighbourhood of sewers, ashpits, or cesspools may be of some importance in the case of children, and it is possible that in the case of young boys infection may take place directly from the soil, while at play in gravel games. In breast-fed infants infection takes place through the agency of the mother through the water used in bathing. In all these cases infection of the body takes place through the gastro-intestinal tract. It is probable that this is the only method of infection, except *in utero*, where the bacilli may pass to the fetus via the umbilical vein.

SYMPTOMATOLOGY.—The symptoms of typhoid fever vary very considerably in individual cases in adults, but in children this is especially noticeable. The incubation period is usually about fourteen days, but much shorter (three days) and longer (twenty-three days) periods have been noted. During this period there may be no symptoms whatever, or a certain amount of headache and lassitude may be noticed. In an interesting group of cases, in which the infection has been a mixed one, there is a history of a gastro-enteritis at the time of infection, followed about a fortnight later by the onset of typhoid fever.

Usually the onset is quite insidious, and is ushered in by increasing headache, stomata, and abdominal pain; epistaxis often occurs early in the disease. The patient at last takes to his bed, and this is usually taken to mark the onset. In

the first week there is a steady increase in the fever, which mounts up 1° to $1\frac{1}{2}^{\circ}$ higher each evening, eventually reaching 103° or 104° F. The pulse rises to 90 or 100 beats per minute, and is dicrotic and of low tension. This combination of high temperature of gradual onset with a comparatively low pulse-rate is very suggestive of typhoid fever. The tongue becomes white and coated, and the abdomen is slightly distended and tender. Gurgling of gas in the intestine in the right iliac region may often be elicited on pressure at this stage. The state of the evacuations varies much; they may be constipated, or two or three loose stools may be passed daily. Enlargement of the area of splenic dullness may be observed. Headache increases, and there may be sleeplessness or mental confusion at night. A short, dry cough often is troublesome, and bronchitic sounds may be heard in the chest. A peculiar brown discoloration of the palms of the hands has been observed in many cases, and the patients are said to exude a characteristic odour. In the second week the fever remains high, with small morning remissions; the pulse becomes quicker; the face is dull and heavy, with flushed cheeks and moist skin. At the beginning of the second week the characteristic rash appears; it consists of slightly raised, circular, rose-coloured, flattened papules from 2 to 4 millimetres in diameter. They disappear on pressure, and last in all two or three days. Fresh crops of papules come out during the second and third weeks. They occur chiefly on the abdomen, chest, and back, very rarely on the limbs or face, and vary in number from two or three to a very abundant eruption. Typhoid bacilli have been found in the spots. The spleen is now palpable, and is usually soft, although occasionally it feels quite firm. Sometimes it is slightly painful on pressure. The gastro-intestinal symptoms become more pronounced; the lips become dry; the tongue is dry and covered with brown fur; diarrhoea increases, and profuse pale yellow (pea-soup) stools are passed. Abdominal distension and general tenderness increases. Increasing deafness is frequently noticed. During the third week the symptoms gradually subside. This is the usual course in children. In severer cases, however, the pulse becomes more rapid; marked morning remissions of the temperature occur. Weakness and loss of flesh appear, and delirium or mania may be present. In the third week intestinal hæmorrhage or perforation of the bowel are the chief complications to be feared.

During the fourth week the temperature gradually drops to normal, and the tongue becomes clean, first at the tip and sides, and last of all on the dorsum. In severe cases heart failure is to be apprehended during this week, the apex beat becoming displaced towards the left, and the first sound getting gradually weaker, and finally may disappear altogether, whilst the pulse becomes more and more rapid and feeble.

During the fifth and sixth weeks appetite returns. Irregular rises of temperature often occur, due to emotional causes resulting from the visit of friends, or to constipation. Relapses are common during these weeks, when the disease may be reproduced in miniature, though occasionally the relapse may be more severe than the original attack. During this period occur also many of the complications, such as thrombosis and neuritis.

The above is a brief account of a moderately severe case of typhoid fever. It remains to consider the variations which may occur in children.

Typhoid in the Fetus.—If a pregnant woman contracts typhoid fever, abortion takes place in 50 to 70 per cent. of the cases, the fetus dying *in utero*. It seems well established that typhoid bacilli can pass from the maternal circulation into

the foetus through the placenta. The disease is thus a primary typhoid septicæmia in the foetus. In a certain number of cases the child may be born alive suffering from the infection. The cases present no special features, an acute cachexia, rapidly fatal, being the usual condition. Enlargement of the liver and spleen and raised temperature sometimes occur, and jaundice has been noted. After death the bacilli are usually found in the liver and spleen, which are swollen and show hæmorrhages. Intestinal lesions are not common. Occasionally children are born alive with no symptoms of disease. Widal's reaction may be present in their serum, in which case they must have had the disease in utero and recovered, or the agglutinins may have passed from the mother to the child. The agglutinin reaction is, however, not always present. Briefly, typhoid fever in pregnant women usually causes abortion. Of those children born alive, the majority rapidly succumb to an acute typhoid septicæmia.

Typhoid in Infancy.—There is a considerable difference between the symptoms of typhoid fever in infancy and the disease in older children. The disease is a mild one, and is very liable to be mistaken for one of the forms of enterocolitis so common at this age. This has led to considerable doubt as to its frequency in young infants. The most striking feature is the short duration of the pyrexia, which is generally about a fortnight, and rarely exceeds three weeks. Diarrhoea occurs in some cases, but is not so common as in adults, and constipation is generally present. Distension of the abdomen is usually present. The spleen is usually enlarged. Persistence of the splenic enlargement after the temperature has subsided is said to indicate a tendency to relapse. Both epistaxis and the rose-coloured spots are less frequent in infants than in older children. Bronchitis is present in most cases, but, contrary to what would be expected, broncho-pneumonia is not common, though when it does occur the cases are usually severe, and the intestinal lesions are liable to be overlooked. Nervous symptoms are not so common as in adults, and the children are not especially restless. Symptoms simulating meningitis are sometimes present, but true meningitis due to the typhoid bacilli can occur; in these cases typhoid bacilli can be found in the cerebro-spinal fluid by lumbar puncture. Widal's reaction can usually be obtained during the second week, and this is a very important point in the diagnosis, in view of the fact that defervescence is so rapid in infantile typhoid, and that from time to time cases of apyrexial typhoid fever occur.

Typhoid Fever in Older Children.—**Symptoms.**—In the majority of cases the onset is gradual. Lassitude, headache, and drowsiness are the usual early symptoms. Vomiting is a much more common symptom of the onset in children than in adults, and in children a sudden onset of the disease should not negative a diagnosis of typhoid. Occasionally an abrupt onset with high fever and symptoms simulating meningitis occurs. Epistaxis is no more common in children than in adults. The ambulatory form of onset is not rare in children, since the whole tendency of the disease in them is to run a mild course. An interesting group are those cases which begin with a severe bronchial catarrh, and which simulate broncho-pneumonia or miliary tuberculosis.

The pyrexia is rarely so high in children as in adults, and is much better borne. The short duration of the pyrexia (eleven to fourteen days) in many cases is striking.

The pulse-rate is seldom markedly increased, and the combination of a moderate pulse-rate and a high continued fever is striking.

The condition of the bowels in the typhoid of children is interesting. Diarrhoea is rarely profuse, and is not so commonly a feature as in adults, and in children constipation may be present all through the attack. Tenderness and pain in the right iliac fossa occasionally give rise to a suspicion of appendicitis.

The spleen is usually markedly enlarged in children, and can be palpated at the end of the first week. Occasionally it is very firm in texture.

The rash in children is not so constant or so profuse as in adults, but cases differ much one from another. A fine desquamation of the skin may follow the rash. Herpes of the lips does occur in the typhoid of children, and more frequently than in adults. Occasionally nodes of the ears, nose, or genitals supervene, and multiple areas of gangrene on the skin have been noted.

The condition of the blood is important. There is usually very little reduction in the red blood-cells in the early stages, and the colour of the mucous membranes is retained until well into the third week. It is in the later stages that both the red blood-cells and hæmoglobin may be diminished. There is no leucocytosis, and as a rule leucopenia is a feature and is progressive. The *Bacillus typhosus* is frequently found in the blood-stream, and may sometimes be demonstrated in the blood by culture before the Widal reaction can be obtained.

COMPLICATIONS.—Gastric symptoms are uncommon even in children, and vomiting is a rare event unless associated with peritonitis. Diarrhoea is variable, and is said to be more common in children, but in many cases constipation is marked throughout the illness; it may be the result of improper feeding, and modifications of the milk given by dilution, decalcification or peptonizing, may cure the condition at once.

Shock or collapse from the bowels may occur, often without warning. The patient becomes collapsed, and the temperature falls 6° or 7° F. in a few hours. Death may occur before the blood appears in the stools.

Perforation of the intestine may occur in the third week. Sudden sharp abdominal pain may occur, but in profound toxæmia the patient may not be able to give expression to his symptoms. A fall in temperature with a steady rise in the pulse-rate are important symptoms. Limitation of respiratory abdominal movements, increased rigidity, and distension and dulness in the flanks, are found. Obliteration of the liver dulness is a valuable sign, especially if the abdomen is not greatly distended. In a few hours the signs of general peritonitis appear, the temperature again rises, the pulse becomes more rapid, feeble, and threadlike, vomiting and hiccough occur, and a marked sinking in of the eyes and cheeks is noticed. Perforation is nearly always fatal unless treated by operation. Out of twelve cases under six years old, six recovered as the result of operation and surviving the perforation.

Cardiac failure is a not uncommon complication of the third and fourth weeks, and the daily examination of the heart should never be omitted. The onset of cardiac failure is marked by a gradual weakening of the first sound of the heart, which may disappear entirely. The cardiac apex may also be displaced to the left. The occurrence of these signs demands instant treatment. They are due to myocardial degeneration.

Asteritis may occur with or without blocking of the vessel. Gangrene of a limb has followed. Venous thrombosis is a not uncommon sequel of typhoid fever. Femoral thrombosis is the most common form. Swelling of the leg and more or less pain in the groin are present.

Respiratory complications are uncommon. Epistaxis does not occur more frequently in children than in adults. Laryngitis and ulceration of the pharynx are rare in children. Bronchitis is an accompaniment of almost every case, but is not so often followed by a severe broncho-pneumonia as might be expected.

Nervous System.—Meningeal irritation is not uncommon in children. Headache, convulsions, photophobia, and retraction of the neck may occur. In these cases a clear fluid under high pressure is obtained by lumbar puncture. In other cases a true meningitis due to the *E. typhosus* may be present, and the fluid obtained by lumbar puncture may contain pus and typhoid bacilli, either in pure culture or mixed with other organisms.

Xeroderma, generally of the lower limbs, with marked wasting, loss of knee-jerks, and tenderness of the valves, is an occasional sequel. Otitis media occasionally occurs.

Urinary complications are rare. Retention of urine may be found at any stage, and may be a cause of abdominal pain. In the third week typhoid bacilli are often found in the urine, without any symptoms of inflammation of the urinary tract. In other cases a pyelitis or cystitis may be found.

Bone lesions are not uncommon, and in children may occur during the febrile period. In our case multiple periostitic nodules were present on the frontal bone, ribs, and tibiae. The acute inflammation rapidly subsides, but it is remarkable that many years later an abscess may form, and a pure culture of typhoid bacilli has been obtained from an abscess in connection with a bone twenty years after the original attack.

Typhoid spine is a well-marked condition due to peri-ependylitis. The pain is usually severe, and there may be slight deformity. It is often confused with tuberculous caries of the spine in children.

DIAGNOSIS.—In children, typhoid fever with diarrhoea with green stools, intoxication, and persistent pyrexia, can only be distinguished from enteritis by Widal's test. Enlargement of the spleen and the presence of rose spots are of much value. The character of the pulse and temperature should be carefully observed. In typhoid fever the pulse is slow in proportion to the temperature in the early stages.

Acute solitary tuberculousis may often be mistaken for typhoid fever in children. A negative Widal's test and absence of typhoid bacilli in blood-culture is of decisive importance. The pulse is relatively more rapid in tuberculousis. Bronchitis, enlargement of the spleen, and Ehrlich's diazo-reaction, are common to both diseases. Examination of the fundus oculi may show exudidal tubercles, and I have seen the diagnosis made by this alone. The diagnosis from meningitis is made by lumbar puncture. *Broncho-pneumonia* with delayed appearance of the physical signs may be a difficulty, but the absence of Widal's test is important. Of the gastro-intestinal manifestations, *opercula* with deeply-seated localized suppurative has led to error. Careful abdominal and rectal examination should lead to a correct diagnosis in these cases.

PATHOLOGY.—The specific lesion of typhoid fever is found in the lymphoid tissue of the intestine. In the earliest stage swelling of Peyer's patches in the ileum and caecum is found, and there is also hyperplasia of the solitary lymphoid follicles. The vessels are engorged; there is a great increase in the lymphocytes in the follicle. Some endothelial cells are also present, and typhoid bacilli may be

found in the swollen patch. In a few cases resolution may take place and the follicle may shrink, but usually necrosis of the lymph follicle occurs and a slough is formed. The extent of the necrosis is variable; it may be superficial, or may reach deeply into the muscular coat, or even to the peritoneal surface. The necrotic follicles are mainly found in the intestine adjacent to the ileo-cæcal valve. Towards the end of the third week of the disease the sloughs separate, and an oval ulcer is formed, with a smooth, clean base. During this period hæmorrhage from a vessel at the base of the ulcer or perforation of the bowel may take place. Healing takes place by granulation over the base of the ulcer, and the healed ulcer consists of a minimal amount of scar tissue, is somewhat depressed, and generally pigmented. The ulcers never encircle the bowel, and consequently stenosis of the intestine does not follow. Perforation of the bowel is usually in the ileum. The mesenteric glands are greatly swollen, and often show necrotic areas and small hæmorrhages.

The spleen is large, very soft, and dark red in colour. Cloudy swelling occurs in the liver and kidneys and groups of bacilli may be found in these organs. Catarrhal inflammation of the bile-ducts and gall-bladder, and of the pelvis of the kidney, are sometimes found. Ulceration of the posterior wall of the larynx has been described, and bronchitis and hypostatic pneumonia are common. Endocarditis and pericarditis are rare. Atheroma of the aorta is not uncommon in young subjects dying of typhoid fever. Thrombi may be found in the veins, and sometimes in the arteries. Meningitis is rarely found, and the exudation is generally sero-fibrinous. Occasionally the hyaline degeneration of the muscles described by Zenker may be found. The rectus abdominis is said to be the muscle most commonly affected.

PROGNOSIS.—In children the mortality from typhoid fever is much lower than in adults. This is due to the milder nature of the disease, and to the less frequent occurrence of the severe complications, such as hæmorrhage and perforation of the bowel.

TREATMENT.—It is necessary to recognize cases of typhoid fever as early as possible, and to prevent the spread of the disease by destroying the typhoid bacilli in the discharges as they leave the patient. The stools contain the bacillus at the end of the first week. They should be mixed with twice their volume of 1 in 20 carbolic acid, and allowed to stand for several hours before being thrown away. Solution of chloride of lime can also be used. The urine should be mixed with an equal volume of 1 in 20 carbolic, and then be thrown away, after standing for three hours. Urethrin may be administered to the patient, and will cause a disappearance of bacilli from the urine.

All the linen and any articles in contact with the patient must be soaked for two hours in 1 in 20 carbolic lotion, and then sent to the laundry to be boiled. It should be firmly impressed upon those who are in charge of the patient that any object coming in contact with the patient, or with the discharges from his body, must be sterilized. The nurse should wear an apron made of rubber when working over a typhoid patient. This should be frequently washed with carbolic acid or bichloride of mercury solution. After washing or touching the patient, the nurse should always rinse her hands thoroughly in a solution of lysol (1 in 200). In summer the windows should have fly-screens. When epidemics are present, drinking-water and milk should always be boiled.

Vaccination.—This method is rarely useful during a case of typhoid fever in a child, but prophylactic vaccination by Wright's method should always be used in children who are likely to be exposed to the infection (see p. 23).

Treatment of the Acute Diet.—Milk and water are the essentials during the fever. In the very acute and severe stages of the disease, water may be given alone for twenty-four hours, especially if there is abdominal distension. Infants may be fed on whey or on peptonized milk. Liquid custard is useful as a change from milk. If there are many curds in the stool, the milk must be sterilized and diluted, or water substituted for a time. Each case must be treated on its merits; and if the child has a good appetite, and appears to digest the food well, more liquid food should be given, and such things as junket and bread jelly may be added to the diet. No solid food need be given until the temperature has been normal for a week. For constipation the best treatment is the use of a small enema every morning or every other morning.

Antipyretic measures are not so necessary as in adults. Tepid sponging of the skin should be used if the temperature is over 103° F., but the cold baths which are so useful in adults are unsuitable for young children.

Intestinal antiseptics are not of much value in typhoid. Calomel in doses of $\frac{1}{4}$ grain every four hours may be used in cases of obstinate constipation, but should not be prescribed as a routine. Benzo-naphthol, cyllin, nascent chlorine, sulphurous acid, and other antiseptics, have not been proved to influence favourably the course of the disease.

The Treatment of Special Symptoms.—If hemorrhage occurs, absolute rest is essential, and as little movement of the patient should be allowed as possible. A light icebag should be placed on the abdomen, the food restricted for twelve hours, and calcium lactate in doses of 15 grains should be given every four hours.

In cases of collapse saline should be given beneath the skin. For perforation of the bowel, operation should be performed as soon as the diagnosis is established.

In high fever with evidence of cardiac weakness, such as increasing rapidity of the pulse and weakness of the first sound of the heart, brandy should be given.

The bacilluria of the third week should be treated by the exhibition of urotropin in 5-grain doses three times a day.

PARATYPHOID FEVER.

Within the last ten years there have been described a large number of cases of a disease which very closely resembled that due to the B. typhosus, but in which the blood fails to give the agglutinin reaction to that organism. In these cases bacteriological examination of the blood and faeces reveals the absence of the B. typhosus, and the presence of one or other of two closely related bacilli, which have been named paratyphosus A and B. Moreover, the blood of the patient agglutinates the organism responsible for the disease. According to Lorrain Smith, paratyphosus B is found in 80 per cent. of the cases, and this organism resembles B. coli in its cultural reactions, but does not form indol in broth cultures. Paratyphosus A resembles the typhoid bacillus in its characters. The bacilli are actively motile, stain by ordinary aniline dyes, but do not retain the stain by Gram's method.

SYMPTOMATOLOGY.—The disease is not rare, and cases have been described in children. The symptoms are those of a mild case of typhoid fever. The illness begins with anæsthesia, headache, and irregular fever. The tongue is moist and coated, and labial herpes is common. The abdomen is distended, and constipation is the rule. The splenic dulness is increased, but the spleen is not always palpable. Rose spots are present in some of the cases. Enteritis is common. The temperature varies between 100° and 103° F., and may fall in two to three weeks by crisis. Relapses may occur. Complications sometimes are found, and intestinal hæmorrhages have been present in 5 per cent. of cases. Perforation has not been recorded. Albuminuria, nephritis, and septic conditions, occur.

Examination of the blood shows absence of leucocytosis, and sometimes leucopenia, just as in typhoid fever; but on performing the Widal test to the B. typhosus, the patient's serum fails to show the agglutinin reaction, while to either paratyphosus A or B the reaction may be positive even with high dilutions.

PATHOLOGY.—There have been necropsies in several cases, though a fatal result is rare. Typical ulceration of Peyer's patches is not found, but a superficial ragged ulceration has been found in the ileum just above the ileo-cæcal valve.

Diagnosis depends on the agglutinating reactions of the serum. If a case which is clinically a mild attack of typhoid fever fails to give a positive Widal reaction to the B. typhosus after the tenth day of the disease, paratyphoid fever should be suspected, and the serum should be tested for paratyphoid agglutinins and the bacilli recovered from the faeces.

The **PROGNOSIS** is good. About 4 per cent. of the recorded cases have proved fatal.

The **TREATMENT** is exactly similar to that already described for typhoid fever.

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INFLUENZA.

Influenza is an acute, highly infectious disease. There are two conditions to which this term has been applied:

I. Epidemic Influenza, a pandemic disease due to the bacillus described by Pfeiffer. It begins in the East, and spreads over Europe with great rapidity, large numbers of people being attacked. The rapidity of its spread is that of the modern methods of travel. After a great pandemic, sporadic cases due to the same bacillus often occur for some years.

2. Pseudo-Influenza, or catarrhal fever, the clinical symptoms of which resemble those of influenza. Examination of the nasal discharge shows that this may be due to various bacilli, of which the more common are the pneumococcus, the *Micrococcus catarrhalis*, and the *Bacillus septus*. These organisms may be present in pure culture, or the infection may be a mixed one.

The bacillus described by Pfeiffer in 1892 was isolated from the nasal and bronchial secretion. It is a very short, non-motile organism contained in enormous numbers in the secretions. It stains well with Löffler's methylene blue. The incubation period is short, being under four days. One attack does not protect from another.

SYMPTOMATOLOGY.—The onset of influenza is acute, and the variability of the symptoms is very great. In infants and children they are usually not so severe as in adults. The chief danger is the supervention of broncho-pneumonia, which is of the secondary type and is always a dangerous complication. The temperature rises suddenly to 103° or 104° F. There are muscular pains, vomiting, and great prostration. A mild coryza and some slight redness of the fauces often occur. As a rule the fever subsides in two or three days, but the child is weak and anæmic for some time afterwards. In uncomplicated influenza there is no leucocytosis. Such are the usual symptoms in a mild case, but there are distinct variations in type.

Nervous Type.—The onset is abrupt, with high fever of fluctuating character. Vomiting, headache, and convulsions, are common. Head retraction and delirium may simulate meningitis. These symptoms are generally toxic, and pass off in a few days, but in other cases definite cerebro-spinal meningitis occurs, and the influenza bacillus has been found in the lumbar puncture fluid on many occasions. Abscess of the brain has been found in fatal cases. Not infrequently myelitis may develop, with resulting spastic paraplegia. In a recent case in a child the myelitis occurred fourteen days after the attack of influenza, and resulted in gross spastic paraplegia, which persisted for fifteen months and then gradually improved. The ultimate prognosis in these infectious cases of myelitis is more hopeful than in some other forms. Multiple neuritis does occur in children, but is perhaps not so common as in the influenza of adults.

Respiratory Type.—In this group, in addition to the symptoms of the uncomplicated variety, there is severe catarrhal inflammation of the upper respiratory tract, which often spreads down the bronchial tubes and results in a secondary broncho-pneumonia. Severe coryza and pharyngitis, accompanied by a distressing cough, are common. A very troublesome complication, especially common in children, is otitis media. The temperature may persist for days from this cause, and does not fall until the evacuation of the pus through the drums, either spontaneously or as the result of puncture. Swelling of the cervical glands is also common. Broncho-pneumonia may follow in a case of this type. The infection is probably a mixed one, the pneumococcus being present as well as the influenza bacillus. As a rule these cases are serious and resolution is prolonged, the temperature falling by lysis. Pleurisy and empyema are frequent complications.

The **Gastro-Enteric Type** is common in children. The onset is sudden, with violent vomiting, abdominal pain, and profuse diarrhoea. The temperature is raised. The spleen may be enlarged. In very young infants the collapse and prostration may be extreme, and death may occur in three or four days, the babe being seemingly overwhelmed by the intensity of the poison.

Febile Type.—In this type there may be prolonged fever, which may closely simulate typhoid fever. Such cases are often very persistent, and recovery may only be brought about by change to a warmer climate.

PATHOLOGY.—There is no special pathology of influenza. The chief complications in the pulmonary and nervous systems have been mentioned. A not uncommon pulmonary complication is the supervention of diffuse bronchiectasis. Pericarditis and vegetative endocarditis are occasionally found after death, and in some cases the bacillus of Pfeiffer has been cultivated from the blood during life. Nephritis is occasionally observed.

DIAGNOSIS.—During an epidemic the diagnosis is easy. The postural position, out of all proportion to the fever and physical signs of disease, is a most important diagnostic feature. Bacteriological examination of the sputum will reveal the presence of Pfeiffer's bacillus. In many of the cases of pseudo-influenza this bacillus is not present, but other organisms are present in the nasal and bronchial secretions, though the clinical symptoms may closely resemble those of true influenza.

PROGNOSIS.—In children the prognosis is as a rule good. Few die except from pulmonary complications. In very young infants, however, death may occur from involvement of the gastro-intestinal tract. Convalescence is often prolonged, and unless the greatest care be taken a relapse may follow, or a condition of malnutrition or chronic ill-health may render the child an easy victim to some other form of acute disease.

TREATMENT.—Children with influenza should be isolated whenever possible. Fumigation of the rooms with formalin vapour should be undertaken after an attack. The nasal and bronchial secretions should be disinfected. In every case influenza should be treated as a serious complaint, and the child put to bed. A liquid diet should be given while the temperature is high, but as soon as possible the child should be encouraged to eat a full and generous diet. At the outset a teaspoonful of castor-oil or a grain of calomel should be given. Free perspiration should be encouraged by hot drinks. For a child of one year old the following mixture may be given: Sodii salicyl. 2 grains; sodii citrat. 4 grains; glycerine, 10 minims; aq. anethi ad ℥i; every four hours. A high temperature should be treated by tepid sponging. For delirium and restlessness an ice-cap may be tried, and small doses of phenacetin (1 grain) every four hours are useful. Severe vomiting should be treated by giving small doses of iced champagne by the mouth, or an enema containing 7 grains of potassium bromide. In children over three years of age tannate of quinine may be given—1 to 2 grains in hot milk three times a day. Cardiac complications, such as feeble pulse and dilatation of the heart, must be carefully watched for, and brandy given. In urgent cases, $\frac{1}{15}$ grain of strychnine may be given as a hypodermic injection to a child one year old. Pneumonia must be treated on the usual lines. Persistence of bronchitis, with a tendency to dilatation of the tubes, must be met by creosote inhalations and the exhibition of cod-liver-oil internally. A change to a warmer climate is often of great value in such cases. The child must be carefully nursed for some time, and care should be taken to avoid chill. After the attack is over it is a good plan to move the child to another part of the house, as reinfection may take place from the sick-room.

SEPTICÆMIA AND PYÆMIA.

The septic infections of children may be divided into three groups. They all result from the entrance of micro-organisms or their poisons into the system through a breach in the cutaneous and mucous surfaces of the body. The three different results which follow the entrance of bacteria into a wound are not uncommonly combined in a particular case, but it is convenient to study them separately.

The first is septæmia, or septic intoxication. In this the bacteria themselves remain localized to the wound, but their poisonous products or toxins gain entrance to the blood-stream. Provided the dose of poison absorbed from the wound is not a fatal one, it is gradually neutralized and eliminated from the body. As the poisons in the blood can only become increased by absorption of fresh supplies from the original wound, it follows that, if the body is able to deal with the amount of poison already in the blood-stream, removal of the micro-organisms from the wound will produce a cure of the disease.

In the second form of septic disease, septicæmia, or septic infection, we have the actual entrance of the living micro-organisms themselves into the blood-stream, where they grow and multiply. In this form of disease the poisons are being produced by the bacteria themselves in the blood-stream, and consequently removal of the organisms from the original wound of entry will not bring about a cure.

In pyæmia the living organisms are again in the blood-stream, but, in addition, abscesses are formed in the various organs of the body. The usual way in which these abscesses are brought about is by the formation of septic emboli. The wall of a vein near to the original wound becomes injured by the micro-organisms or their poisons. In consequence a clot forms in the vein, this clot becomes infected by the pyogenic cocci, and is softened and broken down. Portions of the softened clot become dislodged, and the mass of blood-clot and organisms are swept away to the lungs, where they are arrested in the terminal branches of the pulmonary artery. Lodging in this small vessel, usually where it bifurcates, the organisms in the septic embolus grow and multiply. Inflammatory changes take place around the blocked vessel, polymorphonuclear leucocytes infiltrate the surrounding tissue which are killed and subsequently liquefied, and the result is the formation of a small abscess.

It is clear, however, that the dislodgement of septic emboli from septic thrombi in veins in the neighbourhood of the wound will only account for the occurrence of secondary abscesses in the lungs. In pyæmia, however, the abscesses are found in all parts of the body, and must clearly have been due to some process occurring within the systemic arteries.

Several explanations have been given:

1. It has been supposed that in the neighbourhood of the secondary abscesses in the lungs thrombosis occurs in the radicles of the pulmonary veins. These clots become infected, and portions of them become dislodged and pass to the left ventricle and so to the systemic arteries, and, being carried to various parts of the body, result in the formation of multiple abscesses in the organs.

2. Whilst it is not possible for portions of infected clot to pass through the pulmonary capillaries, the organisms themselves can do so, and are found in the arterial blood-stream. It has been thought that colonies of micro-organisms may

collect in the arteries, and by the aggregation of red blood-cells and leucocytes a particle might be formed in the arteries themselves, sufficiently large to occlude one of the smaller systemic capillaries. A more probable method, by which the cocci circulating in the arteries may produce pyæmic abscesses, is that the organisms become attached to the endothelial cells lining the wall of a capillary, and growing there ultimately occlude the lumen of the vessel.

3. In view of the fact that in certain forms of pyæmia abscesses are liable to form in special structures only, such as the joints, it would seem that in certain cases the cocci may display a selective affinity for certain structures which provide a more suitable nidus for their growth than other parts of the body. Such selective affinity may be due to the nature of the structures concerned, or may be determined by injury.

4. It has been pointed out that systemic abscesses are more frequently found in the kidneys than in other organs of the body. Recently it has been shown that the kidneys play a very large part in the excretion of living organisms from the body. The bacilluria of the third week of typhoid fever is a well-known example. It is therefore possible that the organisms may pass into the urinary tubules from the blood-stream, and, finding there suitable conditions for growth, may multiply, and form a small abscess. The excretory activity of the kidneys for living organisms may thus be the reason why abscesses are so frequently found in these organs. The same view will probably account for the occurrence of abscesses in the parotid, and it may well be that the multiple cutaneous abscesses, so often seen in wasted children, are the result of the excretory activity of the sudoriferous and sebaceous glands.

In portal pyæmia, a not uncommon form in new-born babies as the result of umbilical infection, the umbilical veins become thrombosed, and portions of clot, becoming detached, pass to the liver and lungs. In another condition, portal phlebitis, the suppuration takes place into the walls of the vein, the cavity of which becomes converted into a sac containing pus. This also is sometimes seen in the umbilical sepsis of newly-born children.

The Acute Septic Diseases of the New-Born.—In the new-born child any of the three varieties of infection above described may occur. In some there is only a localized external inflammation; in others, a true septicæmia without any large local lesion; in others, multiple abscesses may form in the viscera, joints or tissues—a true pyæmia. The organisms concerned are streptococci and staphylococci; more rarely, other pyogenic organisms are found.

In four-fifths of the cases the point of entrance is the umbilical wound. Bottle-fed infants are more frequently attacked than breast-fed babies, as they do not receive the protective substances which have been shown to occur in the mother's milk. Weak and poorly-developed children are more liable to septic infection than well-nourished babies, while premature children are especially predisposed to succumb to an infection owing to delay in the closure of the umbilical vessels.

Infections of the Cord.—Instead of drying up in four or five days, the cord may soften and putrefy; this is due to want of cleanliness on the part of the child's attendants, which permits septic organisms to reach the cord. As the cord is in close proximity to the fresh umbilical wound, a form of true septicæmia may be thus induced. The cord should, however, never be pulled off, as the putrefaction can be rapidly controlled by powdering with iodoform, after which the cord will dry up and separate satisfactorily.

Infection of the Umbilical Wound.—1. *Acute Umbilical Cellulitis (Omphalitis).*—In rare cases shortly after separation of the cord there occurs reddening and edema of the skin around the navel, which quickly spread over the abdomen and down towards the pubes. Seropurulent or hemorrhagic discharge takes place from the navel. Pyrexia, abdominal distension, and loss of appetite, generally occur. Sometimes abscesses form, and occasionally gangrene and peritonitis lead to a fatal termination. Treatment consists in the use of fomentations and the opening of abscesses when present.

2. *Acute Umbilical Arteritis.*—The extension of septic material from the umbilicus may take a course along the umbilical arteries, the vessels may contain septic thrombi, and the pus may spread along the course of the arteries to the bladder. Occasionally there ensues a general pyæmia with pus in the joints, kidneys, and bones.

The diagnosis is not easy in the early stages, nothing but a little thin pus exuding from the navel, but sooner or later grave symptoms of septic poisoning set in.

3. *Acute Umbilical Phlebitis.*—This affects the umbilical vein, which becomes thrombosed and may contain pus. The liver shows acute inflammation and often multiple abscesses. Peritonitis and septic lenticulo-pneumonia are common. The spleen is enlarged and soft, and embolic foci are found in the kidneys. The diagnosis of phlebitis is usually made clear by the onset of jaundice, which increases and may become very marked. In premature children this illness may give rise to very sudden collapse, while in stronger children high fever, with vomiting and diarrhoea and frequently cerebral symptoms, supervene. The diagnosis has to be made from acute gastro-enteritis, pneumonia, and meningeal hæmorrhage. Umbilical phlebitis is nearly always fatal, but when the artery is attacked, patients occasionally recover. Erysipelas of the umbilicus is referred to in the article on Erysipelas.

GENERAL TREATMENT.—These diseases should be prevented by careful attention to the umbilicus at the time of separation of the cord. Once the condition has set in, antiseptic dressings must be applied to the umbilicus, stimulants and saline injections must be given in collapse, and bromide and chloral by the rectum if convulsions occur (see Chapter I. Diseases of the New-Born).

Septicæmia and Pyæmia in Older Children.—The usual conditions found in older children are—

1. An infected wound such as an infected compound fracture.

2. Acute disease of the middle ear is a common cause of both septicæmia and pyæmia in children.

3. Acute septic epiphysitis and osteomyelitis. This is a very frequent cause in children. Thrombosis of the veins, with subsequent septic disintegration of the clot, is especially liable to occur in acute diseases of bone, because the veins are enclosed in bony canals and do not collapse with pressure in the same way as veins in other situations.

4. In infective endocarditis there is always a condition of general septicæmia.

These conditions are fully dealt with under their respective headings.

ERYSIPELAS.

Erysipelas is an acute infectious disease, due to the *Streptococcus pyogenes*. The term is applied to an inflammation of the skin, the infection being confined to the lymphatic spaces just below the epidermis; it is usually unaccompanied by any great swelling of the tissues, except in such situations as the eyelids, scrotum, or larynx. The affected skin is of an intense red colour, and there is a well-defined edge, which is slightly raised above the surrounding healthy skin. In addition petechial hæmorrhages may occur into the inflamed area of skin. The disease spreads rapidly, usually by continuity, and large areas of skin may be thus involved.

SYMPTOMS.—The incubation is variable, from three to seven days. One attack does not protect from another; in fact, it increases the liability to a second attack.

Erysipelas in infancy may be divided into erysipelas neonatorum and erysipelas of infants. *Erysipelas neonatorum* is a very fatal disease. It is often umbilical in origin, the streptococcus entering the tissues through the umbilical slough. Local redness rapidly spreads over the abdomen, and, in some cases, into the limbs. The temperature is high (103° to 105° F.) and the constitutional symptoms severe. The disease is often ushered in by a convulsion. Sloughing of the skin around the umbilicus may occur; infection of the umbilical vein is common, leading to multiple abscesses in the liver, and in many cases peritonitis follows. In other cases the point of entry is an abrasion in the napkin area. These cases are not so fatal as those of umbilical origin.

Vaccination is responsible for a few cases. The streptococcus may be invaccinated, and the disease will then start on the third day, or it may be accidentally introduced at some subsequent period.

TREATMENT.—Prophylaxis is of the utmost importance. Cleanliness in dressing the umbilicus and frequent cleansing of the buttocks are important. If the mother has any symptoms of puerperal fever, the child should be taken from her and fed with the bottle. Infants nursed by mothers with puerperal sepsis may develop erysipelas. A suitable food must be found for the child, and small doses of brandy may be required to maintain its strength. Locally ichthyol ointment, 10 per cent. in lanoline, may be used. If there is any tendency to sloughing around the umbilicus, 5 per cent. peroxide of hydrogen should be syringed frequently into the wound.

Erysipelas in infants is not so fatal a complaint as when it occurs in the first two months of life. There is usually a definite point of entry. Occasionally it follows the operation for circumcision. The temperature is raised and the inflammation of the skin is very similar to that seen in adults. Owing to the more delicate structure of the infant's skin, hæmorrhages often occur. After four or five days the temperature falls, the redness and swelling diminish, and a brown scaly desquamation of the skin takes place.

PATHOLOGY.—Sections of the inflamed skin show that the cocci lie chiefly in the lymph spaces just below the skin, and are most abundant in the zone of spreading inflammation. The tissues are swollen with serous fluid, in which pus cells are present. Generally the spleen is enlarged, and is soft and dark red in colour. Parenchymatous changes may be found in the kidneys and liver.

DIAGNOSIS is usually easy. The raised temperature is associated with the inflamed area of skin with its definite, raised spreading edge.

PROGNOSIS is very grave in the first three months of life, and when the navel is the point of entry, the disease is almost uniformly fatal. In later infancy and childhood recovery is the rule, even in those cases in which a large area of skin is affected.

TREATMENT must be local and general. The best local applications are gauze compresses wrung out of hot 1 in 60 carbolic lotion. Ichthylol, 10 per cent. in alcohol, may be used. If the disease shows a tendency to spread, the healthy skin should be painted with a strong solution of nitrate of silver or with the liniment, $\frac{1}{2}$ oz. inch from the inflamed surface. The object of this is to excite a leucocytosis, which will destroy the organisms as they reach this zone. For the high fever the patient may be sponged with tepid water, or antipyrin (3 grains) may be given to a child of four years old. Small doses of iron and quinine are valuable as tonics after the attack.

OTITIS MEDIA AND ITS RESULTS.

Acute inflammation of the middle ear is a very frequent affection in infancy and childhood. It is one of the commonest causes of obscure pyrexia in children. The middle ear consists of the tympanic cavity and its contents, the mastoid antrum, the mastoid cells, and the Eustachian tube. The upper part of the tympanic cavity is called the "attic." From the attic the aditus leads to the mastoid antrum and cells. At birth the tympanic cavity is lined by embryonic mucous membrane, and is filled with a jelly-like substance. This soon becomes absorbed. The Eustachian tube is wider and more patent than in the adult. The petrosquamous suture, which crosses the roof of the tympanum, is patent in infancy. Through this suture vessels pass from the dura mater to the tympanum, and there is thus free communication between the lymphatics of the middle ear and those of the middle fossa of the cranium. Infection of the middle ear through the Eustachian tube is therefore more easy than in adults, while the communication with the brain makes the cerebral complications of otitis media more common in infants.

ÆTIOLOGY.—The usual path of infection in otitis media is through the Eustachian tube, and the disease is generally preceded by an infective process in the naso-pharynx.

The most frequent causes are:

1. An acute post-nasal catarrh, especially if associated with adenoids.
2. Acute specific fevers, such as measles, scarlet fever, hooping-cough, typhoid, malpox, diphtheria, influenza, and pneumonia.
3. The entrance of fluid through the Eustachian tube in nasal douching or bathing. Fluid may also be forced up the tube during coughing or vomiting.

Rarely the middle ear may become infected in other ways. Occasionally the inflammation in cerebro-spinal meningitis may spread outwards into the ear. Direct wounds of the tympanic membrane may be produced by the insertion of a foreign body, and may infect the middle ear. Lastly, suppurative otitis media may be a part of a general pyæmia.

The actual cause of the inflammation is microbial. Usually staphylococci are

present, but pneumococci and streptococci are common, and other micro-organisms have been frequently observed.

SYMPTOMS.—The onset of acute otitis media is sudden. In infants and young children earache is always a symptom of middle-ear inflammation. Usually there is pyrexia, which may be extreme. In tiny babies there is continual crying and restlessness, and their hands are constantly put up to their heads. The child cannot lie on the affected side. Sometimes there is marked tenderness of the affected ear, and the baby cannot bear to have it touched; but in many cases in infants pain and tenderness are not marked. In severe cases there may be convulsions, head-retraction, and vomiting, symptoms which closely simulate acute meningitis. In other cases the symptoms may be gastro-intestinal in character, marked drowsiness and apathy, with occasional vomiting, and more or less diarrhoea and wasting may be the main features. In all cases of obscure pyrexia, without evidence of disease sufficient to account for it, the tympanic membranes should be examined. Generally the membrane is red and bulging, but in infants the drum may look almost normal, yet pus may exist in the middle ear, and produce the most alarming cerebral symptoms, which disappear on incision of the drum and escape of the pus.

In older children the symptoms are much more characteristic. Earache is present, accompanied by deafness and pyrexia. Pain is usually sharp and paroxysmal, and may radiate up the side of the head and downwards into the neck. The pain is usually worse at night, and often prevents the child from sleeping. The temperature varies from 100° to 103° F., and severe headache, restlessness, and delirium often occur. One ear only may be affected, but as a rule in children both ears are involved. The appearance of the tympanic membrane varies very much. In infants, as has been stated, the membrane may be nearly normal in appearance in certain cases, but as a rule rapidly becomes congested, and vessels may be seen radiating over the drum. Later on the membrane becomes a dull red, and bulges into the external auditory meatus. If recovery is going to take place without perforation, the red colour gradually subsides, and the membrane is left dull and lustreless. If, however, perforation is imminent, a yellow spot is frequently seen upon the membrane at the point when the bulging is most marked, and sooner or later a purulent discharge appears at the external auditory meatus. The course of acute inflammation of the middle ear is rapid; the condition either subsides or passes on to acute suppuration with perforation of the membrane in five or six days. After perforation has taken place, the intense pain disappears, and the pyrexia subsides, though not always immediately. The complications of acute suppuration in the middle ear are rare. Mastoid disease is very uncommon unless the membrane has perforated; if, however, pain and pyrexia continue when there is a free purulent discharge from the external ear, it usually means that the inflammation has extended to the mastoid or to the cranial cavity.

Meningitis also is a very rare complication of acute otitis media, and when it does occur it is usually infants that are affected. Occasionally some paresthesia of the limbs is present, when the tension in the tympanic cavity is greatest; but it usually passes off after perforation of the drum, and is completely recovered from.

The **DIAGNOSIS** has to be made from meningitis, and usually this is somewhat difficult in children. The appearance of the membrane and the absence of any changes in the cerebro-spinal fluid should, however, make the condition clear. It

is important, especially in infants, not to mistake pulmonary and gastro-intestinal symptoms for the primary disease in the middle ear.

PROGNOSIS.—In acute otitis media recovery is usually complete as regards hearing, and the inflammation may completely subside. If adenoids are present, it is possible that relapses will take place, that the drum will become perforated, and that eventually a condition of chronic middle-ear suppuration will ensue. If the acute otitis media is a complication of one of the acute specific fevers, the prognosis is not so good, because perforation of the drum and chronic suppuration frequently result.

TREATMENT.—If there is much pain and fever, the child must be put to bed, a liquid diet given, and the bowels opened with calomel. The ears should not be syringed, nor must inflation of the Eustachian tubes be practised. A nasal douche should not be used, for all these measures may result in forcing the inflammatory material in the middle ear into the mastoid antrum. If adenoids are present, they should be removed after the acute attack has subsided. Paracentesis or incision of the tympanic membrane may be urgently called for if there are any cerebral symptoms, such as drowsiness, vomiting, head retraction, or convulsions. It should certainly be performed without delay if the patient is an infant, or is suffering from an acute specific fever. An inspection of the tympanic membrane will generally give some indication as to whether paracentesis is necessary, but it must never be forgotten that in infants pus may be present in the middle ear with a drum of nearly normal appearance. In all cases of doubt it is better to perform this slight operation.

Chronic Middle-Ear Suppuration.—This is the result of acute suppuration of the middle ear, and is due to neglect of treatment, so that the tympanum becomes infected by other micro-organisms, or more commonly the perforation is placed in such a situation that there is insufficient drainage. Unhealthy surroundings, anemia, and rickets, will also tend to prolong the disease; while local conditions in the ear, such as the presence of adenoids, the formation of aurial polypi, or widespread destruction of the tympanic membrane, will also tend to make the disease become chronic.

Symptoms.—After the acute stage of otitis media has passed away, the treatment of the discharge from the ear is very frequently neglected; the deafness, especially when only one side is affected, is not troublesome, and the discharge may go on for years without producing any urgent symptoms. In many cases the aural affection may be entirely forgotten until the patient is seized with some intracranial complications or with septic thrombosis of the lateral sinus. The changes which take place in the tympanic cavity are important. Very often caries of the ossicles results, and in some cases chronic suppuration occurs in the region of the attic, when the upper and posterior part of the tympanic membrane is perforated. Disease of the bony walls of the tympanic cavity is common, and granulations or polypi spring from the diseased structures. Polypi may grow to a large size, and even protrude through the external auditory meatus. They are usually a sign that caries of the walls of the tympanum are present, and frequently that mastoid disease exists.

Complications.—The chief complications of chronic middle-ear suppuration are as follows:

1. *Acute Inflammation of the Mastoid Process.*—In an infant the mastoid is rudimentary, and is in consequence not so frequently affected as in older children. The pus, too, often forces its way through the squamo-mastoid suture. The symptoms of acute mastoiditis vary according to the virulence of the infection. As a rule there is a history of a well-marked suppuration in the middle ear of several weeks' duration, and, in spite of free drainage, pain is experienced over the mastoid process, and pyrexia continues. On examination the auricle is displaced downwards, and projects away from the head in a downward and forward direction. Palpation over the mastoid elicits tenderness, and there is as a rule discharge of pus from the middle ear. The proper treatment is to open and drain the mastoid process.

2. *Chronic Disease of the Mastoid Process.*—This may exist for a long time without producing any other symptoms than some headache, dizziness, or neuralgia, provided that the drainage from the mastoid process into the external meatus is free. The temperature remains normal provided there is no retention of pus. In cases where a chronic otorrhea does not clear up in spite of careful treatment, chronic disease of the mastoid should always be suspected. In these cases it is better to do what is known as the "complete mastoid operation," the tympanum, the attic, the antrum and mastoid process, being converted into one large cavity, which drains into the external auditory meatus.

3. *Facial Paralysis.*—This is a not uncommon complication of chronic mastoid disease, and is due to caries of the bony canal containing the nerve. It may also be due to injury during the course of operation. When it is due to acute middle-ear suppuration prognosis is hopeful, and the facial paralysis will disappear on relief of the tension. When, however, it is due to caries of the Fallopian canal, to tuberculous disease, or to injury during an operation, prognosis is very unfavourable, and permanent facial paralysis ensues.

4. *Intracranial Abscess.*—In cases of chronic disease of the middle ear which is left untreated, the septic process often spreads in a quite gradual and insidious manner into the cranial cavity. Three main varieties of intracranial abscess occur:

First, *extradural abscess.* In this the collection of pus lies between the dura mater and the bone, generally in the middle fossa of the skull above the tympanum. Sometimes there may be no symptoms of extradural abscess, and it may be only discovered while doing a complete mastoid operation. As a rule, however, there is a severe and persistent headache, and a suggestive sign is the discharge of a large quantity of pus from the external meatus, the extradural abscess draining directly outwards. The abscess must be opened and drained.

Secondly, *temporo-sphenoidal abscess.* The symptoms of abscess of the brain may be divided into an initial stage, where there is headache, pyrexia, and vomiting; then there is a stage of latency, during which time the patient may have headache and be out of health, but able to get about and do his work for a variable time, lasting from a few days to several months. Then, more or less suddenly, symptoms of intracranial pressure occur. These are headache, vomiting, and optic neuritis, a subnormal temperature, and a slow full pulse, between 50 and 60 beats per minute. Very often there is a marked change in the mental condition of the patient, who becomes dull, slow, and increasingly drowsy. In cases of temporo-sphenoidal abscess there may be paresis of the opposite side of the body, and it situated on the left side, aphasia. In cases of cerebellar abscess ataxia, facial paralysis, and weakness of the muscles, are frequently seen on the side homologous

in the abscess. If the abscess is not opened, death sooner or later occurs from pressure on the respiratory centres, the occurrence of meningitis, or general septicæmia.

5. *Meningitis* is a comparatively rare complication of acute otitis media, and then occurs only in infants. It is usually a sequel of chronic disease of the middle ear or mastoid process. The chief symptoms suggestive of meningitis in a case of chronic otitis media are restlessness, mental irritability, pyrexia, and headache. The symptoms often come on gradually, and the mental changes are the first noticed. Vomiting and dizziness occur, and irregular fits and twitching of the limbs are common. Optic neuritis is not often found, owing to the rapid course of the disease. The headaches increase, and the patient generally screams with pain. The pulse is usually rapid and irregular, the patient soon falls into a state of complete unconsciousness, and dies in the course of a few days.

6. *Septic Thrombosis of the Lateral Sinus*.—If the floor of the tympanic cavity becomes carious, the jugular vein, which lies just below it, may become thrombosed. In chronic mastoid disease the infection will spread to the lateral sinus, which lies on its inner surface. Involvement of the sinus is usually a gradual process; the wall of the sinus gradually becomes inflamed, and thrombosis takes place within it. The thrombus itself is then invaded by micro-organisms, and softens, so that portions break away and are discharged into the jugular vein. As a rule this produces recurring rigors and an irregular temperature, but the patient's mental condition remains clear. If the neck is palpated, thrombosis may be made out in the jugular vein, or a thickening of the tissues around may be apparent. If nothing is done the symptoms become those of pyæmia, and abscesses of the lung, pericarditis, suppuration in the joints, and enlargement of the spleen, occur, and death takes place from exhaustion. The treatment is to remove the infected clot from the sinus after ligation of the jugular vein.

ACUTE EPIPHYSITIS.

SYNOPSIS.—Acute septic epiphysitis; Panosteitis; Osteomyelitis; Necrosis; Periostitis.

This acute disease of bone is one of the most serious met with in childhood. It often ends fatally; and even if recovery takes place from the acute stage, the subsequent injury to the bone entails a long and exhausting illness. The condition is essentially a septic infection of the soft vascular tissue between the epiphysis and diaphysis of a growing bone. It is therefore confined to the period of growth of the bone, and occurs only in childhood and youth. Frequently a history of an injury to the bone affected is obtained. The injury may be due to a strain, a blow, or a fall. It is probable that the result of injury is the formation of a small clot in the rapidly-growing vascular tissue, and that this, becoming infected by pyogenic cocci circulating in the blood, is found by them a suitable abode, and their multiplication results in an abscess. The subsequent history of the case depends partly on the virulence of the infecting organism, and partly on the position of the abscess in the circular disc of growing bone. If the organism is virulent, the child dies in a few days of septicæmia or pyæmia, pericarditis being extremely common. If the patient is able to localize the infection to the bone affected, the subsequent history depends on the position of the small abscess. If it lies near the periphery of the growing diaphyseal disc, it will probably reach

the periosteum of the bone, and a collection of pus will form between the periosteum and bone (acute periostitis, subperiosteal abscess). If the primary focus lies near the centre of the diaphysis, the pus will find its way in one of two directions. First, and most extensively, the whole of the medulla of the diaphysis becomes infected (acute osteomyelitis). In rare cases the abscess perforates the epiphysis itself and opens into the neighbouring joint (acute epiphysitis, acute septic arthritis of children). In other cases pus may be found both within the bone and beneath the periosteum. The result is generally the death of the shaft of the bone, with much limitation of growth of the limb, and if the joint is affected it usually becomes completely disorganised.

Ætiology.—The disease is commonest between eight and twelve years of age, but tiny babies may suffer. It is rare after eighteen years of age. Males are more frequently affected than females, probably owing to their greater liability to injury. The conditions necessary for the development of the disease are—

1. *Lowered Vitality.*—An attack of some acute specific fever, such as measles, influenza, or scarlet fever, has frequently occurred some time before the onset of the disease.

2. *The Presence of Micrococci in the Blood.*—It is assumed that these gain entrance through a small boil or skin abrasion or through the tonsils or naso-pharynx. Possibly they may also enter from the intestine. In dogs feeding on putrid meat will cause a simple fracture of bone to suppurate.

3. *A slight injury to the growing end of a bone determines the point of attack of the micrococci by providing them with a suitable nidus.* The organisms found are staphylococci, and more rarely streptococci or pneumococci. A very similar but much more chronic and less virulent infection is sometimes set up by the *B. typhus*.

Symptomatology.—The epiphyses affected, in order of frequency, are those of the upper end of the tibia, the lower end of the femur, the upper end of the femur, and the upper end of the humerus. More rarely the epiphyses about the ankle and elbow joints may be attacked. After a slight injury the child is suddenly seized with severe pain near a joint. In babies a convulsion, and in older children a rigor, often takes in the disease. The temperature rises to 103° to 105° F., the child is pallid, and delirium is common. Attempts to move or touch the affected limb make the child cry out with pain. The local signs depend on the situation of the disease. In the most common situation—i.e., the epiphyses round the knee-joint—the skin may show a reddish blush, and some slight oedema may be present. On deep pressure over the shaft of the bone near the epiphysis, exquisite tenderness can be elicited. Septic poisoning becomes more and more marked, and the heart should always be carefully auscultated for pericardial friction. Death may ensue in coma in a few days. In the less virulent cases the whole limb may become tense and red. Involvement of the neighbouring joints must be carefully watched for; there is usually a serious synovitis before the perforation of the pus into the joint actually takes place. In other situations—e.g., the upper epiphysis of the femur—the local signs may be very slight owing to the thickness of the surrounding tissues. The joint may be freely movable, until the synovial membrane itself has become affected, and it is rare to find any redness or oedema of the parts around the hip-joint. Here the only local symptom is pain. Septic arthritis of the hip-joint is a common result of the involvement of this epiphysis.

PATHOLOGY.—When examined after death, it is rare to find the lesion limited to the diaphyseal diast. Occasionally, however, in very acute cases, there is seen a small abscess in this situation. The epiphysis may be found partially detached from the shaft, or the periosteum raised by a brownish purulent fluid. Pericarditis, pleurisy, and septic abscesses, are found in these rapidly fatal cases. In the cases which become chronic there is usually necrosis of the whole shaft of the bone. The dead mass becomes surrounded by an invaginating sheath of newly-formed bone, and is then known as a "sequestrum." At certain points where the periosteum and soft tissues have been destroyed completely, no formation of new bone can occur. The apertures thus left in the casing of new bone (involucrum) around the sequestrum are known as "cloaca." Purulent material from the centre of the bone collects at these cloaca and makes its way to the surface, so that sinuses are left communicating with the interior of the bone. If by removal of the dead sequestrum healthy granulations can be induced to form, and the suppuration ceases, the sinuses will close up, and a useful limb may result. If, on the other hand, chronic suppuration continues, the patient becomes progressively weaker, and amyloid disease may supervene.

DIAGNOSIS.—In the first year of life the symptoms are often at the beginning ascribed to meningitis or gastro-intestinal disease. Especially is this the case with the more deep-seated epiphyses, such as the head of the femur, where a correct diagnosis is most difficult owing to the absence of local physical signs and the difficulty experienced by the small patient in indicating the seat of the pain. Later, the most serious disease is often mistaken for acute rheumatism; such an error in diagnosis is extremely serious for the patient. The pain and swelling in the affected joint, with perhaps a pericardial friction sound, simulate an attack of acute rheumatism to some extent. In the latter disease, however, many joints are involved, and the pain flies from joint to joint. Moreover, the swelling and tenderness are not limited to the joint in acute epiphysitis, and the most tender point is upon the shaft of the bone. Rigors and delirium are very uncommon in acute rheumatism, and the temperature is seldom so high as in acute epiphysitis. It is most important to make a correct diagnosis at once, as the only hope of saving the patient's life lies in early incision. Later on, as the skin becomes red, the complaint may be confounded with cellulitis or erysipelas. In the latter case there is no tenderness on deep pressure or on nipping the bone between the fingers, nor are the constitutional symptoms so severe. The pericardial hæmorrhages of scurvy may simulate the complaint, and cases of scurvy were formerly operated upon and called "acute hæmorrhagic periostitis." In both there is a raised temperature and a tender swelling at the end of a long bone. In scurvy, however, the tenderness is rarely limited to one bone, and the swelling itself is often bilateral. The age of the patient (eight to fourteen months), the marked anæmia, and the sperry condition of the gums, should in most cases serve to make the diagnosis clear.

The **PROGNOSIS** is very grave. In the virulent cases the patient may die in three or four days of septicæmia, without the small focus in the epiphysis having been even suspected. If, however, the abscess is opened and free drainage secured, the septic intoxication as a rule rapidly subsides, though this is not always the case. The growth of the limb will nearly always be affected, even in cases which recover, and if chronic suppuration persists the limb may have to be sacrificed.

TREATMENT.—A longitudinal incision must be made at once down to the epiphysis. The finger should be introduced, and the extent of periosteal detachment should be ascertained. If pus is found beneath the periosteum, it must be washed out; and if the periosteum is widely detached, other incisions may have to be made to secure free drainage. The question as to whether the bone should be trephined and the medulla exposed has to be faced. If pus has been found beneath the periosteum, the case may be one of acute periostitis without any osteomyelitis, and by exploring the interior of the bone the medulla is exposed to infection. It is, however, safer to explore the interior of the bone in all cases, as failure to relieve the osteomyelitis will cause death from pyæmia. Moreover, if osteomyelitis is not present, exploration of the medullary cavity is not likely to lead to infection, as the purulent material is no longer under tension. If no pus is found beneath the periosteum, the bone must be trephined, the suppurating medullary tissues must be removed by scraping, and the opening in the shaft be enlarged. In cases where it is found that the whole shaft of the bone is completely necrosed, removal of the whole dead shaft of bone has been suggested. This is possible in the case of the tibia, the fibula being left as a support, but in the case of the femur it is impracticable. This, however, makes the operation a much more serious one, but may save the patient a long and trying illness. Subsequently necrosed bone and sequestra must be removed as soon as they become separated, and in cases where chronic suppuration is undermining the strength of the patient amputation will have to be considered.

The general treatment is most important. A full, easily digestible diet, strychnine, stimulants, and quinine, must be used. In cases where chronic suppuration persists, and the patient is otherwise in fair condition, a vaccine should be prepared, and the patient injected with the organisms obtained from the sinus.

GONORRHOEA.

Children are attacked by the gonococcus in the genital tract, the conjunctiva, the joints, and the mouth, while in some cases a general gonorrhoeal pyæmia or septicæmia may follow. The affection of the genital tract produced by the gonococcus has been discussed under Vulvo-Vaginitis, in the chapter devoted to the Genito-Urinary System (Chapter XII, p. 659).

Gonorrhoeal Conjunctivitis.—The gonococcus is the infecting organism in the majority of cases of ophthalmia neonatorum, and is responsible for nearly all the graver cases. Sometimes the eyes are infected by the maternal vaginal secretion during labour, but usually the infection occurs immediately after birth, because the infant's eyes are usually tightly closed during delivery. Occasionally the eyes may become infected some days subsequently to birth, and cases may occur in older children as a complication of vulvo-vaginitis of gonorrhoeal origin.

The incubation period is from two to three days, when slight irritation of the eyelids is noticed. Inflammatory oedema of the eyelids rapidly comes on, and the lids become greatly swollen and stick together, so that they are separated with difficulty. Copious purulent secretion takes place from the conjunctiva, which becomes scarlet and swollen. In cases which are vigorously treated and progress

invariably, the swelling gradually subsides and the discharge becomes mucopurulent, and the condition clears up in from one to four weeks. In neglected cases or in ill-nourished infants the cornea is liable to become infected. It becomes cloudy, ulceration takes place and may lead to perforation of the cornea, evacuation of the aqueous humour, and prolapse of the iris. In such cases permanent blindness ensues, and gonorrhoeal ophthalmia is responsible for over one-third of all cases of blindness.

The **DIAGNOSIS** is made by examination of the purulent discharge. The gonococci are readily stained by methylene blue, and occur in pairs upon the surface of the polymuclear leucocytes. As a rule, in preparations only a proportion of the cells have gonococci attached to them. The gonococcus is a Gram-negative organism.

The **PROGNOSIS** is good, provided treatment is begun before the cornea is invaded. Efficient treatment usually protects the cornea, but a guarded prognosis should be given in weakly or premature infants. Permanent blindness often results, but in some cases opaque patches only occur in the cornea.

PROPHYLAXIS is all important in averting this grave disease. If the mother suffers from a purulent vaginal discharge or leucorrhœa, antiseptic vaginal douches should be given before birth, and the greatest care should be taken to wash the maternal secretions from the eyes before the lids are opened. In all such cases Codd's method of dropping 2 minims of a 2 per cent. solution of nitrate of silver into each of the infant's eyes should be adopted. This sometimes causes irritation of the conjunctival sac, and a 1 per cent. solution is now generally preferred, and appears to be effective. In the case of vulvo-vaginitis in elder girls, the strictest care should be exercised to prevent infection of the eyes by the child's finger or by towels.

TREATMENT must be prompt, and on the first sign of reddening of a newly-born baby's eye, active treatment should be undertaken. The conjunctival sac must be thoroughly irrigated with warm boracic lotion. The lids should then be everted and painted once with a 2 per cent. solution of silver nitrate. The conjunctiva should then be irrigated again with normal saline solution to prevent caustic action of the silver nitrate. After this, warm compresses should be kept over the eyes to prevent adhesions of the lids and to reduce œdema. While the acute inflammation lasts, the eyes must be irrigated every two hours, and after each washing a few drops of 10 per cent. protargol should be instilled into the conjunctival sac. If the cornea becomes hazy or shows signs of commencing elevation, a few drops of atropin (2 grains to the ounce) must be applied. If only one eye is affected, the other eye must be protected by a suitable shield, and a drop of 1 per cent. solution of silver nitrate placed daily in the healthy conjunctival sac.

Gonorrhœal Arthritis.—Arthritis due to the gonococcus is never primary, but is a sequel of a gonorrhœal infection of either the vulva or the conjunctiva. It occurs as a rather infrequent complication of vulvo-vaginitis (q.v.). In the case of gonorrhœal ophthalmia, the arthritis nearly always makes its appearance three weeks after the onset of the ocular inflammation. The joints most commonly affected are the knees and wrists. R. C. Lucas describes two types of this condition—a mild variety of synovitis without reddening of the skin, which quickly subsides, and a severe form, in which there is much peri-articular inflamma-

tion. In the latter form recovery is very slow, and the arthritis lasts for weeks. Deutchmann in 1890 demonstrated gonococci in the synovial fluid from the inflamed knee of an infant, three weeks old, suffering from ophthalmia. Gonococci were also found in the pus from the conjunctiva. The joints rarely suppurate, and resolution takes place even in severe cases. In those that do suppurate a mixed infection of the joint probably occurs. Thus Griffon in a case found both gonococci and staphylococci in a suppurating joint of a child suffering from gonorrhoeal ophthalmia.

The **DIAGNOSIS** has to be made from syphilitic joint disease in young children. Syphilitic epiphysitis is most common about the end of the first year of life, while the arthritis of gonorrhoeal origin occurs as a rule during the first month.

A mistaken diagnosis of acute rheumatism is little likely in infants with ophthalmia neonatorum, but may be made in older girls with vulvo-vaginitis.

The **PROGNOSIS** is on the whole good. Most of the cases do well. In cases of mixed infection with streptococci and staphylococci, pyæmia and death are not uncommon.

TREATMENT.—Thorough treatment of the ophthalmia or vulvo-vaginitis to which the arthritis is a sequel is most important. When once a cure has been obtained of the primary condition, the arthritis usually subsides. As regards the joints themselves, in little babies all that is required is to wrap them up in cotton-wool and bandage them lightly to protect them from injury. In older children the joint should be supported by a splint if painful, but it should not be kept in a fixed position for too long a period, as adhesions are likely to form. In obstinate cases good may often be done by the use of gonococcus vaccine. Bier's venous congestion method may also be used for older children. Massage and passive movements must be persevered with if stiffness and muscular wasting result from the arthritis.

Gonorrhoeal Stomatitis and Rhinitis.—This is a rare complication in children, and can be diagnosed only by bacteriological examination. The mucous membrane of the nose and mouth are red and swollen, and a small amount of sticky, purulent secretion occurs (see also Chapter IV., p. 141).

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ACTINOMYCOSIS.

Actinomycosis is a chronic infective disorder, produced by the ray fungus, or *Streptothrix actinomyces*. It is now certain that this name is given to a number of affections produced by similar, but not identical, organisms of the streptothrix group. The disease is frequent in cattle, and as a rule gives rise to a local affection of the jaws, tongue, or throat, the parasitic origin of which was first discovered by Billinger in 1877. Later, Penick showed the identity of the disease in man and cattle, and in this country the first undoubted case was described by Acland.

ETIOLOGY.—The parasite belongs to the streptothrix group, and in the pus from the affected area yellowish opaque granules, about a millimetre in diameter, can be seen. Microscopically, the organism stains well by the ordinary basic dyes, and retains the stain by Gram's method. A dense mass of branching filamentous threads can be seen, and isolated segments resembling bacteria may occur. In some cases club-shaped bodies occur; these appearances are more frequent in pus than in the human cases, but are variable, and may be only involution forms of the streptothrix. The parasite has been cultivated on nutrient agar, and the disease has been reproduced by inoculation.

SYMPTOMATOLOGY.—The fungus in many cases reaches the body through a lesion of the digestive or respiratory tract, or through the skin, and is probably conveyed by grains of wheat or barley, or by grasses. It is common in stablemen and gardeners, while in many cases a history of chewing cane of barley or oats can be obtained; further in a large number of recorded cases, remains of a cereal (grass-seeds or oat-grains) have been demonstrated in the lesions. The disease is three times as common in men as in women, while in children it is rare, only two patients of thirty-nine being under fifteen years of age.

Pulmonary Actinomycosis.—This is a chronic disease of the lungs, characterized by cough, fever, wasting, and a mucopurulent expectoration, which is sometimes offensive. The physical signs are generally unilateral, and are those of a chronic destructive process, usually at the base of the lung. The characteristic granules are to be found in the sputum, and the parasite may readily be demonstrated by Gram's method of staining. The pleura may become invaded, and empyema is not an infrequent complication. The presence of subcutaneous abscesses or of erosion of the vertebrae or ribs should, in a case of chronic local lung affection, lead to a careful search for the presence of actinomycetes in the discharge from the lesions.

Alimentary Actinomycosis.—The fungus has been found in the cavities of rumen teeth, and the jaw, which is so commonly affected in cattle, may also be attacked in man. A chronic enlargement of the jaw, with invasion of the tongue and floor of the mouth and the skin of the cheek, occurs, and often simulates a sarcoma. Ulceration of the intestine may be found, and several cases of appendicitis, due to the streptothrix, have been recorded. Abscesses in the liver occur. The symptoms are not characteristic of this form of infection, and are similar to those produced by more common lesions of the alimentary tract. The diagnosis is to be made only by finding the organism in the discharges.

Cutaneous Actinomycosis.—When the skin is invaded, either primarily or secondarily to a lesion of the alimentary or digestive tract, the appearances are somewhat characteristic. Raised, pulpy, fleshy masses of a dark red or purple colour occur, and in them small fistulous openings are found discharging pus in which the yellow granules can be found.

Generalized Actinomycosis.—The disease is usually chronic and local, but in certain cases the fungus finds its way into the vascular system, and metastases occur in other organs. Abscesses have been recorded in the brain, and in some cases the symptoms are suggestive of pyæmia.

PATHOLOGY.—The result of inoculation by the streptothrix is a gradual invasion of the surrounding tissues. Inflammatory areas coalesce and unite to form large granulating areas. In certain situations (e.g., the jaw) there is a very

marked proliferation of fibrous tissue around the focus of disease. Finally, expulsi^on takes place. Metastases may occur from involvement and perforation into a vein. In the liver the appearances of the lesion are peculiar. A portion of the organ is invaded by alveolar honeycombed abscesses, like sponges soaked in pus. The individual foci of inflammation are small, and the alveolar framework consists of fibrous tissue. In the lungs the lesions may be mistaken for those of tuberculosis.

PROGNOSIS.—Actinomycosis of the skin and superficial structures is curable, but in the case of visceral lesions recovery is rare, the average duration of the disease being about ten months after the first symptoms have been noted.

TREATMENT.—The most satisfactory method of treatment is the administration of iodide of potassium internally. The drug must be pushed, and at least 60 grains per day given. This has proved a curative measure in many cases. Where the disease is superficial, as in the skin or jaw-bone, total excision of the diseased area should be undertaken if possible, while in other cases incision of the abscesses, removal of diseased bone, and local antiseptic treatment, can be used with success.

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MALARIA.

Malaria is a specific infectious disease due to the invasion of the blood by parasites, which belong to the genus *Plasmodium malarie* of the *Haemosporidia*. In children the disease produces acute paroxysms of fever, and also a chronic malarial poisoning.

ETIOLOGY.—The *Plasmodium malarie*, discovered by Laveran in 1881, is introduced into the human body by the bite of mosquitoes of the family *Culicidæ* and sub-family *Anophelinae*, which have themselves become infected by sucking blood from individuals affected by malaria. This was shown by Ronald Ross in India in 1897. Malaria occurs at all ages. It is even possible that the fetus may be infected in utero by parasites passing from the mother's blood through the placenta. In quite young children, however, the disease is easily contracted. Possibly this is because of their thin, delicate skin, which renders them peculiarly subject to mosquito bites. Malarial infections in children are very prevalent in the tropics, and as the children grow up they appear to acquire a relative immunity to the disease. Malarial fevers are widespread all through the temperate and tropical zones. It is rare in cold climates, and is especially common in low marshy regions. The largest number of cases appear in September and October.

There are three main types of malarial fever, according to the species of the parasite causing the infection:

1. Tertian fever, due to the *Plasmodium vivax*.
 2. Quartan fever, due to the *Plasmodium malarie*.
 3. *Adiuvans*-autumnal fever, due to the *Plasmodium innocuatum*.
- Each parasite exists in two separate phases or stages.

1. A stage in human blood, when an asexual cycle of development takes place, thus causing the symptoms of malaria, man acting as the intermediate host.

2. A stage which begins in human blood, where asexual forms of the parasite or gametocytes are formed. These are sucked by the mosquito into its stomach, where fecundation of the female element by the male takes place. This fertilized female element then penetrates the epithelial lining of the stomach, and remains in the stomach wall for seven days, developing into an oöcyst, which ultimately contains a large number of sporozoites. The oöcyst bursts, and the sporozoites pass into the body cavity of the mosquito, reach its salivary glands, and are inoculated into the blood-stream of the next human being bitten by the mosquito. Here such sporozoites may enter a red corpuscle and give rise to either the sexual or the asexual cycle described above.

In the *Plasmodium vivax*, or parasite of tertian fever, the asexual cycle takes place every forty-eight hours, and the febrile attacks occur every other day.

In the *Plasmodium malariae*, or parasite of quartan fever, generation takes twenty-two hours, and the fever occurs every three days.

In pernicious malaria the fever is continuous, the cycles taking place irregularly. The effect produced by the parasites on the red corpuscles varies; in the tertian form the red cell increases in size and becomes paler, while in the quartan form the red cell shrinks and retains its normal colour.

The effect on the red cells in pernicious malaria is not so constant, but this form is at once distinguished from the other forms by the crescentic shapes of the gametocytes.

In both tertian and quartan fevers the sexually differentiated forms or gametocytes are difficult to distinguish from fully grown parasites about to undergo sperulation, but in the pernicious malaria the gametocytes are sausage-shaped or crescentic in type, with a central nucleus. In the male the pigment is scattered through the cell, while in the female the pigment is more aggregated round the nucleus.

SYMPTOMATOLOGY.—When the parasites are first introduced into the blood their number is small, and for a time they produce no appreciable effect. This is the so-called "incubation period," which lasts as a rule about fourteen days, but may be shorter or much prolonged. During this period the parasites multiply in geometrical progression by passing through asexual cycles in red blood-cells, and their increasing number soon begins to bring about a reaction on the part of their host in the shape of the symptoms of fever. These febrile paroxysms coincide with the sperulation of the parasite, and are usually ushered in by a few hours of headache and unpleasant sensations in the epigastrium. There are three stages to the paroxysm, the cold, hot, and sweating stages.

In the cold stage the temperature begins to rise and the feelings of malaise increase. The patient begins to shiver, the face and skin are blue and cold, and in the fully-developed rigor the body trembles so violently as to shake the bed. Nausea and vomiting are common, and headache is not uncommon. The chill lasts from ten minutes to an hour, and the temperature rapidly rises to 106° or 106½° F. As the chill subsides the hot stage is ushered in by intermittent heat flashes. The skin becomes intensely hot, the face and eyes are flushed, and the pulse, though remaining rapid, is full, and the patient may complain of throbbing in the head, and is often restless and delirious. In children convulsions often take place, and in them splenic enlargement is especially noticeable. The hot stage

lasts three to four hours and passes into the sweating stage. Profuse perspiration sets in and the uncomfortable sensations disappear, and within a few hours the temperature returns to normal, the pulse being slow and regular, and the patient usually goes off to sleep. Labial herpes and bronchitis are common during the paroxysm, which lasts in all about twelve hours.

The regularly intermittent fevers are the tertian, where the paroxysm occurs every third day; and the quartan, where the chill comes on every fourth day. Double infections are common; thus a double tertian affection gives daily (quotidian) paroxysms, and is a common type. Double quartan infections give rise to paroxysms on two successive days, with a day of intermission following, while three groups of quartan parasites may occasion quotidian paroxysms. After a few days the paroxysms may cease spontaneously, but, if untreated, the condition may become chronic. These fevers yield at once to the exhibition of quinine.

In the remittent, continued, or astivo-asthenal fevers, the parasite is the *Plasmodium immaclatum*; the period of the cycle of development is forty-eight hours, and these fevers have been called malignant tertian fevers. Owing to the presence of groups of these parasites, the fever tends to be remittent and irregular. The paroxysms are of long duration, often lasting twenty-four hours.

There is severe headache, and nausea, vomiting, and diarrhoea, especially in children, are frequent. A dull drowsy, apathetic state may supervene, and this, in conjunction with the continued fever, may suggest a diagnosis of enteric fever. Manifestations of the gravest character arise in the course of these astivo-asthenal infections. Cerebral involvement is common; delirium and mania, and, in children, repeated convulsions, occur. This may pass into a comatose state, with evidence of increased intracranial pressure, such as full slow pulse and Cheyne-Stokes respiration. The tongue may be dry and coated, and death occurs after a few days. In these cases the cerebral and spinal capillaries have been found crowded with the parasites. In other cases intestinal symptoms may be prominent. Acute vomiting and diarrhoea, with collapse, may be the chief features. Jaundice may occur. Acute bronchitis and a petechial skin eruption are found in the acute cases.

Occasionally, in visceral manifestations, examination of the blood from the ear or finger may fail to reveal parasites. Very often, however, large hyaline cells can be seen containing malarial pigment, and often fragments of red corpuscles, and this appearance is common in the pernicious forms. According to Koplik, the tertian parasite is the one most commonly found among children in New York, the quartan form being only rarely present.

In infants and young children the typical paroxysms are not so common, and are often replaced by some other symptoms, such as vomiting, convulsions, or acute diarrhoea. In cases of sudden fever with an acute splenic enlargement, when accompanying any pulmonary or intestinal symptoms in a district where malaria is common, the blood should always be searched for malarial parasites. Infants of all ages may be attacked, and Howard has recorded a case of a paroxysm in an infant six hours old, whose mother was suffering from malaria.

PATHOLOGY.—In acute cases of astivo-asthenal infections the organs have a slaty-grey colour due to the accumulation of pigment formed by the parasites. These are present in greatest numbers in the spleen and bone marrow, but in certain cases the brain may be the seat of an intense affection, and the capillaries crowded with pigmented parasites. The spleen is enlarged and softened and of a

slay colour, and the liver is enlarged. The bone marrow is often extensively pigmented, and in chronic cases there may be a great increase in the red bone marrow. In other cases this may fail to respond, and fatal aplastic anemia result.

DIAGNOSIS.—While malaria in adults can often be easily diagnosed by the symptoms, in children this is far more difficult. The only way to make a certain diagnosis is by finding the malarial parasites in the blood. The examination must be made, however, before quinine has been given. The various types of the parasites may be distinguished by the criteria already given. In malaria the leucocytes are reduced in number, while in septic infections they are increased. In making these observations, the peculiarities of the condition of the white cell content of the blood of young children must be remembered.

PROGNOSIS.—The prognosis is good, provided that the child is treated with quinine and can be removed from the malarious district. Relapses may, however, occur, even after long intervals without any symptoms.

TREATMENT.—Prophylaxis.—In a malarious district instruction must be given to protect young children from the bites of mosquitoes. Suitable clothes and fine netting for the face, and mosquito netting at night, must be used.

The general treatment is that of an acute febrile disease. In the cold stage stimulants and a hot bath may be needed, while in the hot stage tepid sponging of the skin and an icebag to the head are useful. The bowels should be well opened by calomel. For infants under two years old, the bisulphate of quinine may be given in water in doses of 1 to 2 grains. If it is vomited, double the quantity may be given *per rectum*. It should not be given hypodermically, as induration of the skin, and often sloughing, are apt to occur.

Older children take without difficulty the tablets composed of chocolate and lactate or sulphate of quinine, which are nearly tasteless. The rectal method of administration may be combined with the oral. Children bear the administration of quinine well, and full doses should be given. Thus, an infant with a sharp attack of malaria requires 8 to 10 grains of the sulphate in the day. In chronic cases with anemia, smaller doses of quinine with iron and arsenic should be employed.

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CHAPTER XXI

CONGENITAL SYPHILIS

A. M. COSSAGE

INTRODUCTION.—*Organism of Syphilis.*—Syphilis is a specific disorder caused by a living contagium, and characterized by a prolonged and chronic course. It has been shown, with a probability almost amounting to certainty, that the contagium is an organism, first described by Schaudinn, which belongs to a higher class than the bacteria, and is known as the *Spirochaeta pallida* or *Treponema pallidum*. Like the other organisms of its class, it has not been grown outside a living body, but it differs from them in that in its natural state it has only been found in human beings, and is directly conveyed from one person to another without the intervention of an intermediate host. It is possible to transmit the disease in a modified, but still recognizable, form to monkeys, a fact which has helped in the elucidation of many obscure points. These higher organisms have a marked point of distinction from the bacterial class in that they are susceptible to the influence of drugs administered to their host, and it is possible by the ingestion of certain chemical substances to kill most of the organisms present in an infected individual and incidentally to cause the disappearance, temporary or permanent, of the manifestations of the disease.

Effects of Infection.—The inoculation of the virus causes first a local lesion, which takes about a month to develop, the later stages being accompanied by swelling of the adjacent lymphatic glands. This is followed, about two months later, by a generalized infection, as shown by eruptions on the skin and mucous membranes. After the lapse of some years isolated local lesions tend to develop in any part of the body, with much destruction of tissue and scarring. At all stages the presence of the virus leads, however, to the development of a "granuloma," which is an inflammatory new growth formed of embryonic cells; these cells are round cells, epithelioid cells, and often giant cells, which are partly of vascular, partly of connective-tissue origin. The new growth is situated near the wall of an artery, but, like a tubercle, has no bloodvessels within itself. It is always associated with changes in the adjacent small bloodvessels, there being inflammatory thickening of the internal and external coats of both arteries and veins. The initial lesion is situated just under the cutaneous epithelium, and as it increases in size tends to ulcerate. The secondary lesions are also mainly superficial on the skin and mucous membranes, but generally only ulcerated in moist situations, such as the mouth or around the naris. The tertiary lesions appear in the deeper structures, grow to a larger size, and frequently end in the death of their central cells and the formation of an abscess or deep ulcer.

Children of all ages are as liable to infection as adults, but, owing to the fact

that the contagium is usually passed from one individual to another during sexual intercourse, cases of acquired syphilis are naturally much rarer in childhood. Infants can, however, be infected by their parents before they are born. This leads to what is known as "congenital syphilis," or improperly as "inherited syphilis," a disease differing in some of its clinical aspects from the acquired complaint. Much controversy has arisen over which parent is to be regarded as the source from which the infantile complaint is derived. There is no doubt as to the power of a syphilitic mother to infect her unborn child, but it has been regarded as impossible for the father to infect the child without first giving the disease to the mother. Against this view there is a long array of clinical facts proving that a man who shows no infective lesions can beget syphilitic children without the mother exhibiting any recognizable sign of the complaint, and that in these families treatment of the father alone may result in the escape of the next child. Finger has discovered that the semen of a syphilitic man may, when inoculated into a monkey, produce a primary sore, although the spirochaeta, which may occur in the testicle has never yet been found in the semen. Hence the possibility of congenital syphilis being derived from the father alone must be accepted. Many who acknowledge this consider that the diseased foetus is certain to infect its mother during gestation, founding their belief on the occurrence of tertiary symptoms in some women who have had no previous sign of syphilis except the bearing of syphilitic children, and, further, on the fact that the suckling of a syphilitic infant by its mother does not lead to her infection, while if another healthy woman suckle the same syphilitic child she may develop a chancre on her breast (Coller's law). There are, however, some possible exceptions to Coller's law and the infectivity of congenital syphilis has certainly been exaggerated. Again, tertiary lesions are not unknown in men when no history of secondary or primary manifestations can be obtained, and their occurrence in some women is no proof that the mothers of all syphilitic infants are themselves affected. Coutts has suggested a probable explanation of the undoubted immunity of these women, that it is due to the absorption through the placenta into the maternal circulation of some chemical product of the growth of the virus in the foetus.

Acquired syphilis in infants and children differs in no way from the disease in adults in its pathology, course, and symptoms. In congenital syphilis, on the other hand, the foetus is probably infected at the moment of impregnation of the ovum, and develops hand in hand, so to speak, with the syphilitic virus. Not unnaturally, therefore, it suffers from the action of that virus much more severely than when there is, at a later age, a gradual spreading of the contagium from a small local lesion. Hence death is a very frequent and early result of fetal syphilis, mostly from the severity of the poisoning; whilst death in acquired syphilis is almost always due to gross lesions of vital organs or to degeneration of the blood-vessels, both of which occur late in the disease.

In severe infections the foetus dies and is aborted. Pregnancy may also be prematurely terminated by so-called "gummatous disease of the placenta" interfering with the proper supply of blood, the disease being confined in practically all cases to the fetal portion of the placenta. In slighter conditions of placental disease the foetus may be born alive, but much below the usual weight. The viscera, which are only affected in the late stages of the acquired disease, are often in congenital syphilis attacked early and severely, causing death either *in utero* or in the first few months of extra-uterine life. In these visceral lesions the spiro-

chests can be found in enormous numbers, a great contrast to their scarcity in the lesions of acquired syphilis. The liver, owing to its situation and great importance in the fetal circulation, is the viscous most frequently affected and a peculiar type of cirrhosis results, where the new fibrous tissue, besides being interlobular, also invades the lobules and penetrates between the individual cells. The growing bones are also often attacked; indeed, Wegner and Taylor state that in dead syphilitic fetuses there is invariably disease at the epiphyseal lines, and an alteration in bone development. This is shown by the presence of a yellow irregular line at the junction of the epiphysis and diaphysis, which on examination with the microscope is found to be due to a proliferation of new cells (i.e., a granulomatous new growth). By this new growth the vessels are pressed on and the circulation interfered with, so that the nutrition of the cartilaginous cells is affected and ossification proceeds irregularly. In some cases death of tissue may result, leading to separation of the epiphysis from the diaphysis.

After the marriage of two individuals, one of whom is syphilitic, the common sequence of events is that the wife has a succession of miscarriages, the fetuses on each occasion being killed by the severity of the infection or by disease of the placenta. This is naturally the more likely to take place where the wife is afflicted with the disease than where the husband alone is concerned. In the vast majority of cases, however, the man is the source of the contagium, and in the largest proportion of these the woman never shows any sign of infection. After several miscarriages, a child may be born at term, which probably dies within a short time of birth, showing syphilitic lesions of the viscera; then, later, children may be born who appear healthy at birth, and only show signs of syphilis four to six weeks afterwards. Though this is the typical course of events, yet sometimes a perfectly healthy child appears between two syphilitic ones. The explanation offered by Coats of the gradual diminution of the intensity of infection seems to the writer the most convincing. Since infection means the transference of the living contagium from the sick to the healthy, differences in the severity of infection probably result more from the condition of the infected individual than of the infector. In ordinary clinical experience there is no difference in the severity of the complaint in adults according as to whether the person from whom they derive infection is in the early or late stage of the disease, although the probability of infection gets less as time goes on. The same rule would be expected to hold with regard to infection of the fetus by the male parent and the progressive diminution of severity that is actually met with is probably due to the absorption of antibodies by the mother from each successive syphilitic fetus, which should gradually increase the immunity of all her cells, including the ova, and so modify the severity of infection for future infants.

SYMPTOMATOLOGY.—Bishes.—It is the exception for syphilitic infants who are born alive at term to show any obvious sign of the complaint. When signs are present at birth, such as wasting or a bullous rash, death nearly always ensues after a very brief period. Generally at birth the child appears well nourished and blooming, but after three or four weeks it begins to waste, to snuffle, and then a rash appears on the buttocks and forehead. Snuffling is one of the most constant of the features presented by the infant with congenital syphilis, and is due to swelling and exoriation of the nasal mucous membrane with a certain amount of viscid discharge. It is accompanied by some broadening of the bridge of the nose, which, however, disappears as the child gets older. The more marked and per-

most deformity caused by destruction of bone arises much later in the course of the disease, if it occurs at all. There is always some obstruction in the nostrils, and this may in rare cases be so great that the baby is unable to suck, and has to be fed by a spoon. Accompanying the snuffles there is usually some laryngitis, so that the infant's cry becomes hoarse. The skin rashes appear a little later, and are generally confined to the buttocks and genitals. They are usually erythematous in nature, and are similar in position and appearance to the rash produced in the delicate skin of many infants by the contact of alvine and urinary discharges. They tend, however, to spread farther down the thighs and legs, and up on to the abdomen, and are less bright, resembling more the colour of raw ham. The chin and brows are sometimes involved, and less frequently the palms and soles. The limited character of the distribution of these rashes affords a strong contrast to acquired syphilis, where the general roseola, almost unknown in the congenital disease, is the earliest and most typical of the skin eruptions. After the erythematous, papular rashes with some coarse desquamation are the most common, while vesicular and bullous eruptions are rare. Bullae are, however, more frequent in congenital than acquired syphilis, and a syphilitic pemphigus is prone to appear at or shortly after birth in severely affected infants. In such cases bullae nearly always are found on the palms and soles as well as elsewhere. The progress of these early bullous rashes is grave, and the infants nearly always die. Associated with the skin eruptions there is generally loss of hair, not only on the head, but also in the eyebrows. Some authors describe a profuse growth of black hair as appearing in some syphilitic babies; this "syphilitic wig," as it has been called, cannot, however, be regarded as a definite sign of the disease, since a precisely similar appearance may be found in children where there can be no suspicion of a syphilitic taint.



FIG. 106.—THE APPEARANCE OF THE FACE IN A SEVERE CASE OF CONGENITAL SYPHILIS.

Sores about the lips are not infrequent, either in the form of mucous tubercles or more commonly as longitudinal radiating fissures or rhagades. These fissures give a very characteristic appearance to the patient, and are often followed by marked scarring. They should not be confused with the common shallow ulcers at the angle of the mouth in weakly children. Mucous tubercles may appear about the mouth, and condylomata about the anus, but are decidedly rare at this period, in contrast to their frequency and profusion in the acquired complaint.

The lymphatic glands may sometimes be found enlarged, but never to the extent observed in the acquired disease, where their involvement is invariable.

Epiphysitis.—It has been pointed out that changes at the epiphyses of the long bones have been found in prematurely born fetuses and in infants dying soon after birth, changes said to be constant by some observers. Clinical signs of epiphysitis,

however, are not specially common, and do not appear until the third to sixth week after birth. A child, who has probably already had smuffles and a rash about the buttocks, may seem to be in pain, and may be noticed not to move one or more of its limbs. Tenderness and swelling may be found about several of the epiphyses of the long bones, and, in some cases one or more of the limbs may be apparently paralyzed, hanging quite motionless and flaccid. Neither the pain nor the tenderness are as a rule very severe, and may be quite absent, and

they seem insufficient to account for the pseudo-paralysis which so often accompanies the epiphysitis. When more than one limb is affected with the paralysis, they are not attacked simultaneously, but at intervals of a few days. With appropriate treatment the condition clears up with extraordinary rapidity, though in severe cases there may be separation of the epiphysis, requiring careful adjustment of the limb in splints to prevent deformity. Syphilitic pseudo-paralysis has to be distinguished from scurvy, infantile paralysis, and acute epiphysitis, all of which may produce somewhat similar symptoms. The age incidence of scurvy and syphilitic pseudo-paralysis is different. Scurvy is very rare before the age of six months, whereas the paralysis of syphilis generally occurs before the second month after birth, and is very rare after the sixth month. Pain and tenderness, as well as local swelling, are more pronounced in scurvy. Further distinction between the two conditions will depend on the presence or absence of other signs of the complaints, and, in the case of syphilis, on the previous history of the child and the history of the mother's former pregnancies. Sometimes it



FIG. 161.—SYPHILITIC EPIPHYSITIS AND PSEUDOPARALYSIS FROM A CHILD AGED NINE WEEKS.

may be necessary to await the results of treatment. Infantile paralysis is also rare at the early age when pseudo-paralysis is commonest. Its onset is more sudden, and is followed by rapid degeneration of the affected muscles, with an early reaction of degeneration to electrical stimulation, and there is an absence of any tenderness or swelling about the epiphyseal lines. The intense pain and tenderness, and the marked constitutional symptoms, are usually sufficient to render the diagnosis of acute epiphysitis easy. The appearances under the X rays are quite different in these complaints, and afford a valuable means of diagnosis.

The bones of the hands and feet may also be attacked by syphilitic epiphysitis, especially the phalanges, but this syphilitic dactylitis is rare before the age of twelve months; after this age it is not so uncommon, and is very difficult to distinguish from tuberculous dactylitis. But X-ray appearances are usually very different. There is swelling about one or more of the proximal phalanges, which frequently goes on to suppuration, and sometimes bone destruction. Suppuration is almost unknown in the earlier epiphysitis described above, but even the long bones may be affected with suppuration at their ends during the later stages of congenital syphilis. The fingers may also be affected with suppuration around the nails, but this onychia is also commoner after the patient has passed the first year.

Constitutional Symptoms.—As might be expected, there is often in the congenital disease a marked influence on the general nutrition of the infant. Wasting frequently occurs and in many cases is extreme. The child becomes anemic, and the complexion often takes a characteristic whitish-brown hue, which has been likened to the colour of coffee on milk. Naturally the wasting is more extreme where the abdominal organs have been affected and any considerable degree of marasmus is a sign of serious import. Sometimes wasting is the only sign of the syphilitic taint that shows itself—that is to say, a child of syphilitic parents may show no other sign of being affected. The recognition of this possibility is important, as some of these cases may be saved by the prompt administration of mercury.

Visceral Lesions.—In the class of cases now being considered, which are born healthy, and only show the first signs of the disease at four to six weeks after birth, the viscera are much less frequently attacked than in the infants who die before or soon after birth. Still, they do not always escape, the organ most commonly affected being the spleen, which is enlarged to some extent in the majority of cases. This enlargement seems to be due to a general hyperplasia. The liver is also sometimes enlarged and cirrhotic. In some cases there is a painless enlargement and hardening of the testis.

Ocular Symptoms.—The eyes are attacked in a large number of infants, either asiritis or choroiditis, or both. Choroiditis shows itself by a number of white patches in the fundus surrounded by pigment and by isolated collections of pigment. Hence an examination with the ophthalmoscope may clear up a doubtful diagnosis.

Skeletal and Cranial Manifestations.—Where death does not ensue early, the disease usually responds very readily to treatment, and the infant loses the characteristic symptoms and improves in nutrition. As a class, however, these babies are not as robust as uninfected children and the death-rate amongst them from accidental disorders, such as diarrhoea and broncho-pneumonia, is excessively high. The same is true, according to Dawson Williams, even among the later children of syphilitic families, although they have never shown any sign of syphilis. The



FIG. 102.—SYPHILITIC EPIPHYSITIS AND PHALANGITIS FROM A CHILD AGED TWELVE WEEKS.

interference with the general nutrition leads in nearly all cases to rickets and its consequences, and anemia is frequent and sometimes severe. In the second year of life these children are prone to exhibit all the bony changes in the skull and skeleton which result from rickets, but there are certain alterations in the skull which have been considered as due only to syphilis. Of these, craniotabes—where the ossification proceeds irregularly, and islets of unossified membrane are left, especially in the occipital bone and the posterior portions of the parietals—is by no means confined to syphilitic babies, though perhaps more frequent in them. Parrot, who was responsible for regarding craniotabes as always syphilitic in origin, also described as due to syphilis the formation of nodes around the anterior



FIG. 162.—RADIOGRAPH OF SYPHILITIC TIBIA PRESENTED IN A GIRL, AGED ELEVEN AND A HALF YEARS.

fontanelle, producing what has been called the "natiform skull." This type is to be distinguished from the rickety skull, where the thickening is over the parietal and frontal bosses; but in most cases of congenital syphilis rickets is added to the original complaint, so that a mixed type of thickening of the skull results, in which it is not easy to determine what is due to syphilis and what to rickets.

Relapse.—Unless sufficiently treated, manifestations of congenital syphilis tend to return after their first disappearance, but the phenomena of a "relapse" differ in many ways from those originally present. A recurrence of skin eruptions or of pseudo-paralysis is very rare, the relapse being usually indicated by snuffles and mucous tubercles about the mouth and anus. After the age of twelve months condylomata are not uncommon, while earlier they are rare in contrast to the profusion with which they are exhibited in infants infected with syphilis after birth.

Later Manifestations.—After the third or fourth year of life symptoms begin to appear which may be compared with what are called "tertiary symptoms" in acquired syphilis. Thus, nodes appear on the bones, and gummata in the various tissues of the body. In the absence of treatment these tend to break down, producing abscesses and ulcers, with destruction of soft parts and bone. Thus marked deformities may arise, amongst the most frequent being perforation of the palate and of the nasal septum, with, in the latter case, sinking in of the bridge of the nose. Periosteal thickenings may deform the long bones and skull, and the tibia becomes specially characteristic, the shin being bowed forward like a cutlass or sword. These lesions are, of course, not pathognomonic of congenital syphilis, but, owing to the rarity of acquired syphilis in childhood, may usually be considered

as resulting from the congenital disease. The joints tend also to be affected, generally later than the twelfth year, resulting in a painless enlargement with some fluid effusion, which is usually symmetrical in distribution and is commoner in the knees than elsewhere. Characteristic changes take place in the permanent teeth, which Passini and others have shown to be due to the actual presence of the *Spirochaeta pallida* in the dental follicles, the result on the growth of the teeth being that there is a general hypoplasia; the affected teeth are therefore smaller than the normal and somewhat misshapen. All the teeth may be affected, or only one or two, but the most characteristic are the central incisors, which tend to be peg-shaped, with a central notch at the cutting edge. The canines and molars are less frequently attacked, but may be affected even when the incisors escape; these teeth are poorly developed, with small knobs of enamel projecting, instead of well-formed cusps (Turner).

In these later stages the eye is frequently attacked, but at this period it is the cornea which suffers, whereas in early life it is the choroid. The inflammatory condition of the cornea, interstitial keratitis, is shown by a certain amount of pericorneal vascular injection, and by the cornea itself becoming opaque, giving a ground-glass appearance.

The ear is also often implicated, the auditory nerve being the portion that suffers. The patient gradually, or sometimes suddenly, becomes deaf, and the deafness, once established, is permanent.

These three symptoms — stunted, undeveloped incisor teeth, interstitial keratitis, and deafness — were insisted on by Hutchinson as the three cardinal signs of late congenital syphilis. It is

probable, however, that they may all result from acquired syphilis, provided the infection takes place in early infancy (*cf.* Welander with regard to syphilitic teeth).

Congenital Syphilis of the Nervous System.—The nervous system may be attacked by congenital syphilis as well as by the acquired disease. There is a tendency, however, to exaggerate the influence of syphilis in the production of lesions of the nervous centres. Because a syphilitic meningitis may produce hydrocephalus, convulsions, or imbecility, it does not follow that these conditions are always, or even frequently, due to syphilis. Even when the history of congenital syphilis is clear, it may be difficult to determine whether the symptoms are really due to this complaint. For instance, convulsions may in certain cases be due rather to a consequent rickets than to the initial syphilitic infection, or the conjunction may be accidental. Of the special sense organs, the eye and ear, as has been already pointed out, are frequently affected, and the early occurrence of choroiditis may be emphasized. Paralysis of single cranial nerves is of occasional occurrence—*e.g.*, of the oculo-motor nerve, resulting in a temporary or permanent



FIG. 164.—THE APPEARANCE OF THE PERMANENT TEETH IN A CASE OF CONGENITAL SYPHILIS, SHOWING THE POORLY-DEVELOPED, PEG-SHAPED CENTRAL INCISORS WITH THE NOTCH OF THE CUTTING EDGE.

squint. Such paresis may be due to local disease of the nerve centre, or to the pressure of a tumour, which forms one of the types of cerebral tumour in children. As in adults, there may be degeneration of the cerebral substance, leading to a juvenile type of general paralysis, the chief characteristic of which is a progressive dementia. More rarely, a form of locomotor ataxia, starting about the age of fifteen years, may be met with.

DIAGNOSIS.—The diagnosis of acquired syphilis in the child depends on the discovery of the primary chancre, which is usually situated extragenitally, and not difficult to find. There will also be a well-marked roseolar rash and a profuse eruption of condylomata, and enlargement of the lymphatic glands. In the congenital disease the diagnosis depends upon the consideration of a number of separate points. With a typical rash and rhagades about the lips the complaint may be recognized at a glance, but in other cases the signs are not so distinctive. The rash may have to be differentiated from the erythematous condition produced about the buttocks from the irritation of the alvine and urinary discharges; doubtless this irritation is partly responsible for the special proclivity of the syphilitic rash to affect these situations. In syphilis the eruption has usually a more coppery colour, and tends to spread as discrete maculae over the front of the abdomen and down below the thighs to the legs. The presence of a rash about the chin or forehead, and especially on the palms and soles, would strengthen or make certain the diagnosis. Snuffling is an important and almost constant feature of congenital syphilis, but it must be remembered that it is quite common in infants from other causes. A history of numerous miscarriages in the mother, or of previous children afflicted with snuffles and rash, may indicate that a malarium is of specific origin. An ophthalmoscopic examination is of great value, as choroiditis is a frequent early complication. A painless enlargement of the testicles is in infants certainly syphilitic. The pseudo-paralysis has to be differentiated from infantile paralysis, acute epiphysitis, and scurvy, and its distinguishing features have already been explained. After the second year condylomata about the anus should be searched for in cases of suspicion, and after the seventh year the teeth, eyes, and bones, should be carefully examined. The earlier symptoms improve rapidly under treatment with mercury, and one is justified in laying some stress on the results of treatment in forming a diagnosis. Assistance may also be obtained from the results of the Wassermann reaction.

TREATMENT.—Provided treatment is sufficiently prolonged and efficient, the prognosis in babies who show no signs for the first few weeks of life is fairly good. There is even a fair chance of a healthy child being born to syphilitic parents after a long series of miscarriages, provided they are both rigorously treated. Infants, however, born with signs of syphilis on them, in the great majority of cases die, and this is the reason for the bad prognostic significance of the bullous rashes which nearly always occur at or shortly after birth. Malarium is a sign of bad import, and where it is extreme recovery is rare. Severe rickets increases the gravity of the outlook from the liability to broncho-pneumonia, intestinal disorders, and convulsions. As a whole, syphilitic children die from various complaints during the first year in disproportionately large numbers compared with unaffected infants, and this increased death-rate also applies, though not to the same extent, to the children of syphilitic parents who never show any symptom of syphilis.

Since the living cause of syphilis can be either killed or for a time rendered

insidious by the salts of certain metals, the drug treatment of the complaint naturally first comes up for consideration. The congenital type may be treated before conception, before birth, and after birth. As a rule the male is the parent who is the origin of the infection, and no man ought to be allowed to marry until at least three years have passed since the initial attack, and even then only if there have been no symptoms referable to the disease for over a year. Permission should also be withheld in any case where the treatment has not been sufficient. A syphilitic man has been known to beget a syphilitic child up to as late as ten years after infection. If marriage has already taken place, and if a miscarriage has occurred or a syphilitic infant been born, vigorous treatment with mercury of the father alone over several months may result in the next child being born healthy. Under these circumstances it is probably advisable to treat the prospective mother as well, even if she show no signs of infection. Some authorities consider that the mother of a syphilitic child is necessarily herself infected, and, without acknowledging this, it must be confessed that many persons of both sexes have so few early symptoms that the disease may escape recognition. If the mother is already pregnant, treatment should be persisted in, as this may favourably influence the foetus and may prevent disease of the placenta, with the consequence that a viable child may be born. Pregnant women have even been treated with salvarsan without injury to the foetus.

Children showing signs of syphilis at birth almost invariably die, but of course attempts should be made both by drug treatment and careful hygiene to save them. The more hopeful cases are those, born apparently healthy, who show the first symptoms about the third to the fifth week of life. As a rule these cases respond well to treatment with mercury. Babies take mercury well, and it may be given in disproportionately large doses compared to their age and size. The most convenient preparation is the mercury and chalk powder, which may be given in $\frac{1}{2}$ -grain doses to quite young infants three times a day, and in grain doses to babies a year old. Sometimes some intestinal disturbance and looseness of the bowels is caused, when the drug may be combined with aromatic chalk powder or a very small dose of opium (e.g., $\text{gr. } \frac{1}{4}$ of pulv. ipecac. co. for a child of two months). Fortunately, any such addition is only very rarely required. If it be desired to administer the mercury in the liquid form, $\mathfrak{R} \text{ x. xv. of liq. hydrarg. perchlor.}$ may be given to quite young babies, but this preparation is more likely to cause intestinal troubles than the pulv. hydrarg. c. cet. Inunction is also a convenient method of administering the drug; $\frac{1}{2}$ drachm of the ung. hydrarg. can be rubbed gently into the abdomen, back or axillae, at night, and then covered with a flannel binder, the ointment being carefully washed off in the morning. Care should be taken that a fresh place is chosen on each successive night, otherwise a troublesome rash may be induced. Inunction may, of course, be combined with the internal administration of the drug. Although the manifestations disappear with great rapidity, treatment should be continued for at least a year, and, if possible, for a further six to twelve months beyond this. Where the course has been too short relapses are liable to occur, and it is in the insufficiently treated cases that the more terrible later symptoms are prone to arise, such as keratitis, deafness, gumata, and periostitis.

Recently attempts have been made to kill all the peccant organisms by a single injection, or at most two or three injections, of a germicide. The drugs chosen for this have been various organic compounds of arsenic, and that most widely used has been

di oxydianilinoarsenobenzol, or, to give it its shorter names, "salvarsan" or "606." Although extended research has shown that this preparation cannot be depended on to kill all the spirochætas in every case, yet in nearly all the symptoms disappear with great rapidity. The most favourable results are obtained with the very early cases, but relapses sometimes occur even in these, and nothing definite is yet known as to the number of patients who will relapse and the proportion who will get tertiary symptoms. The preparation is a yellow powder, which just before use has to be dissolved in ethylic alcohol, and then converted into a mono- or disodium compound by addition of sodium hydrate, the excess of sodium hydrate being nearly neutralised. It may be injected deep into a muscle or into a vein. The first method causes a good deal of pain, and there is sometimes sloughing of the parts into which the injection has been made. The venous method seems to give quicker and more certain results, but is possibly more dangerous to life, and, owing to the small size of the veins, is more difficult in the infant. Death has taken place in a certain number of cases, and the method should as a rule be avoided in cachectic infants or those with disease of the central nervous system or eyes, blindness having been sometimes induced. Successful cases have been reported in infants with congenital syphilis, a proportionate dose for the age being given—*e.g.*, about 0.05 grammes. In deciding whether this method should be employed in a particular case, one has to remember its danger and the frequency of choroïditis in cases of congenital syphilis. These infants respond very readily to mercury, and it does not seem justifiable to employ a more dangerous drug, the administration of which often causes much pain, except where there is strong reason to suppose that the parents will neglect to bring the child for sufficient treatment. Still, favourable results have been obtained in that grave class of cases which shows symptoms at birth, and it is possible that some lives may be saved by its employment. Recently the employment of smaller doses has been advocated as being more free from danger. The same total quantity has to be given, and so five or six injections have to be used. An attempt has been made to treat the infant by injection of the mother when she is suckling it. As with the similar attempt to give the child mercury through the mother's milk, the results seem very uncertain, only about half the cases responding to the drug. In older patients, who do not respond to iodide and mercury, salvarsan may be employed, many encouraging results having been recorded.

Acquired syphilis in infancy and childhood stands on a different footing. The patients spread the contagion far and wide, whereas few and simple precautions are sufficient to prevent infection from congenital syphilis, which is very rare even without precautions. The organism as a whole is much less profoundly affected in acquired syphilis than in the congenital disease, and hence the employment of the drug is less dangerous in this class. As salvarsan appears to act more rapidly than mercury, and thus probably deprives the patient more quickly of his power of infecting others, its employment may be advocated where the general health of the patient is good, but it is advisable to give mercury as well.

One of the most important results of congenital syphilis is wasting, and of the cases with marked wasting a very large proportion die; hence the general hygiene must command very careful attention. The infants must have a sufficiency of air, must be suitably clothed, and any affection of the intestines or lungs must be carefully attended to. Above all, the food must be the most suitable obtainable. Breast feeding is the natural form of nutritional supply for all infants under the

age of ten months, and so the mother should suckle her infant, the risk of her being herself infected being infinitesimal even when she shows no sign of the disease. It is not, however, justifiable to allow a syphilitic infant to be suckled by a wet-nurse, as with the close association there is grave risk of her being infected. Thus, when the mother is unable to suckle, or where her milk is insufficient in quantity or quality, it is necessary to have recourse to artificial feeding with all its dangers.

As a rule the local expressions of the disease do not require special treatment. The rash on the buttocks disappears rapidly with the administration of mercury by the mouth. Where, however, there is excoriation of the skin the parts require to be kept very dry and well dusted. In these cases, and where there are anal condylomata, it is advisable to add some calamel to the dusting powder; e.g., a powder may be used consisting of equal parts of starch, zinc oxide, boracic acid, and calamel. Sores about the mouth may be kept clean with glycerine and borax. The nasal swelling which causes the "snuffles" may in rare cases give rise to so much obstruction that the baby is unable to suck. The crusta should be carefully removed after softening by soaking in hot water and oil, and then a mercurial ointment applied to the inner surface of the nostrils. A suitable ointment is the dilute arsenated mercury ointment (5 grains to 1 ounce of vasoline), or one made with 2 grains of calamel to 1 drachm of lard.

In the later stages of congenital syphilis, just as in the acquired disease, iodides have a striking influence over the manifestations. They may be given in increasing doses from 15 to 60 grains a day, and in intractable cases it is advisable to give mercury in addition. Here a combination of liq. hydrarg. perchlor. *ss.* with pot. iod. gr. x-xv. is often of great value. In suitable cases salvarsan may also be employed.

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CHAPTER XXII

TUBERCULOSIS

J. W. CARR

INTRODUCTION.—Despite the great diminution in the death-rate from tuberculosis in this country during recent years, it still remains a more frequent cause of death than any other single disease. The annual report of the Registrar-General

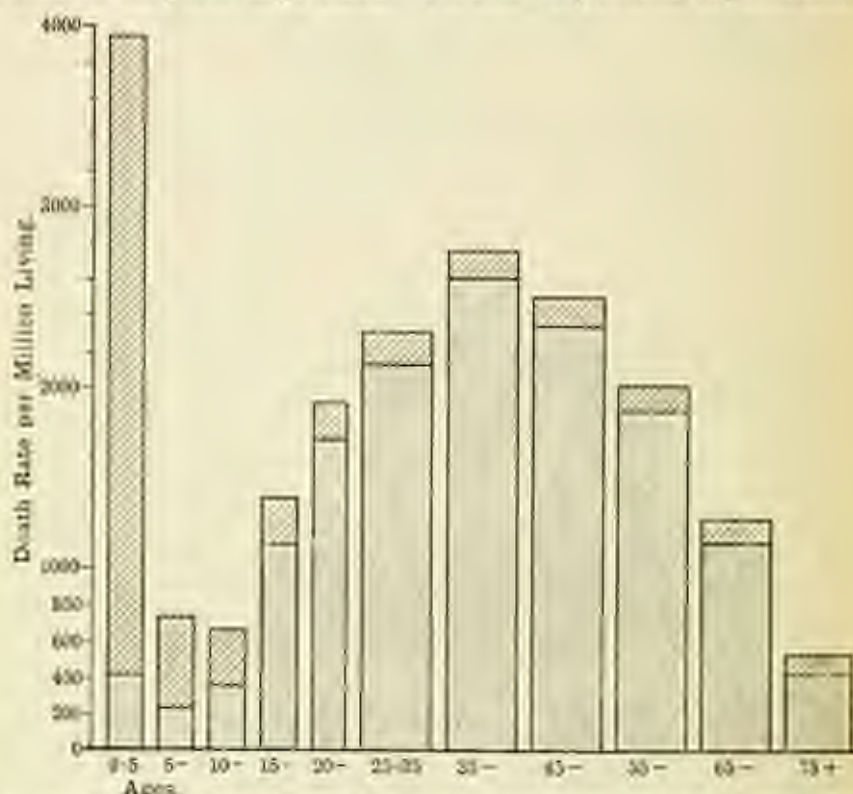


FIG. 165.—DEATH-RATE PER MILLION LIVING IN EACH AGE-PERIOD FROM PHTHISIS (DOTTED), AND FROM OTHER FORMS OF TUBERCULOSIS (LINED). (NEWSCOMBE.)

for 1910 shows that it was responsible for 10.6 per cent. of the total deaths in England and Wales in that year. More than a proportionate share of this high mortality falls upon children. Of the cases verified by post-mortem examination

in the children's hospitals of London, about 35 per cent. are due to tubercle; and if all these are included in which tuberculous lesions are found, without being the actual cause of death, the proportion rises to about 30 per cent. The total number of deaths in early life officially registered as due to tuberculous diseases is, however, considerably less than this, for babies only a few weeks old and children suffering from the ordinary infectious diseases are comparatively seldom admitted to children's hospitals, and hence the number of deaths from tubercle in these institutions is relatively higher. Moreover, many of the large number of deaths in early life certified as due simply to "meningitis" are probably caused by tuberculous meningitis. In 1910 only 2.2 per cent. of all the deaths of infants under six months were attributed to tubercle, the percentage being much reduced by the large mortality during the first few weeks of life from premature birth, congenital defects, atrophy, convulsions, etc. In the second six months the proportion of deaths from tubercle rose to 7.8 per cent., between one and four years to 11.9 per cent., and between five and nine years to 19.9 per cent.

VARIOUS TYPES OF TUBERCLE BACILLI—MODES OF INFECTION.

There are three varieties of tubercle bacilli—human, bovine, and avian; but the last is so rarely a source of danger to human beings that it need not be further considered. At present it is not possible to state what is the relative frequency of infection by the other two varieties, but that bovine tubercle is transmissible to human beings may be regarded as finally settled, the contrary view which was maintained by Koch being undoubtedly erroneous. In the Final Report of the Royal Commission on Tuberculosis the results of their investigations are summarized as follows: "Of young children dying from primary abdominal tuberculosis, the fatal lesions could in nearly one half of the cases be referred to the bovine bacillus, and to that type alone. In children, too, and often also in adolescents suffering from cervical gland tuberculosis, a large proportion of the cases examined by us could be referred to the bovine tubercle bacillus. We have already . . . referred to the importance of infection by the bovine type of tubercle bacillus in cases of lupus occurring in adolescents and children." The Royal Commission found that nine out of twenty of the cases of lupus which they investigated were due to a modified type of bovine bacillus.

Another point in regard to which we cannot at present speak with certainty is as to the relative virulence of human and of bovine tubercle in man. Perhaps this depends not so much upon the particular variety of bacillus as upon the size or frequent repetition of the dose, and also upon the resistant powers of the organism at the time when exposure to infection occurs. It is even possible that infection by comparatively small numbers of bacilli at a time may give rise to some degree of immunity. The Royal Commission on Tuberculosis (*loc. cit.*, p. 46) found "that by the inoculation of large doses of human tubercle bacilli, as also by vaccination with small doses of living bovine tubercle bacilli, the calf can in many instances be enabled to resist the inoculation at a later period of a dose of bovine tubercle bacilli which otherwise would have set up in it severe and fatal tuberculosis." In some of the animals, however, no resistance was produced, and the number of experiments made was insufficient to enable any definite conclusions to be drawn. The question is obviously a most important one, and calls for further investigation.

1. Intra-uterine Infection.—It is now universally accepted that tuberculous disease can only be produced by living tubercle bacilli, which obtain access to the body after birth. It is no doubt theoretically possible for intra-uterine infection to occur through the placental circulation, the placenta itself being usually, if not necessarily, tuberculous. The occasional occurrence of such cases in calves is undoubted, and instances probably occur from time to time in human beings, but so rarely as to be of no practical importance. Still more exceptional, even if it occur at all, is congenital tuberculosis due to the presence of tubercle bacilli in the sperm or the ovum. A diminished resistant power to infection by the microbe may almost certainly be transmitted, but the germ itself hardly ever. Comparatively few children under the age of three months die from tuberculosis, but in those who do the lesions found are always such as can be accounted for by infection from without after birth, usually via the respiratory passages. In a large proportion of these cases the mother is tuberculous. In one of the writer's cases mother and child died within a few hours of each other from pulmonary tuberculosis. There is not at present sufficient evidence to enable us to decide whether tubercle bacilli may get into the milk of a nursing mother who is suffering from tuberculosis, but it is only too obvious how great are the possibilities that a mother with advanced lung disease may infect her baby in other ways, unless the child be promptly separated from her. Cases are also recorded in which a midwife suffering from pulmonary tuberculosis has infected numerous newly-born infants by blowing directly into their mouths for the purpose of inflating their lungs.

2. Inhalation of Tubercle Bacilli.—Tuberculous disease may undoubtedly be transmitted directly from person to person, and the danger must be especially emphasized in the case of children, because it is probably greater for them than it is for adults. The air expired by patients suffering from pulmonary tuberculosis does not contain bacilli, but they are abundantly present in the fine spray or foam exhaled with the cough, and also with sneezing, singing, and talking. It is obvious, therefore, that there must be great risk of infection from this source to a young child brought into close and continued contact with a mother or nurse suffering from advanced tuberculous disease of the lungs and from frequent cough. Nevertheless it is probable that children, like adults, are more often infected indirectly, chiefly by dust containing tubercle bacilli, derived as a rule from sputum which has been allowed to dry instead of being destroyed. Discharges or excreta from other parts of the body affected with tuberculosis, whether from man or (be it remembered) from animals, may also, if allowed to dry, become sources of infection, as, for instance, the stools from patients with tuberculous ulceration of the intestine, the urine from tuberculous kidneys, the pus from tuberculous glands, bones, joints, etc. Sputum, however, is almost certainly the greatest source of danger.

3. Ingestion of Tubercle Bacilli.—Bacilli of the bovine type obtain access to the body mainly with food. The danger from meat is comparatively small, for tubercles are uncommon in muscle, the acid developed as a product of muscular activity being inimical to their growth. Meat may, however, get contaminated during removal from the carcass or caseous glands may remain adherent to it; but the bacilli, even when present, are usually destroyed by cooking, although raw meat or raw-meat juice must be recognized as a possible source of infection.

Tubercle Bacilli in Milk.—The one food which is a source of very real danger is milk. Tuberculous disease is extremely common amongst cows; how frequent in this country we have no reliable statistics to show, but very probably from 20 to 30 per cent. of all dairy cows are affected more or less, and in many European countries the proportion is still greater. This enormous prevalence is doubtless largely due to the highly unhygienic conditions, especially as regards overcrowding, under which the majority of cows are kept, so that when once the disease is introduced into a herd there is every facility for its rapid spread. Fortunately, it does not follow that because a cow is tuberculous its milk necessarily contains tubercle bacilli, although there is always the possibility that it may do, especially if the intestines or the genital passages are infected. By far the greatest risk is in connection with tuberculous disease of the udders, for in this case the milk is almost certain to contain virulent bacilli in enormous numbers. It is estimated that about 2 per cent. of the dairy cows in this country suffer from this form of tuberculosis, and consequently that about 100,000 quarts of infected milk are consumed daily in the United Kingdom. As a matter of fact, owing to the milk from diseased cows being mixed with that from healthy ones, considerably more than 2 per cent. of samples taken at random are found to contain tubercle bacilli, the actual proportion probably averaging about 10 per cent. This dilution of infected milk by pure milk, whilst it increases the chances of drinking tubercle bacilli, is an advantage rather than otherwise, because the number of bacilli ingested at one time may be so much reduced that they become comparatively harmless. Whilst the dangerous potentialities of tubercle-infected milk are undoubted, to determine with what frequency it is actually a cause of disease is a difficult problem. This is partly due to the fact that there is no necessary relation between the part of the body through which the bacilli obtain access and the region in which they subsequently give rise to disease; partly to the fact that, whilst in children tuberculous ulceration of the bowel is exceedingly common, in the majority of cases there is reason to believe that it is set up by swallowed sputum. We know how often this occurs in adults, and obviously it is likely to be still more frequent in children, who cannot possibly be prevented from swallowing infective sputum. For adults, who consume comparatively small quantities of milk, the danger is probably small; but for a young child fed mainly or entirely on infected milk the risk must be regarded as considerable, and it certainly reaches a maximum if the child is given the milk of one cow only, and that cow is suffering from tuberculous udders. Cases are on record which show that children so fed are particularly prone to suffer from tuberculous disease. It is most unwise to feed a child on the milk of one cow only, unless it has been proved by the tuberculin test to be free from tubercle. It must not be forgotten that the risk attendant upon the consumption of tubercle-infected milk extends to the cream, whey, and junket, and to a less extent also to the butter and cheese, made from such milk.

Tubercle Bacilli in Diet.—Another point to be considered is that, even when tuberculous disease apparently starts primarily in the intestine or in the mesenteric glands, it does not necessarily follow that the causal bacilli have been ingested with food. It is, of course, always possible that bacilli which have been inhaled may be deposited on the pharynx, and afterwards swallowed, but there is an especial danger in connection with what may be termed "the crawling stage of life," a stage through which practically all children pass towards the end of the first year, as a preliminary to actual walking, and which in rickety children may be prolonged

until the end of the second year or even later. At this period children are often allowed to crawl freely over dirty floors, staircases, pavements, etc. Naturally, their hands get covered with dirt, which is very likely to contain tubercle bacilli, some of which will certainly be transferred to their mouths and eventually swallowed. The considerable rise in the number of cases of abdominal tuberculosis met with in the second year of life is not improbably connected, in part at least, with infection during crawling. Obviously, in later childhood, also, infection may easily occur through dirty fingers, and, in fact, tubercle bacilli have been found in the dirt on the hands and under the finger-nails of children.

DISTRIBUTION IN THE BODY.

Inoculation of the skin by tubercle bacilli, although it certainly occurs, is so rare that it need not be considered here. For practical purposes we may say that all tuberculous disease is set up by bacilli which enter through the mouth or nose, and therefore come into contact with the mucous membrane of the alimentary or respiratory passages. It is important to recollect, first, that whilst tubercle bacilli may and often do lodge and set up disease in the mucous membranes, they can undoubtedly, especially in children, pass through them without giving rise to any recognizable local lesion whatever; and, secondly, that in such cases they are usually absorbed by the lymphatic channels, although it is possible that they may pass, at times, directly into the bloodvessels. Bacilli which thus enter the lymphatics are for the most part arrested in the neighbouring glands. Many are doubtless destroyed here; but if they are so virulent or so numerous that they escape destruction, or if the glands themselves are already in an unhealthy condition, then the latter become tuberculous and undergo the familiar caseous change. It is even possible that bacilli may pass safely through the glands, just as they may through mucous membranes, without setting up any disease in them, and so reach the thoracic duct and eventually the blood-stream; but probably most of the bacilli which reach the general circulation are derived from caseous lymphatic glands. Bearing in mind these facts, we now proceed to discuss the channels through which the bacilli enter the body, and the way in which they are distributed to different parts and organs.

Cervical Glands.—Tubercle bacilli may lodge on the buccal or pharyngeal mucous membrane, and, if not expelled or destroyed, may pass through it and set up disease in the cervical glands. In swine this may spread to the pleura and to the apices of the lungs, but whether such extension can occur in the human subject is doubtful, as no direct channels of communication exist. Certainly, as a rule the mischief remains localized in the neck, for it has long been recognized that tuberculous disease of the cervical glands rarely spreads to other parts of the body or sets up general tuberculosis. The danger of infection occurring through the throat is probably increased by the presence of adenoids and of hypertrophied tonsils. Bacilli may be present in the crypts of the latter.

Alimentary Canal.—Infection beyond the throat occurs either by ingestion into the alimentary canal or by inhalation into the respiratory passages. It would seem, at first sight, an easy matter to determine the relative frequency of these two paths, for it is natural to assume that when the disease is confined to or most advanced in the abdomen it is an example of tuberculosis due to ingestion, and, conversely

that if localized or most advanced in the chest the case is one of inhalation tuberculosis. Unfortunately the question is by no means so simple as this, for, as already stated, the position of a tuberculous lesion is no necessary guide to the portal by which the bacilli obtained access to the body.

Tracing the bacilli first along the alimentary canal, we find that they hardly ever attack the oesophagus, stomach, or duodenum. No doubt many are destroyed by the acid of the gastric juice, but many escape, in infants all the more readily perhaps, owing to the relative deficiency of hydrochloric acid in the stomach. Reaching an alkaline medium in the lower part of the duodenum, they again become increasingly virulent as they descend the bowel. Any catarrhal condition which may be present favours their lodgment in the lymphoid tissue either of the small or large intestine. Ulceration finally results, especially in the Peyer's patches of the lower part of the ileum, and the disease may extend to the peritoneal coat and eventually set up a chronic tuberculous peritonitis. The mesenteric glands become involved, either secondarily to the intestinal affection, or often, as already explained, independently of any visible local lesion. From these glands the infection may spread to the thorax—to the tracheo-bronchial glands and the lungs—and so eventually lead to fatal pulmonary disease. It has even been suggested that a large proportion of the cases of chronic tuberculous lung disease in adults arise as a result of infection *via* the alimentary canal, and that often, or even usually, in early childhood, the mischief remains quiescent for many years; but, for reasons to be given later, such views are probably erroneous. Finally, ingested bacilli may reach the blood-stream, either by direct entry into the vessels from the surface of the bowel or *via* the lymphatic channels, usually subsequent to, but occasionally independently of, a local lesion either in the intestine or its associated glands.

Respiratory Passages.—We turn next to the consideration of the part played in the production of tuberculous disease by inhalation of the bacilli into the air passages. Formerly this was universally regarded as by far the most important mode of infection, but during recent years, owing largely to the experiments and teaching of Calmette and his pupils at Lille, there has been an increasing tendency to deny the frequency of direct infection of the lungs, and to say that "air infection probably means that the bacillus enters through the mucous membrane of the throat or alimentary tract. Direct infection of the lungs is negligible" (Cantley and Whittie). The old view, however, is not to be so readily discarded. It is hard to believe that the pulmonary tuberculosis, which admittedly can be readily set up in animals by inhalation experiments, is due usually to indirect infection through the alimentary canal, even in the absence of any indication of primary mischief in the intestine or the abdominal glands. It is equally difficult to imagine that ordinary cases of chronic pulmonary tuberculosis in adults, obviously starting locally, are not due to direct infection. Were such cases usually the result of infection by ingestion, we should expect a considerable number, at any rate, to be due to bacilli of the bovine type, whereas it is notorious that in the vast majority the sputum contains bacilli of the human type only. It may be suggested that by long-continued residence and growth in the human body the bovine bacillus may undergo a gradual transformation into the human type, but of this the Royal Commission on Tuberculosis found no sufficient evidence. If we admit that direct infection of the lungs is the rule in adults, it is hard indeed to deny the probability of its frequent occurrence in children. Undoubtedly, in

early life the bronchial glands may be tuberculous without any visible lesion in the lungs, but this is no argument against their having been infected *via* the respiratory passages, for it is only analogous with the frequent caseation of the mesenteric glands without any disease of the intestine. Experimental evidence upon the question is conflicting. Louis Cobbett of Cambridge finds that in guinea-pigs finely-divided carbon particles do not reach the lungs or bronchial glands from the intestine, and that pulmonary tuberculosis does not occur in guinea-pigs fed with tubercle bacilli, except as part of a generalized infection, although it can be readily set up by inhalation of the bacilli. He is of opinion that his experiments lend no support to the theory that the intestine furnishes a common portal of entry for the tubercle bacilli which cause phthisis. Similarly, Leonard Findlay of Glasgow finds that pulmonary anthraxosis can easily be produced by the inhalation of soot laden air, but not by feeding with an emulsion of soot or Chinese ink, although he admits that living bacilli may behave in different fashion from mere lifeless particles of carbon. Other observers have found that a much larger dose of tubercle bacilli is necessary to secure infection through the alimentary than through the respiratory tract, so confirming the old view that they are a much greater source of danger on the bronchial than on the intestinal mucous membrane.

We may note also that in some Eastern countries in which but little milk is consumed tuberculous disease is very common.

On the whole there does not seem, at present, to be sufficient reason for discarding the old belief that pulmonary tuberculosis (when not of the miliary type), whether in children or adults, is due in most instances to direct infection of the lungs through the inspired air, and also that, whilst tuberculous disease of the mesenteric glands points to infection *via* the intestine, that of the bronchial glands usually indicates infection *via* the respiratory passages, although in some instances it may be due to alimentary infection. Certainly it would be as unwise to act upon the assumption that Calmette's conclusions are true, and that direct infection of the lungs is negligible, as experience has shown it was to act upon Koch's statement that bovine tubercle was not transmissible to the human subject.

Bones, Joints, Kidneys, etc.—We have still to explain the occurrence of tuberculous disease in those parts of the body in which direct infection is impossible—the bones, joints, kidneys, brain, etc. Bacilli must reach these through the blood-stream, although precise knowledge as to the exact mode and conditions of infection is still much needed. It has already been suggested that tubercle bacilli may pass directly into the bloodvessels from the alimentary canal, and perhaps from the respiratory passages as well; also that they may enter indirectly *via* the lymphatics, passing unharmed through the mucous membrane of the pharynx, intestine, or bronchi, and also through the glands beyond into the blood. More frequently, perhaps, they get into the blood from a caseous focus, which may be situated in any part of the body, but in children is usually in the internal lymphatic glands. Probably it is only from such a centre that any very large number of bacilli reach the blood at one time—a number sufficient to set up a general and necessarily fatal miliary tuberculosis—for in autopsies upon such cases a softening tuberculous lesion can almost invariably be found if a sufficiently careful search be made. However, bacilli, which reach the blood in either of the two first-mentioned ways may give rise to tuberculous disease in any part of the body, particularly if favouring local conditions are present. The most important of these are trauma and pre-existing morbid states. It is well known how often a slight injury to a

joint appears to be the starting-point of tuberculous disease in it. Presumably the injury temporarily lowers the resistant power, so that any stray bacilli which obtain access to the part find a favourable soil in which to develop. This is, of course, true of other pathogenic microbes besides the tubercle bacillus. Abnormal or unhealthy local conditions in any part of the body also favour the lodgment and growth of the bacilli. This applies especially to the lymphatic glands. The cervical glands are often enlarged and congested owing to eczematous conditions of the related areas of the skin, or from disease of the mouth, teeth, or nasopharynx; the abdominal glands as a result of chronic gastro-enteritis, so common amongst improperly-fed children; and the bronchial glands from chronic catarrhal conditions of the respiratory passages. In each case the glands are rendered less capable of destroying any bacilli which may reach them, either directly from the related mucous membrane or indirectly via the blood-stream.

COURSE OF THE DISEASE.

1. *Arrest*.—The younger the patient, the less likely is arrest to occur. It sometimes takes place, however, and in an early stage the tuberculous focus may be entirely destroyed and replaced by fibrous tissue. This, of course, is an absolute cure. In more advanced cases, after caseation has supervened, the mass may dry up and calcareous deposition take place in it. It is no uncommon experience, in making autopsies on children who have died from diseases entirely distinct from tubercle, to meet with old calcareous foci, especially in the bronchial and mesenteric glands. Unfortunately, such foci may remain for years potential sources of fresh infection, either local or general, for they often still contain living bacilli capable of being stirred into renewed activity by any condition which lowers the child's general health, or by any disease attacking the organ with which the affected glands are connected. This explains the frequency with which acute tuberculous disease follows measles and whooping-cough. Sometimes, no doubt, a fresh infection from without attacks a child recently weakened by one of these diseases, but more frequently they merely excite to renewed activity an old quiescent tuberculous focus.

2. *Direct Extension to Adjacent Parts*.—This occurs, of course, when the lesion does not undergo arrest. In the lungs it plays a most important part in the progress of the disease. Another frequent example is afforded by the little tubercles which can so often be seen studding the peritoneal surface of a tuberculous ulcer of the intestine, an extension which often culminates in chronic tuberculous peritonitis. Similarly, caseous bronchial glands may set up a pulmonary tuberculosis starting from the roots of the lungs.

3. *Indirect Extension*.—This is met with in connection with the inhalation of softening caseous material from one part of the lung to another; in the infection of the bowel by swallowed sputum, an occurrence of especial frequency in early life; in the infection of the lower genito-urinary passages from the kidney, and of the lymphatic glands from disease of the lungs or intestine.

4. *General Blood-Infection*.—This occurs very often in children, occasionally from tuberculous disease of the thoracic duct, but usually from softening of a caseous mass, most frequently in a gland, and its escape into a bloodvessel. In any case an enormous number of tubercle bacilli enter the circulation and set up a general miliary tuberculosis, in which, as a rule, the symptoms of tuberculous meningitis

predominate clinically and are the actual cause of death. It is a remarkable fact, at present not satisfactorily explained, that in some cases of undoubted blood-infection the miliary tubercles may develop predominantly either in the cerebral membranes, the lungs and pleura, or the peritoneum, the other parts of the body being comparatively unaffected, although they rarely, if ever, entirely escape.

SPECIAL FEATURES OF TUBERCULOUS DISEASE IN EARLY CHILDHOOD.

1. *The Important Part played by the Lymphatic Glands.*—This is doubtless a result of the greater functional activity and development of the lymphatic system in early life. In later childhood and adult life the cervical glands are comparatively rarely affected by tubercle, and cessation of the internal glands is usually entirely secondary, both in point of time and in clinical importance, to disease of the lungs or bowel. In young children, on the contrary, tuberculosis frequently begins in the glands, local extension often occurs from them, especially in the thorax, and they are by far the commonest source of general blood-infection. It is of some interest and importance to know whether the disease begins more often in the bronchial or in the mesenteric glands. It would seem that in Edinburgh and Glasgow primary tuberculosis of the abdominal glands is the more common, but in London statistics from all the children's hospitals conclusively prove that the disease starts more frequently in the thoracic glands. Precise figures are not of much value, as a more advanced focus of disease is not necessarily an earlier one; but the writer is disposed to think that in children tuberculosis commences in the thorax about three times more frequently than in the abdomen. As already indicated, opinions differ as to whether this necessarily implies that infection via the respiratory passages is proportionately commoner than through the alimentary canal; the writer certainly believes that it does.

It is important to realize that in the majority of cases we have no certain means of recognizing the presence of tuberculous disease of the internal lymphatic glands. It is true that calcareous mesenteric glands may sometimes be felt, although care must be taken to distinguish them from fecal masses; but the enlargement of the bronchial glands is rarely sufficient to give rise either to definite local physical signs or symptoms, in this respect differing from that due to lymphadenoma. Some help may be obtained from X-ray examination, especially if calcareous changes are present in the glands. Doubtless many an obscure condition of malaise in a child, many an indefinite febrile attack for which no cause can be found, is due to the development of tuberculous disease of the internal glands. The condition is certainly very common, and must from time to time give rise to general symptoms.

2. *The Marked Tendency to Dissemination of the Disease.*—In adults tuberculosis attacks the lungs first of all in the vast majority of cases, and extensive generalization is comparatively rare, for laryngeal and intestinal lesions are almost invariably due merely to secondary infection from the lungs. Even in the comparatively rare instances in which acute miliary tuberculosis supervenes, the primary focus, and that causing the blood-infection, is nearly always in the lungs. In children, on the other hand, pulmonary disease, though sufficiently common, is only one of the many forms in which tubercle may show itself, and, moreover, it is

not very often the primary lesion. Certainly, in the great majority of the cases in which tuberculosis is the immediate cause of death in early childhood, the disease is more or less generalized—that is to say, the conditions found are not due merely to simple extension by continuity, nor to direct infection of one part from another, as of the bowel from the lung, but either miliary tubercles are present in several or in most of the internal organs, or else there are two or more apparently independent tuberculous centres in different parts of the body. A large proportion of the cases, probably more than one-third of the whole, die from a general miliary tuberculosis, the clinical features being usually those of tuberculous meningitis, for the cerebral symptoms completely mask those which might be due to miliary tubercles in other parts of the body. The infective centre from which the blood-infection arises is generally a softening caseous lymphatic gland, situated much more frequently in the thorax than in the abdomen, and very rarely in the external glands; but it may be a breaking-down caseous focus in any part of the body—in the lungs, intestine, bones, joints, or middle ear. Occasionally an operation upon a tuberculous lesion leads to blood-infection and the consequent development of a rapidly fatal general tuberculosis.

3. *The More Rapid Progress of the Disease.*—This accords with the general rule that nearly all diseases tend to assume a more chronic type with advancing years. In most instances, the younger the patient when attacked by tubercle, the less is the tendency to arrest or repair, to fibrosis or calcification, the greater the chance of rapid caseation and softening, the greater, also, as already explained, the liability to dissemination and to general blood-infection.

4. *The Peculiar Character of the Disease in the Lungs.*—In young children tuberculous lung disease differs in many important respects from the same affection in adults, and also in older children, the only special point to note about the latter being that after the age of seven, or even earlier, the disease, though not very common, usually runs a more rapidly fatal course than in adult life. The following features call for special note in regard to pulmonary tuberculosis in infancy and early childhood:

(a) *The starting-point is often different.* In adults the disease usually begins at the apex of the lung, and spreads steadily downwards more or less rapidly. In young children it is quite exceptional to meet with this sequence of events, or, indeed, with any condition even approximating to the familiar chronic phthisis or classic pulmonary tuberculosis of adult life. Frequently the disease starts, apparently, from caseous bronchial glands lying embedded in the lung close to its root. The tuberculous process extends from them along the bronchial septa, and by the time death occurs small cavities are found at or near the root of the lung, with caseous masses around in various stages of disintegration; and outside these, radiating fan-like towards the periphery, small tuberculous nodules, more and more sparsely scattered as the exterior of the organ is approached. In a series of autopsies it is possible to trace every intermediate step from the commencement in the glands onwards to complete destruction of the lung. Occasionally a softening gland ulcerates through into the lower end of the trachea or a bronchus. In rare instances sudden death follows; usually the caseous material is drawn into the bronchioles and pulmonary alveoli, and sets up a diffuse caseation, which generally proves fatal in a few weeks.

(b) *The spread is more rapid and irregular.* This is true both of the cases just described, arising by infection from the bronchial glands, and also of those starting

in other ways. Often there is no special incidence of the disease at the apices, but caseous nodules are found scattered quite irregularly through all parts of the lungs, as are the patches of ordinary broncho-pneumonia. In fact, the distinction between the two diseases during life is often difficult, if not impossible, physical signs, symptoms and course being alike practically identical, and no sputum available for examination.

(c) The physical signs are different in many respects. This may be to some extent inferred from what has already been said in regard to the peculiar distribution of the lesions and the irregular course of the disease in early life, but it is most important to note how slight may be the signs even when the lung mischief is very extensive. We know only too well that in adults pulmonary tuberculosis is almost invariably more widespread and more advanced than the physical signs would lead us to expect, but the discrepancy is still more marked in young children. That miliary tubercles, even when the lungs are riddled with them, may give rise to no abnormal signs except those of a slight bronchitis, is not surprising when we realize that they cause neither consolidation nor softening. Far more astonishing is the fact that signs may be almost as scanty when caseous masses are scattered abundantly through the lungs, provided that there is no extensive softening and no large area of complete consolidation, so that a certain quantity of air is still fairly uniformly distributed through the alveoli. Well-marked cavernous signs are seldom present, and consequently it is sometimes said that cavities are rare in early life. This, however, is incorrect; they are frequently seen on the post-mortem table, but they seldom give rise to characteristic signs, partly because they are often situated in a position unfavourable for physical examination—near the root of the lung, or at the base, or in the centre of a lobe—and partly because the disease so commonly proves fatal, either from the supervention of general tuberculosis or from rapid extension of the local mischief, whilst they are still comparatively small.

(d) Many of the symptoms are different. Haemoptysis and night-sweats are uncommon, and tuberculous laryngitis is rare. In many cases the most marked feature is progressive wasting, together with more or less fever, not necessarily high, for which no obvious or sufficient cause can be found even on careful physical examination. Sputum, though often abundant, is rarely expectorated, and, being swallowed, sets up gastro-intestinal disturbance, and only too often extensive ulceration of the intestine. Consequently wasting and symptoms due to digestive disorder often completely predominate over those caused by the lung disease.

DIAGNOSIS.—The only absolutely reliable test for the presence of tuberculous disease is the discovery of the specific organism—the tubercle bacillus; but in enclosed lesions this test is, of course, not available. Moreover, to find the bacilli in urine or faeces is no simple matter, and sputum is rarely expectorated by children, although some may be obtained by special methods, as, for instance, by passing a tube into the oesophagus and examining any secretion which may adhere to it, or by irritating the throat and collecting any sputum coughed up.

The Tuberculin Tests.—1. *The Subcutaneous Test*.—Short of the actual finding of the organism, the tuberculin test is probably the most useful. Waterhouse, who states that he has only twice been misled by it in over two hundred cases, uses the following method: Old Tuberculin is injected subcutaneously, 0.0005 c.c. for children over five, and 0.00025 c.c. for those younger. If tuberculous disease is present, the reaction usually occurs within twelve hours, but may start in

half an hour. It lasts a few hours, and consists of a rise of temperature of 2° or 3° , with a corresponding rise in the pulse-rate, accompanied by slight redness. If no reaction occurs within forty-eight hours, a dose double the original one should be injected on the third day. If there is again no reaction, the case may be regarded as non-tuberculous; but if any doubt should still remain, a further injection, four or five times the size of the original one, may be given. Unfortunately the test is not available in febrile cases if the mouth temperatures reach 100° F., and the fact that transient inflammatory signs—swelling, redness, and tenderness—may show themselves in the tuberculous lesion, if this is situated comparatively superficially, indicates that there is at least a possibility of starting a quiescent focus of disease into fresh activity. In fact, in many cases of tuberculous lung disease, the injection is followed by a distinct increase in the physical signs. Unless there is a recognisable local reaction, the test, if positive, affords no indication as to the situation of the lesion.

2. *Von Pirquet's test* is obtained by vaccinating the skin with Old Tuberculin. The skin on the inside of the forearm is scratched with a needle, usually in two places, and a drop of 25 per cent. tuberculin rubbed in. A control scratch, without tuberculin, should be made at the same time. If the patient is tuberculous, in from twelve to twenty-four hours, or occasionally a little longer, well-marked redness develops round the scratch, and simple papules, or even vesicles, form, and last for a few days. In cases of advanced or extensive mischief, the reaction is feeble, or may not develop at all, but the chief drawback to the test is that it really proves too much. It would appear that the great majority of children, at any rate in towns, become infected with tubercle before they grow up, and henceforward give the von Pirquet test, although the disease may be absolutely latent. It is common enough to meet with children who, on physical examination, show some doubtful signs in the chest, which are capable of being interpreted as due to tubercle. If, in such circumstances, von Pirquet's reaction is positive, it is easy to assume, and to act on the assumption, that the child has active tuberculous lung disease, an inference which may be entirely unjustifiable from the clinical standpoint. Up to the age of two years a positive reaction probably indicates active mischief, because quiescent or obsolescent tubercle is rare in infancy, but afterwards arrested or latent disease becomes so common that the test almost ceases to have any clinical value. Moreover, like the simultaneous test, it gives no information as to the situation of the lesion.

3. *Cafauette's test*, by the instillation into the conjunctival sac of one or two drops of a 0.5 per cent. solution of Old Tuberculin in normal saline or 3 per cent. boric acid solution, need only be mentioned. In tuberculous cases it is supposed to set up more or less conjunctivitis within a few hours. Apart from any doubt as to its actual value as a test, its use is not advisable, on account of the risk of setting up a severe and even locally dangerous inflammation, especially if the eye is in any way previously unhealthy (see also *Prolegomena*, III., p. 23).

X-ray examination is sometimes of considerable value in diagnosis, particularly in disease of the bones, joints, and lungs, but it does not differentiate between tuberculous and simple broncho-pneumonia. The X-rays may also be of great therapeutic value in the treatment of the early stage of tuberculous disease of the external lymphatic glands, or as a sequel to operative interference.

TREATMENT.—The real treatment is prophylactic. The different ways in which tuberculous disease is spread are now so well recognized that the continued existence of the necessity for curative treatment must be regarded as a confession of failure in preventive medicine. We have to aim, first of all, at the destruction of the tubercle bacilli excreted by human beings, pre-eminently in the sputum, but also from the intestines, the kidneys, and from diseased glands, joints, bones, etc. The desirability also of destroying the bacilli excreted by tuberculous animals must not be overlooked. We have likewise to secure that milk from tuberculous cows—above all, from those with tuberculous udders—shall not be used for human consumption, particularly by children; and, until this most necessary reform is carried out, to insist upon the importance of boiling or pasteurizing all milk which is not known to come from cows recently tested with tuberculin and found not to react.

We have to recognize, however, that a long time must elapse before we can hope to reduce to a minimum the possibilities of tuberculous infection by all the various channels through which it obtains access to the body, and it is most important for the present, therefore, to strengthen our second line of defence—the resistant powers of the organism against the invader. It is unnecessary to enter into details, for everything which improves the general health—plenty of fresh air, with avoidance of overcrowding either by night or by day, sunshine, sufficient and proper food, avoiding excess of carbohydrate, suitable clothing, etc.—tends also to increase the resistant power against tubercle. Catarrhal conditions of the mucous membranes should be especially guarded against, for they not only facilitate the entrance of tubercle bacilli, but also lead to unhealthy conditions of the related lymphatic glands, which greatly favour the lodgment and development of bacilli in them. Particular care should be exercised during convalescence from measles and whooping-cough both to obtain complete recovery and to avoid exposure to infection.

Tuberculin Treatment.—The actual details of the treatment of the tuberculous lesions of different parts of the body are discussed elsewhere, but a brief reference may be made here to the use of New Tuberculin (T.B.), although many points connected with it must still be regarded as *sub judice*. The treatment is useless for glands which are already caseous or suppurating, and as a rule it is not employed for acute or febrile cases; but, if used at all, very minute doses should be given—at first, perhaps not more than 0.000001 c.c. In ordinary chronic cases of tuberculous disease the initial dose should not exceed 0.00001 c.c., and should be very cautiously increased at intervals of seven to fourteen days, until a dose of 0.0001 c.c. is attained. Very much larger doses than this are sometimes gradually reached, allowance, of course, being made for the age of the patient. For strict accuracy the frequency and dose of the injections should be regulated by estimations of the patient's opsonic index to the tubercle bacillus, in order to avoid the risk of giving an infection during the negative phase of diminished resistance induced by a previous one. This danger, however, is practically inappreciable if the dose of tuberculin is increased very gradually and a sufficient interval allowed between the injections. A marked reaction after an injection indicates that the dose was excessive, a smaller one should be given next time, and after a somewhat longer interval than before. The tuberculin is generally administered subcutaneously, but has also been taken by the mouth, in normal saline solution, on an empty stomach, in doses twice as

large as those given by injection. This, however, is not to be recommended, as absorption is quite uncertain and may be very incomplete. In cases in which the disease is likely to be of bovine origin, it may be preferable to give New Tuberculin derived from a bovine source, but Nathan Raw is of opinion that cases of infection with bacilli of the bovine type do best with human tuberculin, and conversely.

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CHAPTER XXIII

DISEASES OF THE SKIN

By H. G. ADAMSON

INTRODUCTION.

A. Eruptions due mainly to Local Physical Causes:

ECZEMA.
NAPKIN-REGION ERUPTIONS IN INFANTS.
CHILBLAINS.

B. Eruptions of Parasitic or Microbic Origin:

1. Eruptions due to Animal Parasites:

SCABIES.
PHELYTHIASIS.
VARIOUS PARASITIC ERUPTIONS.

2. Fungal Infections:

RINGWORM.
TAVUS.

3. Microbic Infections:

IMPETIGO.
DERMATITIS GANGRENOUSA INFANTUM.
VACCINIFORM DERMATITIS.
DERMATITIS EROSIIVA (BETTER'S DISEASE).
IMPETIGO OF BACKHAFF.
MULTIPLE SIMULTANEOUS ABSCESSSES.
SCROFULA.
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3. Microbic Infections—continued:

MOLLUSCUM CONTAGIOSUM.
TUBERCULOSIS OF THE SKIN.
SKIN ERUPTIONS OF CONGENITAL SYPHILIS.
HERPES FAMILIALIS; HERPES ZOSTER.

C. Toxic Eruptions:

ERYTHEMA.
URTICARIA.
LICHER URTICATUS.
URTICARIA PRURIENTISSIMA.
GRANULOMA ANNULLARE.
DROPS ERUPTIONS.
EXEMA RASHES.
VACCINATION ERUPTIONS.
PIMPLES.
FOLLICULITIS.
PITYRIASIS RUBRA.
GENERALIZED EXFOLIATIVE DERMATITIS.
ALOPECIA.
SCLEDERODERMIA.
LEUCODERMIA.
GRANULOMA RUBRA NASI.

D. Congenital Affections:

NAVI.
ICHTHYOSIS.
XERODERMA PHOTODUROM.
XANTHOMA.

INTRODUCTION.—There are certain skin affections which are peculiar to childhood; there are others which are more prevalent in children than in adults; and there are many the characters of which are so modified in early life that they present altogether different appearances from the same affections in older persons. Ringworm of the scalp, lichen urticatus, napkin-region rashes, and the eruptions of congenital syphilis, are seen only in children. Impetigo, in its various forms, is essentially a disease of infancy and childhood, though not confined to that age. Navi and other congenital conditions generally first come under observation at

this time of life. *Tupus vulgaris* usually appears in childhood, and it is of the utmost importance that this disease should be recognized and treated at an early stage. Eczema and scabies are eruptions which have special features in infancy. The eruptions of measles, stheln, and scarlet fever, are mostly seen in the young, as are also the various toxic erythematata which may be confused with them.

In the following pages the more common of these affections and those peculiar to childhood receive most attention. The rarer diseases are only briefly dealt with. As far as possible the diseases considered have been arranged upon an etiological basis:

- A. Eruptions due mainly to local physical causes.
- B. Eruptions of parasitic or microbial origin.
- C. Toxic eruptions, and eruptions of unknown origin, possibly toxic.
- D. Congenital affections.

A. ERUPTIONS IN THE CAUSATION OF WHICH LOCAL PHYSICAL AGENTS ARE A PROMINENT FACTOR.

Eczema is still to be regarded as a common disease, although its limits have been gradually narrowed by the exclusion of many diseases hitherto called eczema. The tendency is to restrict the term "eczema" to affections such as may be covered by the following definition: Eczema is a cutaneous inflammation of the skin of non-bacterial origin, the chief factors in the production of which are probably local external irritation and some unknown toxic or metabolic influence acting as a predisposing cause. Eruptions which are of microbial origin should be excluded from the category of eczema.

ETIOLOGY.—Eczema is common in childhood during the first two years of life. When occurring in older children, it has generally been prolonged from an attack beginning in infancy. Of the predisposing causes of eczema we know little or nothing, and the suggestion that they are toxic or metabolic is purely theoretical. In babies dentition has been thought to be sometimes a predisposing factor, but eczema may appear in infants before the period of dentition. Vaccination has been accused of producing eczema, but eczema is common in unvaccinated children, and by far the greater number of children who are vaccinated never have eczema. The supposed influence of gastro-intestinal disturbances is also difficult to prove. In regard to local causes, it is known that local irritations, such as frequent washing, strong soaps, exposure to cold winds, etc., may be the starting-point of eczema, and that eczema may be kept up and aggravated by local irritation, such as rubbing and scratching; but exactly why the skin becomes hypersensitive to such irritations we do not know. Children who have eczema beyond the age of infancy are sometimes also the subjects of asthma.

Symptoms.—In infants up to two years of age eczema usually begins upon the forehead or the cheeks as a redness and roughness of the skin. On close inspection of the red rough surface it will be seen that it is not really dry; the epidermis is finely fissured in all directions, and clear serum oozes from the minute cracks, and here and there is dried into tiny ridges. At a more advanced stage there are closely-set minute vesicles on a red, hot, swollen surface. As a result of rubbing or scratching the vesicles break, and a raw, weeping surface is produced; the discharge dries into crusts, and we then see inflamed areas covered with crusts,

between which is visible the red, weeping surface beneath. When fully developed, the eruption has a characteristic *mask-like* distribution over the forehead, cheeks, and chin, leaving free the orbits, the nose, and mouth. It may spread over the whole scalp, and patches may appear on the arms and legs, or on the trunk. It is important to note that in infants eczema always attacks the face with this mask-like distribution, that it is often limited to the face, and always more abundant there than elsewhere. *Itching* is a marked symptom, and the rubbing and scratching are the chief factors which keep up the eruption. The pruritus is generally worse at night.

In *older children* the eruption does not especially attack the face, and may be present in patches on the limbs and trunk; a favourite site is also at the bends

of elbows and behind knees. Such patches are circumscribed, red, raised, and covered with pin-point vesicles, or excoriated and crusted from scratching. On pinching up the skin in the affected parts it is found to be thickened by oedema. It will generally be found in these cases that the eczema dates from infancy. Sometimes these children also suffer from asthma, and although this seems to indicate some predisposing constitutional condition, it is probable that the local factor of constant scratching and rubbing is mainly responsible for the continuance of the eczema.



FIG. 106.—INFANTILE ECZEMA: SHOWS THE CHARACTERISTIC MASK-LIKE DISTRIBUTION OF THE ERUPTION ON THE FOREHEAD AND CHIN, LEAVING FREE THE NOSE AND PARTS AROUND THE EYES AND MOUTH.

parts the serous fluid separates the prickle cells and forms intradermic vesicles. Below the oedematous epidermis the bloodvessels of the corium are dilated and engorged. These findings serve to explain the clinical appearances of redness, swelling, vesication, and serous oozing.

DIAGNOSIS.—Eczema is distinguished from *impetigo contagiosa* by the oedema of the skin with pinhead vesication, the pruritus, and the tendency to exacerbation from local irritation. In *impetigo* the crusts are large and superficial, not pinhead-sized, and they reveal on removal a superficially excoriated surface, without any thickening of the skin from oedema. *Impetigo* is more irregular in its distribution, and, unlike eczema, attacks the parts about the nose, mouth, and eyes. There is no pruritus.

Scabies is often mistaken for eczema. If in an infant an eczema-like eruption covers the trunk and limbs, careful search should be made for "burrows." An itchy eruption in another member of the family is strongly suggestive of scabies. It should be remembered that eruptions about the naphis region in babies are never due to eczema (see Naphis, Erythema and Seborrhoea Dermatitis).

PROGNOSIS.—With careful treatment, infantile eczema may be cured in from several weeks to a few months. Even without treatment the complaint tends to disappear at about two years of age; but in some cases it may continue into childhood, or even into adult life. Instances of the sudden death of infants with eczema have been reported, but such cases are extremely rare.

TREATMENT.—The whole treatment of eczema may be summed up in three words—protection from irritation. It is useless to try one prescription after another in the hope of hitting upon a cure. The secret of successful treatment is to keep the affected parts protected from scratching, and this is a matter requiring much care and patience. When there are accumulated scales and crusts, these may be removed by borie starch poultices, or by bathing the part with warm normal saline solution (5i. of salt to 1 pint of boiled water). A dressing of folded lint soaked in normal saline solution and covered with rubber may be employed as a preliminary application in acute cases. In most cases we may begin at once to use a protective paste or ointment. The best application is zinc oxide paste.

Zinc oxide	℥i.
Purp. amyli	℥ss.
Vaseline	ad ℥i.

Fr. pasta.

This is spread thickly over the affected parts by means of a paper-knife. The spread paste is then covered with bits of butter-muslin, and the whole lightly bound on with thin bandages. Several times a day the butter-muslin must be dabbed on its outer surface with liquid vaselin in order to prevent the paste from becoming too dry. Once in twenty-four hours the paste is removed, that which sticks to the skin being gently wiped off with bits of wool dipped in liquid vaselin (paraffinum liquidum, B.P.). A fresh dressing of paste is then applied. In infantile eczema the paste is spread on a mask of butter-muslin with holes for the eyes, nose, and mouth, and with a flap to cover the top of the head. This is bound on lightly with butter-muslin bandages.

In order to prevent the child from scratching the arms may be put into cardboard splints—a roll of cardboard round the arm from shoulder to wrist. The arms can then be moved, but the hands cannot be carried to the face.

To cure a case of infantile eczema it may be necessary to continue this dressing for weeks or months. If there is any exacerbation of the eruption, it is generally found that the child has managed to scratch the parts, but it does not indicate a change of treatment. In cases which hang fire, painting the eczematous parts with a solution of nitrate of silver (gr. x. ad ℥i.) every few days will often hasten the cure. Another useful application is benizalol added to the zinc paste in the proportion of 3ss. to ℥i.

As to general treatment, drugs internally are best avoided. If there is anemia or rickets, the child may have cod-liver oil and iron. The feeding must be regular

and suited to the age of the child. In fat babies the amount of sugar and starchy foods should be restricted. In my experience it is useless to give bromides at bedtime with the view to prevent itching and procure sleep. Quinine (gr. ii.) in sugar-coated tablets has been recommended, but the most successful way is careful dressing and measures to prevent scratching and rubbing.

Napkin-Region Eruptions in Infants.—Infants are liable to certain eruptions on the napkin region, which are of importance because they may be mistaken for the eruptions of congenital syphilis. These eruptions are—

1. Simple erythema (erythema of Jacquet).
2. Streptococcal infections.
3. Seborrhoeic dermatitis.



FIG. 187.—NAPKIN-REGION ERYTHEMA.

The erythematous patches and the flat papules occur only upon those parts which are rubbed by the napkin—namely, the convex surfaces. The *flexura* remains free.

It should be noted that eczema never occurs on the buttocks of infants. The characteristic site of infantile eczema is the face. Only the simple erythema will be dealt with here. The other eruptions are described under Impetigo and Seborrhoeic Dermatitis.

Simple Napkin-Region Erythema (Erythema of Jacquet).—*Ερυθροσύν.*—This occurs in babies whose skin is in an irritable condition from some gastro-intestinal toxæmia. It is sometimes associated with *Scabies urticales*. It is probably a toxic erythema, whose site is determined by local irritation from the napkin.

Strawberry.—It begins as erythematous blotches on the convex surfaces of the buttocks, thighs, genitals, and abdomen, and appears sometimes on the calves and heels when these come into contact with the napkin as the infant lies with its knees bent up. The flexures, which are protected from friction, are free from eruption, and this distribution on the convex surfaces only is one of the most striking features of the eruption. At a future stage of the eruption these appear as the erythematous patches flat papules, from $\frac{1}{8}$ to $\frac{1}{4}$ inch in diameter. At a still further stage these papules may become excoriated and even ulcerated, so that they closely resemble syphilitic ulcers.

Diagnosis.—These simple eruptions are often mistaken for the eruptions of congenital syphilis; this is, of course, a serious error to make, as it concerns not only the child, but also the parents. In simple erythema the rash is confined to



FIG. 168.—ERUPTION OF CONGENITAL SYPHILIS.

The disc-like copper-coloured patches cover upon flexures and convex surfaces alike.

those parts which come into contact with the napkin, and the flexures in these regions are free (Fig. 167). In syphilis the central parts of the face and the palms and soles are commonly affected, and the flexures in the napkin region are involved equally with the convex surfaces (Fig. 168). In syphilis, too, the eruption is of a more coppery tinge, and there may be stiffness, hoarse cry, mucous patches or fissures at the angles of the mouth, or other indications of syphilis.

Treatment.—The buttocks must be washed regularly in warm water, and the napkins, which should be of a soft material, must be changed frequently. A simple grease, such as liquid vaselin, is better than any other application. When

there is ulceration, zinc ointment with 2 per cent. salicylic acid may be used; $\frac{1}{2}$ grain of grey powder may be given at bedtime for a week, and a sodium bicarbonate mixture three times a day.

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Chilblains (Erythema Pernio).—Chilblains may be defined as a localized and persistent exudative erythema, occurring on the extremities in predisposed subjects as the result of the action of a moderate degree of cold. There is usually a feeble circulation clinically manifest as a coldness of the extremities. It has been suggested that there is also a defective blood coagulability. Chilblains occur chiefly in children or in young persons, and in the winter-time, rarely persisting through the summer. They consist of circumscribed, livid red, intensely itching patches, which appear especially upon the fingers and toes, on the sides of the feet, on the heels, on the ears, and, less often, on the nose and other parts of the face. The colour of the patches disappears momentarily on pressure, and when the affected parts become hot the colour changes from a livid red to a bright red. They occur chiefly during cold, damp weather, and subside during warmer or drier weather. Vesication may occur in severe cases, and this may result in ulceration, commonly known as "broken chilblains."

DIAGNOSIS.—Chilblains are to be diagnosed from the much less common tubercles of the acute *sicofulorum* type (p. 1125) by the smaller size and the more papular, less diffuse character of the lesions in the latter affection, together with their tendency to necrosis, leaving pitted-out scars; also by the fact that "tubercles" are more widely distributed on the limbs, and that they do not itch, and that they may persist during hot weather. It has been suggested that "chilblains" are forms of tubercles, but there is really no evidence that they are such.

TREATMENT.—It is probable that some of the many local applications recommended owe their reputation to the fact that chilblains tend to disappear with a spell of milder weather. Among these commonly employed are tincture of iodine or valogen iodine (which latter preparation does not stain), liniment of camphor and magnesium capici are useful stimulants. Broken chilblains must be dressed with boric acid ointment or with boric acid fomentations if severe. The child should wear thick loose boots and warm gloves and socks, and should take regular daily active exercise, as skipping. Warm milk may be taken with the meals and at bedtime. Calcium chloride (in doses of 10 grains in sugar-water every four hours for a few days) has been strongly recommended. Perhaps one of the most effectual treatments is by galvanic hand baths once a day. An ordinary medical battery is used, and two small basins of water; the two hands and the two heels from the opposite poles of the battery are put one into each basin, and a feeble current (about 2 to 4 milliamperes) allowed to pass for ten minutes.

B. ERUPTIONS OF PARASITIC OR MICROBIC ORIGIN.

1. ERUPTIONS DUE TO ANIMAL PARASITES.

Scabies.—*Ætiology.*—Scabies is a common disease in infants and children in hospital and dispensary practice, and it may sometimes be met with in better-class patients, the child having generally become infected from the nurse. It occasionally occurs in epidemics in institutions. It is an infectious disease due to an animal parasite, the *Acarus scabiei*. The lesions are produced by the female acarus, which burrows in the epidermis.

Symptoms.—The essential features of the complaint are nocturnal pruritus and the presence of the burrows. The burrow appears as a sinuous line, from $\frac{1}{4}$ to $\frac{1}{2}$ inch in length, generally black, from included dirt, but, in clean children, often white. On examination of the burrow with a lens there can be seen at one end a yellowish opaque spot, just visible to the naked eye. This is the female acarus, which can be readily removed with the point of a needle. Mounted on a slide in liq. potassæ, it is seen to be an animal having an oval body with eight legs; the front legs with suckers, the back legs with spines. The burrow contains a string of ova, which have been deposited by the acarus as it makes its way between the layers of the epidermis. Sometimes towards the end of the burrow and slightly behind the acarus itself there is a pinhead-sized vesicle, known as the "pearly vesicle."

The burrow, with its "pearly vesicle," is found especially between the fingers, on the sides of the hands, on the wrists, and on the sides of the feet. Generally, in addition to the burrows, there are scratch marks and tiny scratched papules scattered over the trunk and limbs. Sometimes there are in addition impetiginous vesicles or pustules and crusts, especially about the hands and feet, due to secondary infection, or there may be eczematous patches on the trunk and limbs.

Except in young babies the face is unaffected; in infants at the breast there may be impetiginous or eczematous lesions also on the face. Sometimes in infants the whole trunk and face may be covered with eczematous patches, and a universal "eczema" in a baby is always highly suggestive of scabies.

Diagnosis.—A generalized itching eruption in a child below the age of six years is generally one of two things—scabies or lichen urticatus. The diagnosis between these affections may be difficult. Pruritus in another member of the family suggests scabies, but a certain diagnosis can only be made by discovering the primary lesion, the burrow and the acarus in scabies, and the wheal with a central papule in lichen urticatus. All cases of generalized eczema and of impetigo about the hands and feet should be suspected as scabies, and carefully examined for the burrows of that affection. Scabies with a secondary bullous impetigo must not be mistaken for pemphigus.

Treatment.—The object of the treatment is to destroy the acarus. The itching and secondary eruptions will usually subside when this is accomplished. The simplest treatment is to give a hot bath with plenty of soap on three successive evenings, the hot bath to be followed byunction with ung. sulphuris (gr. x. to gr. xxx. ad ʒi) according to the age of the patient—the patient sleeping each night in the same nightclothes, which become saturated with the ointment. The secret of successful

treatment is that it should be thorough for a short period. Sulphur ointment applied for too long produces an eczematous eruption. After the course of three baths andunctions, a tar lotion (liq. picis carbonis, 5i. ad 3xx.) should be mopped on daily for a week; or if there is still impetigo, the baths may be continued, and a mild boric acid ointment used until this is healed.

Pediculosis Capitis is very common in children of the poor. It is rare to find a child in dispensary or hospital practice who has not got "sits" in the hair. The "sits," or eggs of the head louse, are seen as small, oval, whitish bodies upon the shaft of the hair; they are attached by a tube-like structure, which encircles the hair. Except in severe cases the lice are only seen on careful examination. In bad cases the whole head may be swarming with lice and their ova, and such cases are generally complicated by impetigo-contagiosa, the whole scalp being covered with crusts. Milder forms of impetigo contagiosa of the occiput are also usually secondary to pediculosis.

DIAGNOSIS.—Scales of pityriasis or dandruff must not be mistaken for the ova of pediculi. The latter are firmly attached to the hair by a chitinous sheath, and can be made to slide along the hair, but cannot be picked off.

TREATMENT.—For the less severe cases a few washings with soap and water to which a little oil of paraffin has been added suffice to remove the pediculi and ova. If there is impetigo, the hair must be cut short, the crusts lathered off, and a white precipitate ointment applied. In severe cases the scalp should be mopped over with oil of eucalyptus and methylated spirits, and afterwards washed with soap and water and paraffin.

Pediculosis Corporis is very rarely seen in children;

Pediculosis Palpebrarum is sometimes met with in ophthalmic practice. The ova or sits are attached to the hairs of the eyelashes like a chain of beads, while the parasite is seen clinging to the hair at its base. The parasite is that of *Pediculus pubis*.

TREATMENT.—The lice and ova should be removed with forceps.

Eruptions due to Fleas, Bugs, Gnats, Caterpillars, etc.—The bites of the flea (*Pulex irritans*) and of the bug (*Acanthia lectularia*) are too well known to need description. The dark puncture of flea-bites must not be mistaken for purpura. In rare instances bullous lesions are produced by flea-bites. The harvest-bug (*Leptus autumnalis*) may give rise to a pustule eruption of bright red, extremely itching papules upon the forearms or legs, and gnats (*Culex pipiens*) and mosquitoes so wheals or so itchy, indurated swellings, which leave a hard papule behind when they subside. An itching eczema or urticaria, sometimes accompanied by conjunctivitis and irritation of the throat, may be caused by the loosened hairs of the *Bombix processionea*, or processionary caterpillar. Some skins are irritated by caterpillars quite harmless to most persons.

2. FUNGUS INFECTIONS.

Ringworm.—Ringworm is an affection due to invasion of the skin, hairs, or nails, by certain parasitic mould fungi. In children it may affect the hairy scalp (*Tinea tonsurans*), the glabrous skin (*Tinea circinata*), or the nails (*Tinea unguium*).

ETIOLOGY.—In ringworm of the scalp infection usually takes place from child to child by direct contact, or through the medium of a hair-brush, the cap of an infected child, of the barber's instruments, or in some such way. Less often it may be caught from the dog or the cat. Ringworm of the body may be associated with scalp ringworm in the same child or in another, but when present alone it is often of animal origin (cat or dog).

Symptoms.—*Tinea Capitis.*—Ringworm of the scalp occurs in two main clinical types—small-spored ringworm and large-spored ringworm.

Small-Spored Ringworm of the Scalp.—In typical cases of small-spored ringworm the scalp presents circumscribed, rounded, more or less scaly patches, over which most, but not all, of the hairs are broken short. The broken hairs look opaque compared with the surrounding healthy hairs, and on passing the finger over the patch they feel velvety—not bristly, as do normal hairs which have been cut short with scissors. The stumps have lost their elasticity, and remain sticking up in different directions when firmly brushed with the forefingers, instead of flying back to their former position. On picking up a bundle of stumps between the finger and thumb, they come away with fractured shafts. It is these opaque, lustreless non-elastic, fragile hairs which characterize a patch of ringworm. Sometimes the patches may be covered with thick white scales, masking the stumps, which are matted down by the scales, and only discovered with difficulty. On the other hand, the patch may be almost free from scales, and consist solely of the stubble-like ringworm stumps. If a child is seen during the first few weeks after infection, there may be but a single obvious patch. Soon, however, fresh patches appear on other parts of the scalp, and in long-standing cases almost the whole scalp may be involved.

Large-Spored Ringworms are of two clinical forms:

1. Those in which there are groups of three to half a dozen broken hairs scattered over the whole scalp. The hairs are sometimes long and embedded in scales, and sometimes broken quite short level with the epidermis, to form "black dots." This form of ringworm may be easily overlooked by those not familiar with it.

2. A second form, in which there is an isolated, sharply-defined ring or patch, with considerable inflammatory redness and broken stumps, not infrequently with hair the ring or patch on the glabrous skin of the face or neck.

Tinea Circinata.—Ringworm of the face, trunk, or limbs, may occur in children, either in association with ringworm of the scalp or independently. In the latter case it is often of animal origin—commonly a cat or dog ringworm. Body lesions associated with small-spored scalp ringworms may be round red scaly patches or rings of various sizes. The animal ringworms usually show some inflammation in the form of slight vesication or small crusts at the margin of the ring. Exceptionally the ringed lesions may be very numerous.

Tinea Unguium.—Ringworm of the nail is not a common affection. The ordinary small-spored ringworm of the scalp does not attack the nails, and the fungus is always of a large-spored variety. The nails attacked become roughened, opaque, and pith-like. Diagnosis from other dystrophies of the nail can only be made by the discovery of ringworm fungus. Filings from the nails should be soaked in liquor potassæ and examined under the microscope.

PATHOLOGY.—More than thirty varieties of ringworm fungus have been isolated. Some forms are found only in human beings, but a large number are ringworms

which affect animals, and which are occasionally seen in human beings. They are two main groups: *Microspores*, or small-spored; and *Trichophytes*, or large-spored. The varieties most commonly met with in children are—

1. *Microsporus audouinii*—responsible for the small-spored ringworm (i.e., the majority of scalp cases).
2. *Microsporus* of dog and cat—a few scalp cases.
3. *Trichophyton endothrix*—(a) of yellow culture, (b) violet culture—to which are due the large-spored ringworm of the scalp.
4. *Trichophyton auto-endothrix*—several forms, but particularly that derived from the cat, which gives rise to most of the body ringworms of children.

The ringworm fungus is probably nearly related to the common mould fungus. It consists of mycelial threads and spores. In cultures fruit organs are also formed, which are not seen when the fungus grows on the skin or hair.

The method of invasion in ringworm of the scalp is as follows: The fungus first grows in the horny layers of the epidermis, and gives rise to a small scaly patch. It is only after the fungus has gained a hold in the epidermis that the hairs are attacked. Threads of mycelium then grow down into the hair, and finally the fungus completely fills and (in some forms) surrounds the hair shaft. As a result the nutrition of the hair is interfered with, and the hair breaks off. If any attempt is made to pull out the hair, it breaks in the follicle, leaving mycelium and fungus behind in the remaining portion of the shaft. As will be presently explained, it is this circumstance which renders the cure of ringworm of the scalp so difficult.

DIAGNOSIS.—Tinea Tonsurans.—Diseases which are often mistaken for ringworm are *psoriasis capitis* or "dandruff," *alopecia areata*, and the bald areas sometimes left after impetigo and other inflammatory conditions of the scalp. Or ringworm may be overlooked and mistaken for *psoriasis*. Such mistakes can only be avoided by becoming familiar with the appearance of the diseased hair-stump of ringworm: for in doubtful cases a correct diagnosis depends upon the finding or not of the ringworm stump. To confirm the diagnosis, a stump should be mounted as a slide in liquor potassæ and examined under the microscope, when the fungus will be clearly seen. It should be remembered that ringworm does not attack the scalp in adults.

Tinea Circinata.—When a circinate eruption suggests ringworm, the only certain way of deciding is to search for fungus in the scales. A small scale of epidermis at the margin of a patch should be separated with a scalpel or forceps, placed on a slide, and mounted in liquor potassæ. After soaking for half an hour, fungus, if present, will be seen as mycelial threads amongst the epithelial scales.

PROGNOSIS OF Tinea Tonsurans.—If left untreated and under the old methods of application of antiseptics, the disease may remain for months or years. Occasionally the ringworm patches become spontaneously inflamed, leading to red raised boggy swellings occupying the area of the patches. This is known as *borrie*. As the result of this spontaneous inflammation the hairs fall out, and when the inflammation subsides the patches are cured.

TREATMENT OF RINGWORM OF THE SCALP.—The hair ought to be cut quite short over the whole head as soon as the diagnosis is made. The extent of the disease is thus more easily seen, treatment is facilitated, and further spread more readily prevented. Parents will often object to the cutting of the hair, and an exception may be made in the case of a girl with only one patch of ringworm, but the greater

liability of the disease to extend should be pointed out. The difficulty in curing ringworm is due to the fact that locally-applied parasitocides do not reach the disease in the follicles. A cure can only be brought about by removing the infected hair. Epilation is useless, because the hair breaks. If the ringworm patches can be inflamed by local applications, the hairs will come out as a result, and a cure will thus be effected. Certain applications will sometimes bring about this desired result, but only in a very small proportion of cases. Applications which may be tried are—(1) Equal parts of common salt and vaseline (iodii chloridi, ss. ; vaselini, ss. ; ft. ung.). (2) Ung. hydrarg. nitratis (B.P.). In a very small proportion of cases the ringworm patches become inflamed by such application, and the hair falls out, leaving bald patches upon which the new healthy hair grows.

The only certain cure for ringworm is the X-ray treatment. By this method depilation is produced, and the fungus comes away with the hair. A measured dose of X-rays is given to the part affected, or, if the whole scalp is involved, to the whole scalp. In order to X-ray the whole scalp, five of these measured doses are given—one to the vertex, one to the crown, one to the occiput, and one on each side of the scalp (Kienbock-Adamson method). During the third week after the X-ray applications the whole of the hair, both diseased and healthy, over the regions exposed falls, and leaves the scalp quite bald. In six weeks from this time—two months from the X-ray applications—the hair begins to grow again. The child is free from infection as soon as all the hair has fallen—that is, in from three to four weeks after the X-ray applications. After the X-ray applications no further treatment is required beyond daily washing of the scalp, and the application of a simple ointment, such as ung. hydrarg. amm. dil. (gr. xv. ad ss.), to prevent the falling hairs from infecting others. The child should also wear a washable cap. The supposed danger of injury to the brain from X-ray treatment of the scalp does not exist. The real danger is in the production of permanent baldness, but the modern methods of measurement reduce that to a minimum when the treatment is carried out by an expert.

There is no need for X-ray treatment in *Tinea circinata*. An ointment, such as ung. hydrarg. nit., should be rubbed on daily, or the patch may be painted every two or three days with tinct. iodii.

In *Tinea unguium* the nails should be painted daily with tinct. iodii, and a rubber finger-stall worn over each affected finger. A cure takes from three to six months.

Aerles should not be fomented, but an antiseptic ointment (such as ung. hydrarg. amm. chlor. ss. with resorcin gr. xv.) should be applied.

REFERENCE.

SABOURAUD, R.: Les Tégumes. Masson et Co., Paris, 1916. (An exhaustive work on ringworm embracing all the recent discoveries in regard to this subject, and giving complete bibliography.)

FARUS is an affection of the hairy scalp and other parts of the skin due to a mould fungus nearly related to the ringworm fungi. It is rare in this country, except among Polish Jews and Russians in the East End of London. It is commoner in Scotland. There is also a farus of mouse origin which may affect human beings, the glabrous skin, and not the scalp, being infected.

SYMPTOMS.—The characteristic feature of favus in the production of a *crustacea*, or favus cup. This is a sulphur-yellow pinhead-sized to pea-sized disc with a cupped centre. It consists of the mycelium and spores of the favus fungus growing round a hair. Sometimes large numbers of these favus cups are seen; sometimes they are masked by impetiginous crusts. The hairs are invaded by the fungus, but they do not break off as do the ringworm hairs. A notable feature of favus is that permanent scarring is produced. In neglected cases favus may spread to the trunk and limbs, and it may infect the nails. In favus of mouse origin the patches are always limited in extent, and resemble patches of circinate ringworm, except that they present here and there favus cups.

TREATMENT.—The treatment of favus of the scalp is by X-rays as for ringworm. No other treatment is effective.

3. MICROBIC INFECTIONS.

Impetigo.—There are two forms of impetigo, one the impetigo contagiosa of Tilbury Fox, due to streptococcus infection; the other impetigo of Bockhart, of staphylococcus origin. It was at one time supposed that all forms of impetigo were due to staphylococcus infection, but it is now known that there is a very large group of eruptions of streptococcal origin. Many of these "streptococcal" eruptions have received special names, but they may be all regarded as modifications of the impetigo contagiosa of Tilbury Fox. They are all essentially vesicular or bullous lesions with a tendency to crusting. On the other hand, in the staphylococcal eruptions, of which Bockhart's impetigo is the type, the primary lesion is a pustule seated in and around a pilo-sebaceous follicle.

Impetigo Contagiosa of Tilbury Fox.—**ETIOLOGY.**—The affection is highly contagious, and is a very common complaint among children of the hospital class. Often several children in one family are affected. Among the children of well-to-do classes it is sometimes seen as "scrum-pox."

SYMPTOMS.—The eruption usually occurs on the face, especially on the parts around the nose and mouth. It forms large amber-coloured crusts which have a "stuck-on" appearance. The crusts can be easily removed with forceps, showing beneath a superficial erosion. The lesions are, in fact, superficial vesicles or blisters, the fluid of which has dried up to form crusts. Generally, on close inspection, one or more small clear vesicles can be seen among the crusts. These represent the earliest stage of the eruption. The eruption is extremely auto-inoculable, and it may spread rapidly over the face and scalp.

Sometimes the eruption first appears at the lower part of the occiput, and it is then always associated with pediculosis capitis, and is probably due to inoculation from scratching.

The glands draining the area affected may become red and inflamed, and may even suppurate. Lesions often occur upon the limbs and trunk, and in these situations they may show a greater tendency to remain bullous. When the eruption attacks the flexures, such as the groins, raw surfaces are produced. The raw surface represents the base of the bulla, and some remains of the thin-walled blister are usually discoverable at its margin. The so-called "post-aural eczemas" are examples of streptococcal impetigo.

DIAGNOSIS.—Impetigo is sometimes mistaken for eczema. In impetigo the crusts are larger, and their removal reveals a superficial excoriation; there is no itching; the eruption is contagious and auto-inoculable. In eczema the crusts and vesicles are minute, and closely set upon a red, oedematous skin; itching is marked; and the eruption is not contagious.

A crusted *lupus vulgaris* must not be mistaken for an impetigo. The long history in such a case should make one suspect lupus (q.v.).

The **TREATMENT** of impetigo contagiosa consists in bathing off the crusts, and then applying a mild antiseptic lotion or ointment to the raw surfaces. Lotion: hydrag. perchlor. (1 in 4,000) and usq. hydrarg. ammon. chlor. (gr. x. ad ʒi) are



FIG. 168.—IMPETIGO CONTAGIOSA (*Staphylococcus Pyrimus*). SHOWING THE "STUCK-ON" CRUSTS.

efficient applications. The removal of the crusts must be thorough, and must be done several times a day. This is a very important part of the treatment, for it is useless to apply antiseptics unless the crusts are removed.

Bullous Impetigo.—In ordinary cases of impetigo, bullæ may occasionally be seen upon the palms and soles, or around the finger-nail in the form of a softness. The reason for this is that the horny epidermis is thicker in these parts, and the serous exudation is retained as a blister instead of drying up into a crust. Sometimes in young children, or, during the hot weather, in a widespread impetigo in an older child, the majority of lesions may remain as bullæ instead of forming crusts, so that such cases are often mistaken for pemphigus. This sometimes happens in extensive impetigo associated with scabies. It occurs particularly, however, in newborn infants who become infected at birth from the midwife or nurse, and it is then called pemphigus neonatorum—a name which was applied to these cases before their true nature was understood. Before the days of aseptic midwifery "pemphigus neonatorum" was of common occurrence, often in large epidemics. Babies with

this affection often die, for the whole skin surface rapidly becomes involved. Large bullæ and excoriated areas form in a few days, and the infant succumbs to septic absorption or to a general streptococcic infection.

The **DIAGNOSIS** of bullous impetigo is to be made from pemphigus. Often the diagnosis can only be made when it is found that the former clears up under baths and antiseptic treatment, while in the latter bullæ continue to appear in spite of these measures.

The diagnosis of pemphigus neonatorum from syphilitic bullous eruption, with which it is often confused, is made upon the following points: The syphilitic baby is wasted, the eruption is present upon the palms and soles, and the bullæ rapidly dry into reddish-brown scabs; they are usually associated with copper-coloured macules on the face and buttocks.

The **TREATMENT** of all these forms of bullous impetigo consists in placing the child into a warm bath several times a day, and dressing the eroded surfaces with a mild antiseptic ointment, such as ung. hydrarg. ammon. chlor. (gr. x. ad 3i.) or boric acid ointment. If these measures are carried out thoroughly and early, the eruption rapidly clears up, and recovery takes place.

Ecthyma.—In children who are badly nourished, the lesions of impetigo may sometimes take on a more serious character. Instead of remaining as superficial blisters, crusts, and erosions, the inflammation extends more deeply, and ulceration takes place. There are then formed dark, opaque, and dirty-looking crusts, with a red, infiltrated halo, and, upon removal of the crusts, punched-out ulcers. After antiseptic treatment, the infiltration of these lesions remains for some time in the form of dull red, flatish nodules which may simulate multiple lupus (q.v.).

Ecthyma is not uncommon as a complication of scabies or of lichen urticatus, probably by infection from scratching.

TREATMENT is by baths, and, in situations where they may be conveniently applied, hot acid fomentations. As the wounds become cleaner, an ointment (hydrarg. ammon. chlor., gr. xv.; resorcin, gr. x.; ung. zinci, ad 3i.) may be used to complete the cure.

Dermatitis Gangrenosa Infantum is an eruption which sometimes follows varicella in infants or in debilitated children. The varicella lesions, instead of drying up, become inflammatory, and rapidly extend as deep punched-out ulcers under a blackish crust. Such cases are often fatal. If recovery takes place, vacuiform scars will be left. *Streptococcus pyogenes* and *Bacillus pyocyaneus* have been found in the lesions, and also in the blood, in some of these cases.

Under **TREATMENT** by prolonged soaking in a warm bath, followed by application of lotio hydrarg. perchlor. (1 in 2,000), the ulcers rapidly heal, and if carried out early the child's life may be spared.

Vacciniform Dermatitis of Infants is a rare form of eruption, probably of impetiginous (streptococcic) nature, which occurs about the genitals in infants. Primarily the lesions are tense bullæ, pea-sized to finger-nail-sized, few in number, and situated about the genitals and perineum. The bullæ quickly rupture, and excoriated discs or sharply-margined circular ulcers are formed, which, unless one

is familiar with the eruption, are sure to be mistaken for syphilitic sores. But the children show no other signs of syphilis, the lesions are without induration, and there are no coppery macules on the buttocks, face, or hands and feet. The result of local treatment by mild antiseptic lotions and ointments will confirm the diagnosis that they are local infections and not syphilitic ulcers.

Dermatitis Exfoliativa Neonatorum (Ritter's Disease).—This is a very rare form of eruption. It is now recognized as being bullous impetigo (pemphigus neonatorum) in which the exudation is little marked, so that scales are formed rather than bullae. True "dermatitis exfoliativa," occasionally met with in older children, is described elsewhere (p. 1157).

REFERENCES.

IMPETIGO.

SABOURAUD: *Art. Impetigo: La Pratique Dermatologique*, ii, 256; *Annales de Derm. et de Syph.*, 1900, i, 62, 328.

VACCINIFORM DERMATITIS.

FOX, T. COLCOTT: *Brit. Journ. Derm.*, 1907, xix, 131.

DERMATITIS EXFOLIATIVA NEONATORUM.

SKIFFER: *Brit. Journ. Derm.*, 1901, xiii, 75 (case with literature).

Impetigo of Boeckhart (Staphylococcic Impetigo).—Boeckhart's impetigo is due to infection of the skin by *Staphylococcus pyogenes aureus*. The eruption consists of pustules arising in and around the hair follicles. The term includes all follicular eruptions due to staphylococcic infection. The lesions may vary in size from pinhead-sized pustules up to large "boils," and they may be few and scattered or closely set over a limited area. In children this form of impetigo is met with mainly under the following conditions:

1. Associated as a secondary infection with pruritic affections, such as scabies and lichen urticatus.
2. As a secondary infection of impetigo contagiosa.
3. As the result of the application of poultices or other dirty dressings to boils or other pustulating lesions, by which means the infection is spread over a larger area.
4. On the scalp in cases of ringworm treated by strong irritant applications.

TREATMENT.—These impetigos are best treated by frequent bathing of the part affected with hot water, followed by the application of an antiseptic lotion (lydiaz, perchlor., gr. i.; resorcin, gr. xxx.; aq. ad 5viij.). The larger lesions (boils) should be incised with wet boric lint, covered with oiled silk, and incised when softened. Vaccines of *Staphylococcus aureus* may be employed in doses of 10,000,000, repeated in increasing amounts every four days, but in children they are seldom required.

Multiple Subcutaneous Abscesses in Infants.—In infants a form of streptococcic infection is sometimes met with secondarily to a bullous impetigo or to an eczema. Numerous subcutaneous fluctuating swellings of the size of a pea to a walnut appear widely scattered over the body. Some of these become reddened at one part when the skin becomes involved, but they seldom break spontaneously. It is uncertain



FIG. 120.—[Infected] OF BOCKHART (Staphylococcal Infection).—SHOWING FOLLICULAR PUSTULES.

whether these abscesses are the result of external infection through the hair follicles, or whether they are due to a blood-carried infection.

THE TREATMENT is to incise each abscess. After evacuation of the pus they rapidly clear up. Incision of the abscess should be followed by a warm bath and bath.

REFERENCE.

RECAUT: Abscess Multiple de la Poie. *Traité de Mal. de l'Enfance* (Blanchet et Coenly), 1903, iv, 305.

Seborrhœa and Seborrhœic Dermatitis.—It was formerly the custom to divide seborrhœas into (1) oily seborrhœa, and (2) seborrhœa sicca. It is only to the first group that the name rightly belongs. Seborrhœa oleosa indicates a hypersecretion of the sebaceous glands, such as is seen at puberty in association with acne

vulgaris and upon the scalp of bald-headed persons. It does not occur in children. The terms "*seborrhoea sicca*" and "*seborrhoeic dermatitis*" were first used owing to a misunderstanding of the nature of the eruptions which they were intended to describe. The scaliness which is a feature of these eruptions was at one time thought to be dried sebum; but although it is now known that the scales in these eruptions are made up of imperfectly cornified horny cells, the result of inflammatory conditions involving the epidermis, the old name is still retained. As a matter of fact, there are many eruptions, probably of different nature, to which this term is still sometimes applied. They include the following:

1. *Persis Caput*.—This is a wax-like coating sometimes seen on the scalp in young babies. It is a relic of the waxy coating which covers the body of the foetus, and is made up of cells probably derived from the horny layers of the epidermis.



FIG. 171.—CRUMBLY ERUPTION.

Folds of the scalp are covered with scales and small crusts, beneath which there is a raw, weeping surface. These cases are distinguished from eczema by the absence of itching and in that the eruption is not made worse by local irritants. They require strong antiseptic applications for their cure, and are often very intractable.

2. *Pityriasis Capitis*, or "*dandruff*" or "*scalp*," is often seen in children of from five to six years of age and onwards. There is a scaly condition of the crown of the head, the sides and back of the head being free. The scales are dry and powdery. A micro-organism, known as the bottle bacillus, and probably the cause of the exfoliation, is found in the scales. This affection is of importance (1) because it may be mistaken for ringworm (see diagnosis of ringworm); (2) because, if allowed to continue, it is apt to pass into a more markedly inflammatory form of "*seborrhoeic dermatitis*," at or after puberty, and then to lead to early baldness.

TREATMENT.—Frequent washing with soap and water is all that is required to remove the complaint.

Pityriasis Alba or Pityriasis Simplex.—Children are liable to dry, pinkish, slightly scaly patches on the face, especially about the mouth, and due to strong alkaline soaps. These disappear when the use of such soaps is discontinued.

There is also another form of pityriasis alba in which similar patches occur as the sequelae of impetigo contagiosa, or are associated with a fissure or crack at the side of the nose or below the lobe of the ear. In these cases the eruption is of streptococcal origin (furfuraceous impetigo), and is cured by local antiseptics:

Resorcin	gr. x.
Liq. picis carb.	℥.
Ung. hydrag. anison. chlor.	℥i.
Vaseline	ad ℥i.
Rt. sig.					

Chronic Forms of Impetigo Contagiosa on the scalp and elsewhere, especially in the flexures or behind and around and ears, are often mistaken for "seborrhoeic



FIG. 172.—SEBORRHOIC DERMATITIS IN AN INFANT.

The eruption consists of circumscribed red areas covered with greasy scales. The favorite situations are the flexures, especially the naso-labial folds, the axillae, and the groins, from whence it may spread widely over the buttocks and thighs.

dermatitis." In these cases the scalp becomes thickly covered with scales and crusts, or scaly and crusted areas may be present in the other situations mentioned. This condition is well illustrated in the photograph (Fig. 171). The important practical point is not to mistake them for eczema. They are more sharply margined than true eczema, they do not itch, and they require strong antiseptic applications for their cure. Ointments containing tar and resorcin will be found most useful:

Olei cadmi	℥ss.
Resorcin	gr. xv.
Ung. hydrag. anison. chlor.	ad ℥i.

Seborrhoeic Dermatitis or Eczema Seborrhoeicum in Infants.—A form of eruption having the characters detailed below is of fairly common occurrence in infants. Upon the vertex of the scalp there are irregular patches, or one large patch, of yellowish, greasy-looking scales. Upon the face, especially in the naso-labial folds, or extending farther on to the cheeks, on the neck, or behind the ears, there are sharply-circumscribed reddish areas covered with yellowish scales, or in parts raw and oozing. In the groins there are similar areas, or the whole region covered by the nappin may be occupied by one large red, raw or scaly, area. The eruption

comes out quickly (Fig. 172). The mother is usually affected with "seborrhoea capitis." These eruptions clear up rapidly on the application of a mild sulphur ointment, and the result of the treatment confirms the diagnosis of "seborrhoeic dermatitis." It is probable that these eruptions are due to an infection by a mild strain of staphylococcus, and that they are of the same nature as seborrhoeic dermatitis, seborrhoea capitis and corporis—flannel rash, of adults.

REFERENCE.

ANAKSON, H. G. : Brit. Journ. of Derm., 1909, xxi, 37.

Verrucae, or Warts, are small growths composed of epithelium with a central axis of bloodvessels and connective tissue.

ETIOLOGY.—They are infectious and auto-inoculable, and it is probable that they are due to a micro-organism as yet undiscovered.

SYMPTOMS.—They occur in two forms: the common wart and the plane wart. The favorite sites of the common wart (*verruca vulgaris*) are the hands, face, and scalp. They may be single or numerous, and vary much in size and shape. They may be flat and disc-like, cylindrical, filiform, or broken up into lobes. Apart from the disfigurement they cause, warts may be troublesome, in that they are liable to get torn and bleed. Or they may become inflamed and suppurate, from the entrance of pus-coeci when they are torn. The evolution and course of warts is capricious. They may come slowly or rapidly. They may remain for months or years. They may disappear gradually or suddenly. Plane warts (*verruca plana juvenilis*) occur mostly in children, scattered over the face or lozems. They are usually very numerous, and consist of small flat, disc-like elevations, of the size of a pin's head or smaller, with angular margins, and pale brown or of the colour of the skin. They may be associated with the larger common warts. They may be very puzzling to those not familiar with them, and are sometimes mistaken for lichen planus, although, apart from the fact that lichen planus is very rare in children, the paler colour of the warts (lichen planus papules being dusky red), and the association with one or more common warts, should prevent this mistake.

TREATMENT.—The cure of warts is not always easy. Their tendency to sudden spontaneous disappearance probably accounts for the reputation of many so-called "cures." One of the best applications is a paint of glacial acetic acid with 1 per cent. of perchloride of mercury. This is applied daily with a pointed match-stick until the wart dries and falls off. Freezing with CO_2 snow is also an effectual method. A stick of solid CO_2 is cut to the exact size of the wart and firmly pressed on to its surface until the skin around the base of the wart, begins to be frozen. This may occupy thirty seconds to two minutes, according to the thickness of the wart. A blister forms, and lifts the wart from its base. The only objection to this treatment is that the blister may be painful and inconvenient. When not numerous, warts may be removed by X-rays. The skin round each wart is protected with lead-foil, and rather more than one full Sabouraud panicle dose is given to the exposed warts. If the dose has been sufficient, the warts drop off in the course of a week. The best application for plane warts is ung. acid. salicylic., gr. xv. ad 5i.

Molluscum Contagiosum is a form of wart in which there occurs a peculiar degeneration of the epithelial cells of which it is made up. They are no doubt infectious. The growths are smooth, tense, shiny projections, with a central depression which was formerly mistaken for the mouth of a sebaceous gland, but which the microscope shows to be due to the degeneration and softening of the cells of the central part of the growth. Like warts, mollusca may suppurate.

The TREATMENT consists in incising the projection and pressing the base between the thumb-nails. The growth shells out, leaving a bleeding cavity in the epidermis. The bleeding soon ceases, and the little wound heals without any scar.

REFERENCE.

GILLowAY, J.: Oxford Albert and Edleston's System of Medicine, 1917, ix. 633 (good bibliography).

Tuberculosis of the Skin.—Tuberculous affections of the skin may be divided into—

1. Those which are known to be due to the local presence of the tubercle bacillus.

2. Those which are believed to be the result of the toxins of the tubercle bacillus. The first group includes lupus vulgaris and scrofuloderma. The second comprises lichen scrofulosorum, acne scrofulosorum, and erythema induratum scrofulosorum, these latter now being classed together as toxic-tuberculoses.

Lupus Vulgaris is the most characteristic form of tuberculosis of the skin. Its study in childhood is of special interest and importance, because it is at this time that it usually first makes its appearance, and that it can be most readily checked and its subsequent ravages prevented.

Ætiology and Pathology.—Lupus is more common in children of the poor than in better-class patients. Generally, infection takes place locally through the skin or mucous membrane, probably from pathogenic persons by kissing or by soiled handkerchiefs, or else by the patient's own fingers. This would account for the frequent occurrence of the disease on the cheeks and nose. Infection of the skin may also take place around sinuses from tuberculous glands or bone. Multiple lesions scattered over the body sometimes occur after measles, probably by entrance into the blood-stream of bacilli from a softened tuberculous focus. The lesions of lupus consist of granulomata with giant cells. The tubercle bacillus is found with difficulty, but its presence can be proved by animal inoculations.

Symptoms.—Lupus vulgaris commonly appears before puberty and after two years of age. It often occurs on the face, especially upon one cheek or on the nose, by infection from the nasal mucous membrane. The knees, buttock, hands, or other parts, may also be the seat of lupus, either in single patches or in multiple lesions. The eruption first appears as a small lupus nodule, or as a little group of nodules, but often when the patient first comes under observation the disease has already spread into a dull red, slightly raised infiltrated patch. Sometimes the patch may be crusted so that its real nature is obscured, and only its slow progress suggests lupus. The lupus nodule is the essential or primary lesion. It is a reddish-brown nodule pinhead-sized to bean-sized with semi-transparent

aspect. A patch of lupus is made up of a collection of such nodules, generally blended together, but if firm pressure is made over the patch with a lens or a watch-glass the nodules stand out as separate brown areas. They cannot be made to disappear, but, the surrounding erythema being pressed away, they show up more distinctly. This glass-pressure test is very useful for the diagnosis of lupus, and also as a means of discovering whether there are still any nodules left in the scar of a patch which has been treated.

After a patch of lupus has existed for some time, or at an early stage in some cases, the nodules may break down and ulcerate, and the ulceration generally becomes covered with crusts. In neglected cases the disease may, during the course of years, spread widely over the face, and involve the eyelids and conjunctive, destroy the cartilage of the nose, and attack the mucous membranes of the lips, producing great deformities.

Sometimes lupus begins in the mucous membrane of the nose, and invades the skin of the nose secondarily by direct extension through the cartilage. When



FIG. 172.—LUPUS VULGARIS: A PATCH OF TWELVE MONTHS' DURATION, MADE UP OF ABOUT A DOZEN LITTLE NODULES.

The nodules are beginning to break through the epidermis and to form crusts.

If lupus attacks the extremities or hat-locks, it is often of a warty or verrucose type (*lupus verrucosus*): instead of the characteristic lupus nodules, there are firm, closely-set warty granulations, which cover the surface of the patch and hide the dermic infiltration. A not very uncommon form of lupus is that in which there are many patches scattered over the body, face, and limbs. These cases generally date from a few weeks or months after an attack of measles (*multiple lupus after measles*). There at first appear numerous papules, which are sometimes mistaken for the rash of chicken-pox. After a time some of these papules develop into lupus nodules, while the majority disappear. The patches which remain eventually spread into typical lupus patches, and a patient may have from three or four to a score or more of such patches in various parts of the body. These multiple patches may sometimes become crusted, simulating impetigo; sometimes scaly, simulating psoriasis; and sometimes warty—*lupus verrucosus*.

DIAGNOSIS.—The recurrence in a child of an inflamed patch which persists for months or years should always raise the suspicion that it may be lupus. Careful examination should be made for lupus nodules; and if the patch is crusted, fomentations should be applied for some days to remove any secondary infection, and so reveal the lupus nodules if present.

TREATMENT OF LUPUS.—The methods of treating lupus are very numerous. The following are those in common use :

1. Excision.
2. Finsen light.
3. X-rays.
4. Selective caustics.
5. Tuberculin.

The method adopted must depend upon circumstances. Small early patches may be excised. Finsen light gives the best cosmetic results, but it requires special apparatus and is very slow. It is nevertheless the ideal treatment for lupus on the face. X-ray treatment is suitable only for ulcerated or warty lupus, not for patches of lupus nodules. A patch of lupus should never be scraped, as the disease nearly always returns in the scar. One of the most generally useful methods is by selective caustics, and the most convenient way of application is in the form of plaster-mulls; these are plasters consisting of a medicament with a rubber basis spread upon muslin. Plaster-mulls (Beierdoef), acid salicylic, c. cresoeti, Nos. 76, 78, 80, and 82, are commonly employed. The plaster is cut to the size of the lupus patch and worn continuously for twenty-four hours. At the end of this time it is removed, the part is bathed, and a fresh plaster is applied. After a few applications the nodules ulcerate, until eventually the whole of the diseased area is removed by the plaster, the salicylic acid picking out the diseased parts and not harming the healthy skin. The application is painful, and may have to be discontinued now and then for several days; but the treatment should not be stopped entirely so long as any ulceration occurs, the sores being allowed to heal up under the plaster. In some few cases of lupus good results are obtained by tuberculin. Personally the writer prefers to use Koch's old tuberculin in weekly doses gradually increased. Three dilutions are made : 1 in 1,000; 1 in 100; 1 in 10—in normal saline solution. The treatment begins with 1 to 2 minims of the 1 in 1,000 dilution. The dose is increased by 2 minims every four days until it becomes too bulky; then the 1 in 100 dilution is used, and finally the 1 in 10, or even the pure tuberculin. The dose is increased so long as there is no febrile reaction.

Scrofuloderma is the name given to the lesions which result from infection of the skin from some deep-seated underlying focus of tubercle, such as tuberculous glands or bone disease. It also includes tubercleous granules, which are subcutaneous abscesses having no immediate connection with deeper lesions, but produced, like multiple lupus, by entrance into the blood-stream of tubercle bacilli from some hidden central focus. The most common situation of scrofuloderma is on the neck or groin, in association with breaking-down tuberculous glands in this situation; they may also occur over tuberculous lesions in joints and bones, or along the course of lymphatics which drain tuberculous lesions, or over an abscess of the lachrymal sac (Fig. 174).

Scrofuloderma is characterized by the soft and friable structure of the lesions

as contrasted with the firmer lupus nodules. They may appear as gelatinous-looking masses covered with thin epidermis, or as gummata with central purulent contents and walls lined with soft granulation tissue, which may burst and give rise to indolent granulating ulcers. There is some reason to believe that these lesions are the result of the combined action of the tubercle bacillus and of pyogenic micro-organisms.

In the TREATMENT of these lesions, their dependence upon more deep-seated tuberculosis must be considered, and the question of surgical treatment of glands, bones, or joints, discussed. So far as the skin affection is concerned the X-ray treatment is ideal, with lesions healing up after a few "pastille" doses. Even the underlying glands or the bone trouble—if in the smaller bones—may be benefited, and sometimes cured, by the X-rays.

Tuberculoid or Toxi-Tuberculoid.

—"Tuberculoid" is a convenient term for certain eruptions of the skin, long recognized as closely related to tuberculosis, but in which the actual presence in the lesions of the tubercle bacillus has not been satisfactorily demonstrated either by the microscope or by animal inoculation. The histology of all forms is similar, consisting of a cell exudation round the bloodvessels, resembling that found in a true tubercle nodule, though generally with more scanty formation of giant cells. It is suggested that these lesions are due to the toxins derived from tubercle present in some other part of the body, but recent research tends to support the idea that they may be due to dead or attenuated tubercle bacilli.

The following affections belong to this group: lichen scrofulosorum, acne scrofulosorum, and erythema induratum scrofulosorum (Bazin's disease).

Lichen Scrofulosorum.—The eruption is seen in children who are the subjects of enlarged glands, scrofuloderma, or other forms of tuberculosis; but it may occur without obvious tuberculosis. Its practical importance is that it is an indication for a thorough medical examination.

PATHOLOGY.—The eruption consists of small papules of reddish-brown colour arranged in groups. Each papule is situated at the mouth of a hair follicle, so that they are evenly spaced over the patch. The groups or patches vary in



FIG. 174.—SCROFULODERMA.

The lesion on the cheek was the result of infection of the skin from a tuberculous abscess of the laryngeal sin; that on the hand was secondary to tuberculosis of the metacarpal bone.

size from areas comprising a few papules to patches an inch or two in diameter. They occur chiefly on the trunk, and there may be half a dozen or more such patches. The eruption does not itch; it may remain without change for months, and may eventually disappear without a trace or may leave pigmented spots or small scars.

TREATMENT.—Cod-liver oil and the usual hygienic treatment for tuberculosis. Locally unguentum acidi salicylici, gr. x. ad ʒi. may be applied.

Acne Scrofulosorum (now often called "papulo-necrotic tuberculide").—The eruption differs from that of lichen scrofulosorum in the size of the lesions and in the manner of their distribution. It occurs particularly upon the extremities, and especially upon the hands and forearms, the buttocks, legs, and feet, and consists of numerous firm, dusky red papules, crusted papules, and if the eruption has been of some duration, punched-out scars. The papules are pinhead to bump-seed-sized at first, and more easily felt than seen; then they become more prominent, and a crust forms at their summit. If the crust be removed, a small ulcer is found beneath, but generally the crusts dry up and fall off without exposing the ulcer, but leaving a punched-out scar behind. The evolution of a nodule occupies a few weeks, and generally lesions at all stages are seen together. A characteristic feature of these cases is that the hands and feet are cold and blue (childlain circulation). The eruption is sometimes associated with lupus, with tuberculous glands, or with bone affections, and it may coexist with lichen scrofulosorum. Scrofulous leucitis is not uncommonly present. The eruption may last for years. It generally improves or disappears in the summer, and breaks out afresh in the winter.

DIAGNOSIS.—The complaint is recognized by the distribution, the blue extremities, the scattered papules going through the stages of necrosis, crusting and scarring, and the presence—sometimes—of definite tuberculous affections. The eruption differs from multiple lupus in that the lesions show no tendency to spread by infection of surrounding skin, as is the case in lupus.

TREATMENT.—With rest in bed the eruption generally rapidly clears up, but often only to return when the child gets about again. The usual treatment for tuberculosis is indicated and any simple mild antiseptic ointment.

Erythema Induratum Scrofulosorum (Razin's Disease).—This is a tuberculide seldom seen except in young girls who do much standing. It occurs on the calves and ankles, as nodules pea-sized to filbert-sized, which go through the same stages as those of the tuberculides just described—namely, necrosis with ulceration and crusting, followed by scarring. As with other tuberculides, its importance rests in the indication that tuberculosis is present. It is also liable to be mistaken for erythema by those unfamiliar with it.

Lupus Erythematosus is so rare before young adult life that it need not be considered here.

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 SUGIURA, J. H.: Seven Years' Experience of the Finest Treatment. *Lancet*, London, 1908, i, 912.

The Skin Eruptions of Congenital Syphilis.—Congenital syphilis is dealt with elsewhere, and will be here considered only in so far as the skin eruptions are concerned. In congenital syphilis there is no primary sore, as in the acquired disease. The skin eruptions are usually confined to the period between six weeks and three months of age. Less often the child is born with an eruption, which then takes a particular form (bullous syphilide), almost invariably fatal. At or about two years of age it is not uncommon to find large condylomata at the anus, and, still later, though rarely, at the age of ten years and upwards, there may be elective tertiary lesions on the face, which simulate those of tuberculosis. The bullous syphilide, formerly called "syphilitic pemphigus," is not to be confused with pemphigus neonatorum, which is a streptococcal infection. The syphilitic eruption is present at birth and consists of reddish-brown plaques or flat papules, together with flaccid bullae, which may occur in all parts, but particularly on the face, the palms and soles, and about the genitals. The eruptions which appear some weeks after birth are the more common. The infant is then born apparently healthy, and at the age of four to six weeks the eruption appears together with snuffles, hoarseness of cry, and fissures of the lips or at the angles of the mouth; the skin becomes "earthy" in colour, and the infant loses its plumpness. The eruption consists of sharply circumscribed, flat, disc-like circular patches, at first red in colour, and later of a brownish or "coppery" tinge. These patches are seen upon the face, around the nose and mouth, upon the buttocks, thighs, and genitals, and upon the soles and feet. In severe cases the skin may be generally involved, but the parts mentioned are always the seat of the more profuse eruption. Sometimes these patches are more infiltrated, and form flat papules, or they may be scaly or crusted in parts, or run together on the buttocks into large sheets. When near mucous membranes, or in the moist flexures, they may become excoriated or ulcerated.

DIAGNOSIS.—They are to be distinguished from the simple napkin erythema, from seborrhoeic dermatitis, and from vacuiform dermatitis of infants. The more common error, however, is to mistake these eruptions for that of congenital syphilis. (For points in diagnosis see p. 1125.)

Herpes Zoster (Zona) is not an uncommon affection in children. Statistics from a children's clinic showed a proportion of 1 to 160 of all skin cases.

ÆTIOLOGY.—The eruption depends upon an inflammation in one or more posterior spinal root ganglia. It is possibly of microbial origin, and has been compared with anterior poliomyelitis, in which the anterior grey matter is involved.

SYMPTOMS.—The eruption consists of groups of vesicles arranged in a broken band around one side of the trunk, or along a limb, or upon one side of the face, the areas involved corresponding to the distribution of the sensory fibres of one or more posterior nerve roots. The most common situations are the chest (*herpes pectoralis*), the lumbo-abdominal region (*herpes abdominalis*), the buttocks and upper part of the thigh (*herpes femoralis*), the neck (*herpes cervicalis*), and the forehead (*herpes frontalis*). The eruption appears suddenly in the form of erythematous patches, upon which there soon develop small vesicles, which rapidly enlarge, until each patch contains of a group of closely set vesicles split-pea-sized upon an inflamed base. There may be from one to a dozen or more patches, and each patch may consist of half a dozen to a score or more vesicles. The vesicles are at first clear, but they may become turbid before they dry up, which happens after a few days.

In a week or two the crusts fall, leaving a temporary stain, but usually no scar. In children there may be malaise and slight fever at the beginning of the attack, but there is seldom, if ever, subsequent neuralgia, as is often the case in adults. If the lesions are inflamed by scratching or by friction of the clothes, or infected, they may ulcerate and give rise to scarring. *Herpes frontalis* affects the region supplied by the supra-orbital division of the fifth cranial nerve, and is sometimes accompanied by conjunctivitis, ulceration of the cornea, and iritis. It may also be followed by scarring. *Herpes cervicalis* involves the back and shoulders, corresponding to the fourth and fifth cervical posterior spinal nerve roots. *Herpes pectoralis* occupies the width of two or more intercostal spaces, most often corresponding to

the third to eighth dorsal roots. *Herpes abdominalis* corresponds to the sixth dorsal to the twelfth dorsal, and *Herpes femoralis* to the first and second lumbar roots. Herpes affecting the arm below the elbow or the leg below the knee, corresponding to the brachial plexus or to the sacral plexus, is uncommon.



FIG. 175.—HERPES ZOSTER PECTORALIS, SHOWING THE CHARACTERISTIC GROUPS OF VESICLES UPON AN ERYTHEMATOUS BASE.

PATHOLOGY.—Destructive inflammation with hemorrhages have been found in the posterior root ganglia, and lymphocytosis of the cerebro-spinal fluid has been demonstrated. There are found in the vesicles of herpes certain bodies which have been described as "balloon cells," but it has been shown that these bodies are nothing more than swollen epithelial cells. The exact relationship of the vesicular eruption with the inflammation of the corresponding root ganglia is not known.

DIAGNOSIS.—The diagnosis is easy. It should be borne in mind that herpes zoster may also result from injuries and disease involving posterior nerve roots, as in spinal cancer, in spinal meningitis, or in sarcoma of the spinal region, and that the eruption is not uncommon in persons taking arsenic.

TREATMENT.—The eruption should be protected from friction by a pad of cotton-wool bandaged on after powdering the skin with starch and zinc oxide powder. If there is elevation, boracic acid fomentations should be applied.

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 READ AND CAMPBELL: *Pathology of Herpes Zoster and its Bearing on Sensory Localization*, 1900.

Herpes Febrilis (Herpes Recurrens) is an eruption of vesicles in groups, most often seen about the lips and nose in association with a common cold. It occurs also with various other febrile affections, particularly with gastric disturbances, tonsillitis, pneumonia, and cerebro-spinal meningitis. It may occur also upon the cheek,

chis, neck, ear, the mucous membrane of the mouth, the buttock, or a finger or a thumb. It tends to recur at intervals of months or years, and generally, but not always, affects the same region at each recurrence.

The eruption consists of one or more groups of vesicles, of the size of a pin's head to that of a split pea, upon an erythematous base. The onset is attended by



FIG. 176.—HERPES FASCIUM—RECURRENT HERPES. A SEVERE EXAMPLE OF FACIAL HERPES.

The boy had had several attacks. There was a temperature of 100° F. and some malaise, but there were no other symptoms. (Photograph by Dr. Halden Davis.)

some burning or feeling of tension at the seat of eruption. After a few days the vesicles dry up into crusts, and the crusts eventually fall off without leaving a scar.

The PATHOLOGY of the affection is unknown.

DIAGNOSIS is as a rule easy. The tendency to recurrence often gives a clue.

TREATMENT.—An attack may sometimes be aborted by a few grains of quinine taken on its first appearance, or by rubbing on a strong sulphur ointment, or by painting on collodion. When the vesicles have formed, calamine lotion or zinc ointment may be applied.

C. ERUPTIONS OF TOXIC ORIGIN.

Erythematæ.—"Erythema" is a term which, used by itself, merely means a redness of the skin. It is only when employed with a qualifying word that it indicates any definite eruption or disease of the skin; for example, erythema solare, erythema ab igne, erythema pernio. The eruptions now to be described all belong to a group in which the erythema is due to some toxin circulating in the

blood, either a product of defective metabolism, or a food or drug poison, or the result of microbial infection. These toxic or infective erythemas include:

1. The exanthemata, scarlet fever, measles, German measles, etc.
2. Roseola and morbilliform and scarlatiniform rashes due to food toxins, serums, serum rashes, etc. (toxic erythemas).
3. Erythema nodosum.
4. Erythema multiforme.
5. Vaccination rashes.
6. Certain drug rashes.

With the exanthematic fevers we are not here concerned, except as regards their diagnosis from toxic erythemas.

ETIOLOGY.—These eruptions are common in children as the result of digestive disturbances, from ingestion of food poisons, especially of shellfish or fried fish, from injection of sera, from the use of horse-serum or diphtheria antitoxin, etc. (see Drug Eruptions, p. 1153).

Toxic Erythemas.—**SYMPTOMS.**—They may take the form of pale red macules, scattered over the trunk or face, sometimes called "roseol," or "rose rash"; of eruptions resembling the dull red crescentic or rounded patches of macules—morbilliform erythema; or of a widespread bright red punctate erythema like that of scarlet fever—scarlatiniform erythema.

DIAGNOSIS.—Their chief importance is that they may be mistaken for the rashes of specific fevers. They are distinguished from these rashes (1) by the absence of fever; (2) by their irregular distribution, often with a tendency to the sparing of the eruption in some regions while other parts are comparatively free; (3) by the evidence of poisoning by foods or otherwise; (4) by the absence of characteristic symptoms of the specific eruptions—the coryza of measles, the sore throat and strawberry tongue of scarlet fever, etc.

Erythema Nodosum.—**ETIOLOGY.**—This disease is more common in children and young adults than in older persons. By many it is thought to be a specific infectious fever (nodal fever), but the correctness of this view is not yet established. Similar eruptions may occur in the course of diphtheria, syphilis, cholera, rheumatism, cerebro-spinal meningitis, and tuberculosis, and by some the characteristic eruption is regarded as a symptom of different infections (see also Chapter XVIII., p. 955).

SYMPTOMS.—There appear upon the fronts of the shins oval, red, tender, and painful swellings of nodes, varying in size from an inch to several inches in length, the long axis of the swellings being in that of the limb. The nodes vary in number from one to two to a dozen or more on each leg. Rarely there may be smaller nodules on the forearms. The onset of the eruption is acute, and it may be associated with malaise, sore throat, shivering, and joint pains, and sometimes with swelling of the joints. There may be some rise of temperature, generally only two or three degrees, but occasionally more. Each node lasts about a week, passing, as it disappears, through the colours of a fading bruise. Fresh nodes may appear from day to day during a few weeks. The nodules do not ulcerate.

PATHOLOGY.—The lesions are of the nature of an erythema exudativum. They are due probably to a toxin, possibly of microbial origin.

DIAGNOSIS.—The diagnosis presents no difficulty.

PROGNOSIS.—Nephritis, otodecanditis, and meningitis have been recorded as complications, but in the vast majority of cases complications do not occur: recovery is complete, and recurrence is rare.

TREATMENT.—The child should be kept in bed, with a cradle to protect the tender nodes from the bedclothes. Lotio plumbi may be applied on lint, and salicylates should be given internally.

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 FACHON, F.: L'Erythème Nodieux chez les Enfants. *Paris Thèse*, 1901.

Erythema Multiforme.—A form of erythematous eruption which is characterized by its symmetrical distribution on the limbs and face, and by the exudative nature of the erythema.

ÆTIOLOGY.—The occasional occurrence of joint pains and swelling in this affection has suggested a relationship with rheumatic fever, but it is more probable that the eruption and the joint troubles are common symptoms of a general toxæmia of unknown origin, quite unrelated to rheumatism. The affection is not uncommon in children. Erythema multiforme forms one of a group of affections which include purpura rheumatica and Henoch's purpura. These affections are all characterized by the symmetrical distribution of the erythematous lesions on the limbs and face, accompanied by joint pains and fever.

SYMPTOMS.—One of the most striking features of erythema multiforme is its distribution—upon the backs of the hands, the forearms, and sometimes upon the sides of the face and neck, and on the legs. The eruption consists of disc-like erythematous patches of a dusky-red or violet colour, sometimes level with the skin (mere blotches or macules), but generally more or less raised into flat tubercles or papules, sometimes with sufficient exudation to produce vesicles or bullæ, and when occurring upon the legs, often with some hæmorrhage into the lesion. The erythematous patches may fade in the centre, so that rings are produced; and, a fresh papule appearing at the centre of the ring, an iris or target-shaped patch is formed. When the extending ring and the central part are bullous, the term *Acropus iris* is used.

The eruption generally lasts a few days to a week, and then fades; but recurrences are frequent, so that the complaint may be prolonged for weeks. There may be a slight rise of temperature with each fresh outbreak. Sometimes the eruption occurs in the mucous membranes of the inner sides of the cheeks and lips.

PROGNOSIS.—The prognosis is good in the milder cases, which are in the majority. But a feature of the affection is its tendency to recur in subsequent years.

PATHOLOGY.—The eruption is not merely the result of vaso-motor dilatation of the bloodvessels, as was at one time supposed. There is evidence of inflammation, in cell proliferation round the bloodvessels of the corium, and in active exudation into the tissues. It probably depends upon the circulation of a toxin, of unknown nature, possibly of microbic origin.

TREATMENT.—During an attack the patient is best in bed. No way is known of preventing recurrences, but salicin or salicylates internally, and lotio calaminæ or lotio plumbi subacetatis locally, are useful during an attack.

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Urticaria.—Urticaria, or nettle-rash, may occur in children as the result of local irritation or injury to the normal skin. Common instances are the wheals produced by stinging-nettles, by the bites of insects, by certain caterpillars, or by sudden blows, as by the end of a whip. Generalized transient attacks of urticaria may also occur as the result of ingestion of food poisons, shellfish or tinned meats particularly. Or certain foods—e.g., eggs, strawberries—may give rise to urticarial attacks in some children. An acute generalized urticaria may also occur as a symptom of septic poisoning from peat-ry pus.

SYMPTOMS.—The characteristic lesion, the wheal or nettle-rash, is well known. It consists of a pinkish, flattened, circumscribed elevation of the skin, arising suddenly, and generally transient, and accompanied by severe itching.

PATHOLOGY.—Urticaria is an inflammatory oedema due to the action of toxic bodies upon the smaller bloodvessels, and indicates a hypersensitiveness of the vessels and tissues to certain proteid poisons.

PROGNOSIS.—Acute cases generally subside in a few days, but cases beginning after some definite food poisoning may sometimes become chronic and last for months.

TREATMENT.—Careful inquiry should be made, with the object of discovering any offending article of food, especially eggs, cheese, strawberries, fish, veal, pork, etc. A saline aperient as follows is often useful in chronic cases: R Magnes. carb. ss ; sodii phosphatis ss ; sodii bicarb., ss ; pulv. sacch. alb., ss ; ft. pulv. S.: "Half a teaspoonful or less to be taken in water three times a day." Small doses of salicylate of soda (1 grain in water every two hours) may be given during an outbreak. Intestinal antiseptics (balel, β -naphthol) have been recommended, but personally the writer has seen no good results from these drugs; nor has he from calcium lactate. An empirical remedy, often successful, is a mixture containing liq. hydrarg. perchlor. R x-xx. taken three times daily. Locally any of the anti-pruritic drugs mentioned under *Lichen Urticatus* may be used.

Lichen Urticatus (Strophulus, "Red-Gum") is a form of nettle-rash or urticaria peculiar to childhood. The urticarial wheals are small in size, and centred by a papule or papule-vesicle. The urticarial part of the lesions is fugitive, and comes out chiefly at night; the papules are more lasting, and during the daytime may be present alone as a papular or papulo-vesicular eruption.

ETIOLOGY.—Lichen urticatus is an affection of infancy and early childhood. It is common between the ages of six months and six years. Few children escape it in some degree. It is generally supposed that chronic digestive troubles are responsible for this eruption—that it is due to a toxæmia of gastro-intestinal origin. But

evidence of digestive disturbance is not always to be found. The affection is generally worse in warm weather, and exacerbations may occur with the setting of a tooth.

SYMPTOMS.—Itching is the most prominent feature of the eruption, and it is usually for the itching, which keeps the child awake, that advice is sought. The eruption consists of rounded or oval wheals of from $\frac{1}{4}$ to $\frac{1}{2}$ inch or more in diameter, of a pale rose to a deep red colour. At the centre of the wheal is a raised firm papule, which may have at its summit a tiny vesicle, and sometimes the central papule may be replaced by a small clear vesicle. The eruption favours certain regions, particularly the lumbar region, the buttocks, the extensor sides of the arms and thighs, and the backs of wrists and hands. There may be a dozen such lesions up to an innumerable quantity. The wheals are seldom seen in abundance during the daytime, the eruption then consisting mainly of the central papules, a wheal being discoverable only here and there. But at night, as the mother will state, the child is covered with wheals (called, by women who do not know a nettle-sting, "histers"). There is often evidence of scratching in torn papules and scratch-marks, and there may sometimes be secondary impetigo.

PROGNOSIS.—The affection is difficult to cure, but children always lose the eruption as they grow older, and often, too, in cooler weather.

DIAGNOSIS.—The diagnosis must be made from (a) scabies; (b) varicella. A scratched, papulo-vesicular eruption in a baby or young child is generally scabies or lichen articatus. The diagnosis is given under Scabies (p. 1127). The vesicular form of lichen articatus is frequently mistaken for varicella, until the eruption has continued for several weeks. Varicella may be distinguished by the presence of lesions on the scalp and in the mouth, and by the fact that the varicella vesicles dry up into a characteristic black scab in a day or two.

TREATMENT.—Any defect in the general health must be corrected. The clothing must be light. Dyspepsia, constipation, or anaemia, especially require treatment. A simple diet and food at regular intervals must be insisted upon. Raw fruit or sweetmeats must be forbidden. For dyspepsia useful prescriptions are the ordinary bismuth, bicarbonate of soda, and magnesia mixtures. For constipation, $\frac{1}{2}$ to 1 grain of grey powder may be given every evening until the bowels become regular. For anaemia, syrupus ferri phosphatis, ʒss ad ʒi, in water three times a day. Cases sometimes improve with small doses of salicin (gr. iii. t.i.d.), with solid (gr. iii-v., t.d.s.) in milk, or quinine (gr. ii.) in sugar-coated capsules at bedtime. As external applications, the various anti-pruritic ointments and lotions may be tried, it being often necessary to change from one application to another. Such are: R β -naphthol, gr. x.; vaselin, ʒi. R Hydrarg. oxid. flav., gr. v.; vaselin, ʒi. R Sodii bicarb., ʒi; aq., ʒx. R Sodii bicarb., ʒi; aq., ʒx. R Liq. plumbi subacet., ʒi; aq., ʒx; Liq. picis carb., ʒi; aq., ʒx.

The warm bath is better given in the morning, as at night it is liable to increase the irritability of the skin. In bad cases the child may be better in bed, with the limbs wrapped in a zinc cream spread on butter-milk—R Zinc oxid., ʒii.; lanolin, ʒi; ol. olive, ʒi; aq. calcis, ʒi.

REFERENCE.

Fox, T. *Concise Brit. Journ. Dermat.*, 1899, i. 133.

Urticaria Pigmentosa is a somewhat rare affection, which makes its appearance generally in early infancy, and which remains until adult life, when it tends to fade away. It occurs in two forms—macular and nodular—which may coexist. The macules or nodules are generally thickly, sometimes sparsely, distributed over the whole integument. They are of a striking yellowish-brown colour. A characteristic feature is their tendency to become tumescent or urticarial on local irritation or from emotional excitement. There is no alteration of general health.

PATHOLOGY.—The cause and the true nature of the eruption is ill-understood. Some observers regard it as a form of urticaria, others as related to atavoid conditions. A peculiarity of the histology is that the lesions are made up almost entirely of mast cells, which are present but sparsely in other chronic inflammatory conditions. The nodular forms are distinguished from the lesions of congenital xanthoma by the presence of the urticarial element, and by the finding of mast cells in microscopical sections.

TREATMENT is directed to controlling the urticaria. No treatment has any influence upon the macules or nodules.

REFERENCES.

- FOX, T. COLCOTT: *Art. Urticaria Pigmentosa*, CHESB. Allbutt's System of Medicine, 1911, p. 234.
 LITTLE, GRAHAM: *Urticaria Pigmentosa*, *Brit. Journ. of Dermat.*, 1909, xvi, 255. (Good bibliography.)

Granuloma Annulare—Lichen Annularis (Nodular-Ringed Eruption—Colcott Fox).—This is a recently described eruption, but, now that it is recognized, it seems to be not very rare. It consists of smooth, whitish, sometimes red, projecting rings upon the hands or feet or buttocks in infants and children. The lesions begin as firm raised nodules, and expand into rings of $\frac{1}{2}$ to 1 inch in diameter. Sometimes the ring appears to be made up of a necklace of closely placed nodules. The rings may be single, few, or numerous. After lasting for some weeks or months, the lesions undergo spontaneous evolution.

ÆTIOLOGY AND PATHOLOGY.—The nature of the eruption is unknown. There is but little evidence to support the view that they are "tuberculides." Histologically the lesions consist of a cell infiltration in the cutis.

REFERENCE.

- LITTLE, GRAHAM: *Proc. Roy. Soc. Med.*, London, 1908, vol. i., p. 95 (Dermatological Section).

Drug Eruptions.—BROCHIDE OF POTASSIUM is often given to infants and children, on medical advice, or in the form of patent "soothing powders." An infant at the breast may also take the drug through the milk of the mother. Some children are peculiarly susceptible to the drug, and an eruption may be produced by very small doses. The eruption is a very striking one. It begins as a vesicular eruption, but soon the vesicles become solid and opaque, forming firm, whitish, tense, button-like projections of the size of a split pea up to an inch or more in diameter. It may be generalized on the face, trunk, and limbs, or may be more restricted in distribution. After a few days the lesions dry into crusts, which eventually fall off.

without leaving scars. If the drug be continued, the lesions may develop large granulations, so that fungating masses are formed.

DIAGNOSIS.—In the early stage varicella or pemphigus may be simulated, but when the lesions have become solid the eruption is so characteristic that it should not be mistaken for any other.

TREATMENT.—The eruption subsides when the drug is discontinued.

ANTIPYRIN RASH.—Antipyrin sometimes produces an eruption which closely simulates measles—namely, a generalized eruption of sharply-defined, closely-set red macules. The macules are, however, generally darker and more purple in colour than in measles, and there is no coryza and no rise of temperature.

BELLADONNA RASH.—Belladonna administered internally or applied externally may sometimes give rise to an eruption like that of scarlet fever. It is generally confined to the shoulders or upper part of the body; there is no rise of temperature, the pupils are widely dilated, and the child may be in a state of excitement. The throat may be congested, and there may be subsequent desquamation.

SEXUAL ERUPTIONS.—Following the injection of diphtheria antitoxin, of anti-streptococcal serum, and of other sera used for treatment, there may occur eruptions accompanied by other toxic symptoms. The eruptions commonly appear about ten days after injection, but they may come out on the first or second day, or as late as three or four weeks. They vary in type, and may be morbilliform, scarlatiniform, like erythema multiforme, or urticarial. A common form is in large erythematous or urticarial blotches, with a more or less generalized distribution. The eruption lasts from two days to a week. It may recur. There is usually a rise of temperature— 101° to 102° F., sometimes higher. There is headache, prostration, and often joint pains, or even effusion into the joints.

REFERENCE.

GOODALE, E. W.: *Rashes Due to the Administration of Animal Serum*. Clifford Allbutt and Holliston's System of Medicine, 1912, ix, 112.

Enema Rash.—An erythematous eruption occurs occasionally, both in children and adults, after the administration of an enema. Its chief importance is that it may be mistaken for the rash of one of the specific fevers. The tendency to enema rash is said to be less in children below six years of age than in those above that age. The pathology of the rash is uncertain. It is probably a toxic erythema due to the absorption of some constituent of the faeces by the large quantity of water injected, and not due, as has been suggested, to some poison in the soap; for it may occur after plain water injections.

SYMPTOMS.—The eruption consists of slightly raised, bright red, almost scarlet erythematous patches of irregular shape, separated from each other by normal skin. With these larger patches are smaller round, flat, papule-like patches, by the confluence of which the larger patches appear to be formed. The distribution is characteristic, on the fronts of the knees, the elbows, the buttocks, and the face about the cheeks and chin. It may spread to other parts, but tends to remain symmetrical, and to affect the extensor rather than the flexor surfaces. In some instances a more diffuse scarlatiniform erythema may precede the eruption de-

scribed. Itching is absent or slight; there is no rise of temperature, nor other constitutional disturbance. The rash appears usually twelve to twenty-four hours after the administration of the vaccine, but the incubation period may be as short as two and a half hours or as long as forty-two. It lasts from twelve to thirty-six hours.

DIAGNOSIS.—The occurrence just after the administration of an vaccine, the characteristic distribution, the absence of constitutional symptoms, particularly sore throat, coryza, pyrexia, and enlargement of the cervical glands, usually serve to distinguish the rash from that of measles, scarlet fever, or *rottheln*. Sore throat, pyrexia, and subsequent desquamation may, however, occur in rare instances of vaccine rash, in which case the diagnosis is extremely difficult.

REFERENCE.

SMITH, G. F.: Erythema Exanthematicum in Children. *Chil. Soc. Trans.*, 1899, XXXV, 11.

Vaccination Eruptions.—Vaccination against smallpox is often blamed for the appearance of eczema and other eruptions which may develop subsequently. There is no evidence that eczema, psoriasis, lichen urticatus, or any of the common



FIG. 177.—GENERALIZED ERYTHEMA AFTER VACCINATION.

The rash was most marked on the vaccinated arm, where it formed an continuous sheet of redness. It appeared eight days after vaccination.

eruptions to which children are liable are ever caused by vaccination. Neither is it possible, with the use of calf lymph, to inoculate a child with syphilis. Nor is there any danger of introducing tuberculosis, since the lymph is taken only from animals proved not to be tuberculous. With antiseptic precautions there can be no danger of introducing infections from outside sources. There remains only the possibility of infection of the wound at a subsequent date, and of certain transitory and harmless toxic erythemas.

Eruptions due to Local Infections.—1, Impetigo or erysipelas may arise as the result of infection of the vaccinia vesico-pustule if precautions are not taken to keep the wound clean, but the evils are to be avoided with proper care.

2. Up to the ninth or tenth day after vaccination re-inoculation on a different site is possible. For example, a patch of eczema, of impetigo, or of herpes may become inoculated from the original vesico-pustule. This is to be avoided by either refusing to vaccinate a child who has any erosion or eruption of the skin, or by keeping the vaccinated part well covered.

Toxic Erythematosa after Vaccination.—Erythematosa may appear after vaccination. They do not differ from toxic erythematosa due to other causes, and may be roseolar, macelliform, scarlatiniform, urticarial, or resemble erythema multiforme. Like "serum naibes," they generally appear after an incubation period of seven or eight days from the date of vaccination. They are of rare occurrence. The case depicted is an unusually severe example.

REFERENCE.

MOMM, M.: Vaccination Rashes. *Brit. Med. Journ.*, November 29, 1890.

Pemphigus is one of the less common affections of the skin.

ETIOLOGY.—The cause of pemphigus is unknown. Three theories are advanced—nervous, microbial, and toxic. The modern tendency is to regard it as due to a



FIG. 128.—PEMPHIGUS VULGARIS IN A CHILD OF TWO YEARS OF AGE.

toxæmia, either acting through the nervous system or directly upon the epidermis. The disease is said to be more common in adults, but the writer's own experience is that in proportion to other skin affections it occurs as often in children as in adults.

SYMPTOMS.—The eruption consists of blebs, which vary in size from $\frac{1}{4}$ inch to several inches in diameter, may occur upon any part of the skin, and are generally widely distributed over the whole body, sometimes sparsely, often profusely. The individual bullæ arise very quickly upon apparently sound skin. They are tense and clear at first, but may become floccid, and the contents turbid later. They may rupture, leaving a raw surface covered with a crumpled epidermis, or the contents may coagulate and form crusts. Eventually they dry up, leaving first red, then brownish stains, which finally disappear without any scar. The bullæ

often tend to come out in groups, and appear in crops, so that in severe cases the patient presents crusted lesions, flaccid turbid bullae, red and brown patches, and recent tense clear bullae. Constitutional symptoms may be absent, or there may be occasional febrile attacks due possibly to absorption from bullae infected by pus cocci.

PATHOLOGY.—There is nothing characteristic in the structure of the bullae. The blood often contains an excess of eosinophile cells, a feature which is regarded as indicating a toxæmia.

DIAGNOSIS.—It is of great importance to distinguish pemphigus from bullous eruptions due to a streptococcal infection. An extensive bullous impetigo will often simulate pemphigus, and it may be possible to make a correct diagnosis only by the result of treatment. A bullous impetigo will quickly clear up with antiseptic baths and antiseptic ointment and lotions; but in pemphigus fresh crops of bullae will continue to come out, in spite of this treatment. Bullous streptococcal impetigo may sometimes complicate scabies, and be mistaken for pemphigus. It must also be remembered that iodides and bromides may give rise to bullous eruptions.

PROGNOSIS.—A cure may result after one attack, but generally there are recurrences, with recovery after months or years, or the disease may be eventually fatal. The prognosis is much better in children than in adults.

TREATMENT.—Arsenic is often credited with good results in pemphigus, but patients do equally well if they are kept in bed and the lesions carefully tended. A daily soaking in a warm bath, to which is added common salt in the proportion of 1 ounce to each gallon of water, as being less irritating than plain water, is followed by dressing the lesions with liquid vasoline on thin rag. In severe cases small doses of tinct. opii may be given, not only to relieve the distress and soreness of the skin eruption, but because this drug seems also to have a good effect upon the disease itself.

Psooriasis.—Psooriasis is a common affection in children from the age of five or six years onwards.

ÆTIOLOGY.—Psooriasis is often a family disease, and one which tends to recur in the same patient. Although evidently an entity sharply marked off from other eruptions by its characteristic and very constant features, nothing is known as to its causation.

SYMPTOMS.—The subjects of psooriasis are generally in good health, and there are no subjective symptoms. The eruption consists of sharply-circumscribed patches of a dull red colour, covered with silvery scales. It is generally widely diffused, with more or less symmetrical distribution. It may appear on any part of the skin, but its favourite sites are the trunk, elbows, knees, and scalp. The eruption begins as small scaly points or patches, which extend at their margins to form sixpenny-piece-sized lesions up to discs an inch or two in diameter. Sometimes rings are formed by the fading of the central part of the lesion, but circinate patches are less common in children than in adults. In a typical case the silvery appearance of the scales at once suggests psooriasis, but in recent cases the patches may be red and smooth, and only show scales on scraping with the nail or a blunt instrument. It is characteristic of the lesions of psooriasis—(1) That the sharply-circumscribed patches grow by extension at their margins, not by coalescence of smaller patches. (2) That they remain always dry. (3) That on scraping the

scales become more obvious, until at length a level is reached where there are no more scales, but a dry, red, smooth surface, which shows bleeding-points on further scraping. (4) That the red patch which remains after the scales have been removed shows no infiltration, and fades on firm pressure—i.e., the lesion is a scaly macule, not a papule. On the scalp the patches are usually more thick and scaly. The nails may be pitted, or there may be scaly patches on the nail-bed, separating the nail at that part from the bed. But nail changes are not very common.

Other characteristic features of psoriasis are—(1) That it is often hereditary. (2) That it may recur again and again in the same patient. The eruption may last for several weeks or months, and then disappear spontaneously, or it may persist, with varying intensity, for years. There are no complications.

PATHOLOGY.—The most marked features of the pathological anatomy of psoriasis are—(1) The dilatation of the vessels of the papillary layer, with enlargement and prolongation of the papille (Alpine papille). (2) A catarrhal inflammation of the epidermis, resulting in oedema and imperfect cornification of the horny cells, so that these adhere to form scales instead of exfoliating normally.

DIAGNOSIS.—In a typical case the diagnosis presents no difficulty. When in doubt, the scraping test already described always gives certainty. The small-patch type of psoriasis in children may not be recognized as psoriasis until the apparent papules are scraped, when the typical silvery scales appear.

Prognosis is good for any one attack, but recurrences cannot be prevented.

TREATMENT.—As a rule neither dieting nor drugs have any influence on this eruption. In a very small proportion of cases arsenic internally seems to hasten the disappearance of the eruption, but its action is so uncertain that it is better to depend on local remedies. In some acute cases salicylates may be tried. In a patient who is subject to psoriasis, no drug will prevent recurrences. In all cases of psoriasis in children it is possible to remove the eruption by local measures. In mild cases, a daily soaking for half an hour in a brim bath, followed by applications of ung. acid. salicylic. (gr. xv. ℥ss. ad ℥i.) may be sufficient. In chronic cases with a few lesions, the best application is tar in the form of liq. picis carbonis, scrubbed into each patch once or twice a day with a stiff brush. In long-standing or



FIG. 179.—PSORIASIS.

extensive cases the child should be put to bed, and rubbed with chrysarobin ointment (ung. chrysarobii, B.P.). This ointment stains linen, so that old clothes should be worn. It produces painful conjunctivitis if it gets into the eyes, and therefore it should not be applied to the scalp, face, or hands. In some patients it results in an acute erythema of the skin, especially of the flexures, and it should at first be used tentatively on one limb. Afterwards it is rubbed into all the patches daily until the patches lose their scales and redness, and stand out as white areas on the normal skin, which is stained reddish-brown by the chrysarobin. The application should be continued for a few days until the white patches also become stained. Then a bath is given, but not during the treatment. The cure of an extensive eruption will take from seven to fourteen days. A tar ointment is used for the scalp, face, and hands, if these parts are affected (ol. cadini, 3i.; vasoline, 3i.). It must be well scrubbed into the patches with a piece of flannel.

Pityriasis Rosea (*insculata et circinata*) is a common though little known eruption, with, however, many characteristic features. It begins as a solitary pink, scaly patch upon the neck, chest, abdomen, arm, or thigh, which is known as the "herald patch." After a week or ten days the "herald patch" is followed by an outbreak of similar patches over the trunk, so that at the height of the eruption the whole trunk is covered with numerous rounded or oval pinkish, scaly patches, finger-nail-sized or larger. The patches are at first little more than pinkish macules. But soon the epidermis over them exfoliates in fine scales, which quickly fall from the central part, so that three zones are produced—a central yellowish zone of finely wrinkled epidermis, outside this a narrow scaly band, and at the margin of the patch a smooth pink edge. Often the eruption itches considerably. It is generally confined to the "vest area"—namely, to the trunk, neck, shoulders, and upper parts of the thighs; but sometimes it may involve the face and the more distal parts of the limbs. The eruption lasts usually about six weeks, and then gradually fades away. The lesions described are those of the common macular type. Less often the patches spread into circles, forming the so-called "circinate type."

ÆTIOLOGY AND PATHOLOGY.—In many respects the eruption suggests an exanthem. There is sometimes a slight febrile disturbance at the onset; it runs a definite course, and it occurs often in small epidemics. Nevertheless, there is no direct evidence that it is contagious, and no organism has been found in the scales. Histologically there is evidence of a superficial inflammation involving mainly the epidermis.

DIAGNOSIS.—The "herald patch," the characteristic distribution, and the patches with their three zones, make the diagnosis easy, though the eruption often puzzles those who are unaware of its existence. It is distinguished from psoriasis by these features and by the absence of the typical silvery scales of psoriasis. The circinate forms differ from ringworm of the body in their wider distribution and in the absence of fungus in the scales.

TREATMENT.—The patient should be given a warm bath daily, followed by immersion with ung. acid. salicylic. (gr. xv. ad 3i.). If pruritus is a prominent symptom, ol. cadini may be added to the ointment in the proportion of ℞. xx. ad 3i.

Generalized Exfoliative Dermatitis (*Dermatitis Exfoliativa*, *Pityriasis Rubra*).—Under this term may be included certain rare eruptions characterized by general-

ized intense reddening of the skin, followed by desquamation involving the whole epidermis. Cases vary in intensity and in chronicity. In some—classed as "recurrent scarlatiniform dermatitis"—the erythema and the subsequent desquamation are like that of scarlet fever, and may be accompanied by fever. In the more chronic forms the redness and the exfoliation persist for weeks or months. Itching is a prominent feature. The desquamation is excessive, so that the bed becomes filled with scales. There may be irregular fever or none. The only



FIG. 189.—DERMATITIS EXFOLIATIVA IN AN INFANT OF TWELVE MONTHS.

There was intense redness of the skin, abundant flaky desquamation, and some pruritus of six months' duration. Under local treatment with greasy applications continued for several months the condition gradually improved, and the child eventually completely recovered.

example of this disease met with by the writer in a child is that of which the photograph is here reproduced (Fig. 189). These cases are not to be confused with Ritter's disease, *dermatitis exfoliativa infantum* (p. 6).

ETIOLOGY.—In some instances this affection appears to follow the application of an ointment or lotion containing some substance to which the patient is hypersensitive, notably mercury and tar preparations. Other cases appear to be started by some drug given internally—mercury, quinine, salicylates. But frequently there is no obvious cause.

TREATMENT.—In view of the fact that these eruptions appear sometimes to be set up by drugs, the less drug treatment employed the better. The case here depicted recovered after many months under the free application of paraffinum liquidum and rest in bed.

REFERENCES.

- MICHAELIS, S.: On Dermatitis Exfoliativa Universalis. *Brit. Journ. Derm.*, 1885, i, 283.
 ANGELOU, H. G.: Generalized Exfoliative Dermatitis. *Clifford Allbutt's System of Medicine*, 1899, ix, 328.

Alopecia.—There are three forms of alopecia, or baldness, commonly met with in children. These are—

1. A diffuse alopecia after febrile or debilitating illnesses.
2. An alopecia in patches following localized inflammations of the skin.
3. Alopecia areata.

Congenital alopecia, either partial or general, may occur, but it is very rare.

Ætiology.—Alopecia results from an interference with the nutrition or destruction of the hair papilla. In most febrile alopecias and in post-inflammatory alopecias this is probably due to a toxin. Nothing is known of the cause of alopecia areata. There is little or no evidence to support the two theories of nervous and of parasitic origin. In the writer's view, the clinical and histological features suggest the possibility of a toxemia. There is a certain family tendency to alopecia, since often two members of a family are affected, though generally at long intervals or under circumstances which seem to preclude infection. This may indicate merely a family sensitiveness of the hair papilla to some toxin.

ALOPECIA FOLLOWING FEBRILE ILLNESSES.—DIFFUSE ALOPECIA.—After scarlet fever and measles, and, in fact, after any illness which leads to general malnutrition, there may be marked thinning of the hair of the scalp, though rarely to the extent of complete baldness. Such forms of baldness are not of serious moment, for the hair grows again on return of health.

ALOPECIA IN PATCHES AS THE RESULT OF LOCAL INFLAMMATIONS.—Bald patches may arise around the site of a boil on the scalp, or they may be produced by patches of impetigo of long standing. They may also result from spontaneous inflammation of patches of ringworm (kerion), or from inflammation artificially produced in the treatment of ringworm. In these cases the hairs become loosened as a result of the deep inflammation, and when the latter has subsided bald patches are left which closely simulate patches of alopecia areata.

DIAGNOSIS.—The history of previous inflammation or the fact that some inflammation is still present gives the clue to the nature of the bald patch, and all that is required in the way of treatment is to bathe off any crusts with warm water and apply a simple antiseptic ointment.

ALOPECIA AREATA is the name given to a special form of alopecia or baldness in patches. It is a common affection in children, and owes its importance (1) to the fact that it may be mistaken for ringworm, (2) that it occasionally becomes universal, and may even lead to permanent baldness.

The usual course of the affection is as follows: A small circular or oval bald

patch suddenly makes its appearance on the scalp, the hairs sometimes coming out in the course of a few hours. For some days the patch enlarges, and fresh patches may appear. The patches are hairless, smooth, shining, and paler than the surrounding scalp. Their margins are abrupt, though close inspection reveals some broken hairs both on the border of the patch and among the long hairs immediately around. Occasionally there may be broken stumps on the patch itself. The stumps seen on a patch of alopecia are characteristic: while retaining their normal lustre and elasticity, they have the shape of a note of exclamation (!), being of normal thickness at their free end, and pale and slender towards the root. They are attached to the skin by an atrophied root, and when pulled out with the forceps come away with a sharp click. Often the eyebrows are also affected with bald patches.

PATHOLOGY OF ALOPECIA AREATA.—Microscopical sections of alopecia areata show atrophy of the hair follicles with destruction of hair papillae, and a small amount of cell infiltration, the cells being mainly mast cells, suggesting a toxic irritation rather than a microbic invasion.

DIAGNOSIS OF ALOPECIA AREATA.—Alopecia areata is most often mistaken for ringworm. It is distinguished by the small bald areas with absence of the typical ringworm stumps. The stumps of alopecia when present are of the note of exclamation type already described.

PROGNOSIS OF ALOPECIA AREATA.—In children the prognosis is good. Recovery usually takes place in from six to eighteen months. Downy hair first appears, and this becomes gradually stronger and pigmented. Relapses are common. In cases where the alopecia becomes universal the prognosis is not so good, though it is better than in adults, complete recovery sometimes taking place after several years.

TREATMENT.—Little can be done to hasten the growth of hair in cases of alopecia areata, but it is usual to prescribe tonics and local stimulating lotions or ointments. The following are examples of such applications: Sulphur ointment well rubbed into the patches; blistering with liq. epispastica; friction with lotions or ointments containing cantharides, or other stimulants; mercury lotions and ointments. Two examples may be given: (1) Ung. hydrarg. oxidi rubr., ℥i.; liq. epispastica, ℞ xv. (2) Acet. cantharidis, ʒss.; hydrarg. perchlor., gr. ii.; spirit. vini rect., ad ℥iii. In the more extensive cases these applications must be diluted.

Scleroderma.—Scleroderma is an uncommon disease. It occurs in two forms: (1) As single patches—*morpheæ*—(2) as diffuse scleroderma.

ÆTIOLOGY.—The cause of the disease is unknown. At one time it was regarded as rheumatic, then as of nervous origin, and now it is thought to be of toxic origin, a view which is perhaps suggested by the fact that it is sometimes associated with erythematous or urticarial eruptions, and with joint pains and swellings.

SYMPTOMS.—*Circumscribed Scleroderma*, or *morpheæ*, occurs as rounded or oval patches one or more inches in diameter, or as bands several inches in length. The patches are smooth and ivory white in colour, and feel hard and stiff like a piece of leather let into the skin. At the margin of the patch there is a narrow band of erythema—the "lilac border." Such patches may occur on the chest or back, on the limbs, or on the face. On the face they usually occupy the area supplied by the first, second, and third divisions of the fifth cranial nerve, but elsewhere

they appear to have no relation to nerve distribution areas. There is generally one patch, but there may be several.

Diffuse Scleroderma may occur as multiple patches or bands with more or less symmetrical distribution on the limbs and trunk, or it may be in large areas involving sometimes almost the whole body. Cases vary much as to the degree of thickness of the skin in the involved parts, and there is also some difference according to the stage of the disease. In the earlier periods the skin may be swollen or doughy, while later it is shrunken and stiffened, as in morphea.



FIG. 181.—SCLERODERMA IN THE AREA OF DISTRIBUTION OF THE SUPRA-ORBITAL NERVE.

The white area was smooth, shiny, with a feeling of stiff leather, and adherent to the skull.

There may be associated recurrent erythematous eruptions in the earlier stages, and pigmentation in the later. The condition known as *sclerodactylia*, in which the hands and fingers are stiffened and contracted into claw-like deformities, is produced when these parts are involved.

PATHOLOGY.—Histologically the lesions are made up of a dense fibrous tissue, while the erythematous margin shows the bloodvessels surrounded

by a cell exudation. The conditions suggest a toxic erythema with cell exudation followed by fibrous-tissue cell proliferation.

PROGNOSIS.—The disease may last for years, and recovery eventually takes place by resolution of the lesions. Sometimes atrophy and pigmentation is left. The generalized cases have in some instances been fatal from emaciation and exhaustion, but complete recovery may be hoped for even in the diffuse form.

TREATMENT.—There is no satisfactory treatment. In the early stages in both forms salicin or salicylates may be tried. In all cases, when the disease has ceased to extend, regular careful massage is of great value in aiding the resolution. Thyroid extract, at one time strongly recommended, has been found disappointing. Generalized cases should be well clothed and well fed, and if possible should live in a warm climate.

REFERENCE.

ABRAHAM, P. S. : Clifford Abbott and Holliston's System of Medicine, 1902, ix, 79.

Leucoderma—Vitiligo.—Leucoderma is an affection due to a disturbance of the pigment formation in the skin. There occur sharply-defined, rounded white patches without infiltration, scaling, or other obvious changes. They may appear upon any part of the body, including the scalp. On the scalp the hair over the patches is sometimes white. Often the skin around the white patches appears to be more deeply pigmented than normally, but in most cases this is mainly an optical

ilation, and it is merely the contrast which makes the surrounding skin appear darker. When the white patches are large and numerous, the intervening normal areas may be mistaken for patches of pigment on a normal skin, though the convex margins of the leucodermic areas should reveal the true nature of the affection. The disease is usually unaccompanied by any general disturbance of health, but it may in rare instances be associated with migraine, alopecia areata, Graves' disease, or scleroderma.

PATHOLOGY.—Usually the only changes found are—(1) Absence of pigment from the leucodermic areas; (2) less often, increase of pigment in the immediately surrounding skin.

Slight cell infiltration round the blood-vessels has been observed in recent cases, and this is thought to point to a possible toxic origin.

TREATMENT.—None of the agents which cause hyper-pigmentation of normal skin, such as local irritants, light, X-rays, etc., will induce pigment formation in the leucodermic areas, and attempts to bleach the surrounding skin by means of lotions containing mercury and peroxide of hydrogen are practically of no avail. Exposure to sunlight should be avoided as much as possible, as this tends to exaggerate the contrast by causing deeper pigmentation of the normal skin.

Granulosis Rubra Nasi.—Under this name Jadassohn described (1901) an affection which, from the considerable number of cases since recorded, appears not to be uncommon. There is a redness with hyperidrosis of the end of the nose and of the alae nasi. Generally the skin covering the cartilage of the nose is most affected, but the redness and swelling have in some cases extended to the upper lip and cheeks. Associated with the redness are minute reddish-brown papules, which give rise to the "granular" appearance from which the affection gets its name. The nose is cold to the touch. Buds of sweat cover the dusky-red areas. Sometimes one or more small cysts (probably dilated sweat ducts from the retention of sweat) are present in addition to the granules. The hands and feet may be cold, with a tendency to sweating.

PATHOLOGICALLY there is found a moderate cell infiltration round the ducts of the sweat glands. It seems probable that the essential part of the affection is hyperidrosis, and that the inflammation and cystic formation are secondary results.



FIG. 182.—VIRILLO, OR LEUCODERMA.

The white areas are the result of loss of pigment. The rest of the skin is normally pigmented.

ETIOLOGY.—It may begin as early as six months, but most cases recorded here have been of eight to ten years of age. There is some tendency to family incidence. Usually the affection disappears at puberty, but the cysts may remain.

DIAGNOSIS.—Several cases have been at first diagnosed as lupus vulgaris. The associated sweating and the fact that the granules fade on glass pressure should prevent this error.

TREATMENT.—Temporary benefit has been obtained from local astringents, such as lead lotion or glycerin of tannic acid; but the only known method of cure is by X-ray applications, which stop the sweating and clear up the slight infiltration.

REFERENCE.

MacLEOD, J. M. H.: Brit. Jours. Dermat. London, 1906, xvii, 242.

D. CONGENITAL AFFECTIONS.

NAVI.—The term "navus" as used in dermatology is applied, not only to vascular overgrowths, but to all congenital localized overgrowths of any of the elements of the skin. It often happens that more than one type of navus is present in the same patient, or there may be other congenital defects.

ETIOLOGY.—The cause of navus is unknown. Local injuries at birth have been suggested as the cause of vascular navus; moles and linear navus have been thought to arise from imperfect joining up of embryonic segments.

VASCULAR NAVI, OR CONGENITAL ANGIOMATA.—Vascular navus are by far the most common congenital defects of the skin. They may be described as localized overgrowths of vascular tissue. There are several varieties—viz., the port-wine mark, cutaneous arteria, subcutaneous arteria, and mixed navi. The appearances of these are familiar, and they need not be here described. The three last varieties may sometimes disappear spontaneously in the course of time, but sometimes they may increase rapidly in size. Apart from the disfigurement they may give rise to, they are also liable to be injured and to ulcerate when on parts exposed to friction.

TREATMENT.—For port-wine marks there is no satisfactory treatment. Small cutaneous and subcutaneous navus are best treated with CO_2 snow, with application of twenty to sixty seconds according to the depth of the navus. The larger and deeper navus may be excised if on the body or limbs, or treated by galvanocautery or electrolysis if on the face.

MOLES, OR PIGMENTED NAVI, are commonly situated on the face, neck, or back, but they may occur elsewhere. They may be single, few, or numerous, and may vary in size from a few lines to several inches in diameter. They are commonly seen as rounded or oval, deeply pigmented elevations, with coarse hairs springing from them. Sometimes they may involve large areas, as the whole of one side of the face or the lower part of the lumbar region, buttocks, and thighs. Sometimes hairs may be absent, or the patches may consist solely of pigment.

The **PATHOLOGY** of soft moles is a subject of discussion. The moles are composed of masses of cubical cells arranged in lines, and it is a disputed point as to whether these cells arise from the epidermis or the endothelial cells of the cutis.

PROGNOSIS.—In the large majority of cases moles are harmless apart from the disfigurement they may cause; but sometimes a mole will take on a rapid growth, and in rare instances in later life malignant growths of a carcinomatous nature may develop upon the site of moles.

TREATMENT.—Unless disfiguring or rapidly growing, moles require no treatment. If rapidly growing they should, if possible, be excised. For small hairy moles the best treatment is destruction with CO_2 snow. The stick of snow is cut to the size of the mole, and firmly pressed on to it for from thirty to sixty seconds. It is best to begin with the shorter time, and repeat the application some weeks later if necessary. A blister forms, then a crust, and when the crust has separated there is left a pale scar. The hair papillae may be destroyed, and hair growth permanently prevented by this method. It has the advantage over the older method of electrolysis that it is comparatively painless. Even large areas of hairy pigmented mole may be destroyed, and replaced by scar tissue, by the use of CO_2 snow.

LINEAR NAevi (unilateral linear warty naevi) is of much commoner occurrence than is generally supposed, but it is often not diagnosed as such. They occur in lines or streaks, which may extend along a limb, across the chest or face, or down the middle line. They are generally unilateral. Sometimes there is a single streak; sometimes many streaks occupy the whole or part of one side of the body. Occasionally they occur on opposite sides of the body, but without being symmetrically arranged. They may be a few inches long, or may extend the whole length of a limb or half round the body. The streaks consist of raised warty growths, sometimes darkened by exposure, and sometimes with their base inflamed by infection by *pus cocci*. In marked examples of this affection the streaks may become projecting warty bands, and are then given the name of *Ichthyosis hyperic*.

PATHOLOGY.—The structure of these naevi differs from that of soft moles in that the cubical cells of the soft mole are absent, and the growth is made up generally of an overgrowth of the elements of the epidermis, horny cells, and prickly cells, but sometimes of hair follicles and sebaceous glands, or even of sweat glands.

PROGNOSIS.—These naevi often do not become noticeable until five to ten years of age or later. They occasionally disappear spontaneously, but generally require surgical or other interference for their removal.

TREATMENT.—They may be removed surgically by excision. They may be burnt away by actual cautery. Or if these measures, which require an anæsthetic, are objected to, they may be treated with CO_2 snow.

Repeated application of salicylic acid (5*ii*) in flexible collodion (5*i*) for several weeks will remove the horny growth to a large extent, and allow the more ready application of the CO_2 snow.

OTHER CONGENITAL OVERGROWTHS classed as naevi are lymphangiomas, xanthomas, adenoma sebaceum, and multiple neuro-fibromata.

REFERENCES.

MOLES.

FOX, WILFRED: *Researches into the Origin and Structure of Moles*. Brit. Journ. Derm. 1905, XLII, 1 (good bibliography).

LINEAR NAevi.

HODARA, MURAHIM: *Journ. des Mal. Cut. et Syph.*, 1909, XVII, 361 (bibliography).

Ichthyosis.—Ichthyosis is characterized by a dryness and hardness of the skin, accompanied by a varying degree of scaldness or hyper-keratitis.

ETIOLOGY.—The disease is a congenital affection, though it is often not noticed until some months after birth. The cause is unknown. It has no special tendency to run in families.

¶ **SYMPTOMS.**—A mild form of ichthyosis to which the term *xeroderma* (dry skin) is applied is very common. The skin is dry and rough and dirty-looking everywhere, except in the flexures—i.e., the front of the neck, the bends of the elbows, the axillæ, the groins, and the bends of the knees—which are quite smooth. The palms and soles are also free from roughness, but they are always deeply furrowed by an exaggeration of the natural lines—"monkey palm." These patients are very subject to chapping of the exposed parts in winter, and to eczematous or impetiginous forms of dermatitis.

In the more severe forms of ichthyosis the skin is covered with scales, which are arranged in a mosaic, each scale being adherent at its central part and free at its margins, giving the appearance of fish skin or, in severe cases, of crocodile hide.

PATHOLOGY.—The skin shows a thickening of the horny layer, with a deficiency of sebaceous and sweat glands. It is probable that the symptoms depend a good deal upon the absence of normal fat and sweat secretion. From the good effect of thyroid extract internally, there is reason to believe that a disturbance of function of this gland is responsible for the skin condition, but there is no direct evidence that this is the case.

The **DIAGNOSIS** presents no difficulty.

TREATMENT.—The skin will become soft and the scaldness disappear when thyroid gland is given internally. But such improvement only lasts while the thyroid is taken, and it obviously cannot be continued indefinitely. The affection lasts through life, but much may be done by simple local measures to keep the skin in better condition. The best treatment is by frequent baths, followed byunction with a simple grease, such as liquid vaseline. It is an advantage to add a few tablespoonfuls of glycerine to the bath. Cod-liver-oil internally is often useful in improving the nutrition of the skin, these patients being generally thin and wanting in cutaneous fat.

Xeroderma Pigmentosum—Kaposi's Disease.—This is a rare affection which develops in early life (first or second year) as a freckle-like pigmentation. The pigment spots are seen first upon the exposed parts—namely, the face and hands—and generally in summer. Later they appear upon the neck, the upper part of the chest, the hands, and the forearms, and in some cases on the legs below the knees. After one or two summers the freckles cease to fade in the winter; telangiectases and small angiomas appear, also smooth white spots of atrophic skin. In course of time the pigment areas and the white spots enlarge and blend. Ectropion may be produced by contraction of the scar tissue. Then there appear small superficial crusted ulcerations and small wart-like growths among the pigment spots, and eventually, occasionally quite early, generally after ten to twenty years, the sores and warts give rise to malignant fungating growths of a carcinomatous nature.

¶ The **CAUSE** of this remarkable disease is unknown. It shows a strong family tendency, though confined to one generation. Sunlight is regarded as a probable exciting cause. There is a close resemblance between this affection and senile

degenerations of the skin and X-ray dermatitis, both clinically and histologically. "Xeroderma pigmentosum may be considered as a precocious senility of the skin" (Sequeira).

TREATMENT.—In the earliest stages the skin should be protected from light by means of a grease-paint coloured with burnt umber, or by a brown veil. Small tumours or ulcers should be excised. X-ray applications in properly-measured doses may also be employed for removal of warts and for healing ulcers.

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 CROCKRAN, H. H.: *Med.-Chir. Trans.*, 1884, lxxv, 189.

Xanthoma is a rare affection of congenital origin, or appearing during the first months of life, characterized, as its name implies, by yellow nodules or tumours. These occur disseminated over the body as firm, elastic and projecting rounded nodules of a bright yellow colour, split-pea-sized to bean-sized. Sometimes certain of the lesions are associated with a vascular naevoid condition.

The duration of the affection is indefinite; in some cases the lesions have disappeared spontaneously.

PATHOLOGY.—Histologically the lesions show cells containing fatty granules, generally regarded as altered embryonic cells. These tumours are probably xerai of the endothelial type in which the xerous cells undergo a fatty change during their dissolution.

TREATMENT.—Lesions in exposed positions may be excised or treated by freezing with solid CO₂.

REFERENCE.

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